NATURE AND NURTURE
ARE WE MISSING A THIRD OPTION?

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A thesis submitted for the degree of

Master of Health Science

at the University of Otago, Dunedin, New Zealand

February 2013

ABSTRACT

In light of my own situation, I have been drawn to question whether all experiences can be explained in terms solely of nature and nurture. This has led me to seriously question the notion of genetic determinism. While the interplay of nature and nurture may be far more complicated than we currently understand, the uniqueness of my situation suggests there may be more to the story which led me to wonder whether, in fact, there may be a third option: nature + nurture + '?'.

This thesis focuses on two areas. Firstly, genetic determinism—the notion that our genes determine who and what we are—is examined. Genetic determinism is the basis of the 'nature' part of the dichotomy. Secondly, if the genetic determinism component of the nature and nurture equation is found lacking, what is it that might be missing?

There are two possible candidates for the third option: emergence and chaos. Emergent properties are those which arise out of the interactions of the components of a system. These properties are an "extra piece" that is present when the system (such as the human body) functions as a whole but which cannot be identified when the system is examined in its component parts (such as organs or tissues). Chaos is behaviour within a system that we cannot predict from looking at the system's initial conditions. For example, fluctuations in the levels of regulatory molecules within a cell can result in fluctuations in gene output or the increased variation in dendrite formation of melanocytes due to an organism having only one functional copy of the tumour-suppressor gene neurofibromatosis type 1 (NF1), instead of two (haploinsufficiency).

Both of these concepts represent new areas of scientific inquiry. While the evidence for emergence is interesting, this thesis concludes it is too early to be drawing any significant conclusions about its role. The evidence supporting the role of chaos is also limited but it is compelling. This thesis concludes that chaos may be the third option in the nature and nurture debate, although its role is likely very small.

Many arguments in bioethics are launched from a position of genetic determinism on the assumption that after an assessment of an individual's genes, we can draw

conclusions about who they are now and who they will be in future. To examine the questions posed by this thesis the areas of genetic testing, genetic modification and cloning are examined as examples of the role of genetic determinism in bioethics. This thesis is significant for bioethics because it demonstrates that one of the tenets of many bioethical discussions is based on a poor understanding of science. In addition to the areas examined in detail, the implications of this thesis for neuroimaging, violent or criminal behaviour, chemical castration of sex offenders, mental illness and synthetic biology or artificial life are also briefly discussed.

ACKNOWLEDGEMENTS

Firstly, I would like to say thank you to my supervisor, Professor Gareth Jones. I am grateful to you for keeping my feet on the ground during such a complex and challenging project. Your constructive criticism and feedback has kept this project on the right path and helped steer me towards a satisfying and relevant conclusion.

A huge thank you also goes to my husband, Elliott, who missed my company (and baking!) on many evenings and weekends while I worked on this thesis. I would not be where I am today without your unrelenting support of my endeavours.

I owe a debt of gratitude to my friends, in particular, Julie, Julia, Ilka and Adrienne. Thank you all for your encouragement, energy and enthusiasm. I would be far crazier were it not for all of you cheering me on and chilling me out.

Thank you also to all of my colleagues at the Otago Community Hospice, in particular, Lyn, Liz and Breffni. Thank you for your patience and understanding. I apologise if my mind may have been elsewhere on occasion!

Finally, I would like to thank my peers in the Bioethics Centre. Especially to Claire, for checking on me when I worked from home and Lynne, Stacey and Albany for your invaluable advice at the end which helped me get this project across the finish line.

Thank you all.

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1 Introduction

I am an anomaly. In the context of the traditional nature versus nurture dichotomy, my life story does not make sense. Nature did not give me a lot to work with; the genes in my family are underwhelming. I come from a long line of individuals who have made very little of themselves; individuals of moderate intelligence who aspire to very little. Nurture was not kind to me either. I was not nurtured in a loving, secure, supportive environment. Rather, the individuals who raised me did so in such a way that I have post-traumatic stress disorder from prolonged childhood neglect and maltreatment.

Perhaps unexpectedly, this is not a sad story. By all accounts, I have exceeded the expectations of my upbringing and genetics. I am happily married. My husband and I have chosen not to have children for now. I have a large, supportive network of friends. I am financially stable, in fact, my husband and I have recently purchased our first home. I have run a successful business. I have a postgraduate tertiary qualification. Alongside my study, I am employed in a professional capacity. None of my relatives—including my brother who, arguably, has the same genes and upbringing—can tick even half of these boxes, let alone all of them. If nature and nurture is all there is, how can this be? How can I be?

While the interplay of these two factors, nature and nurture, may be far more complicated than we currently understand, the unusualness of my situation suggests there may be even more to the story. Examining my own life story led me to wonder whether, in fact, there may be a third option: nature + nurture + '?'. Therefore, the focus of this thesis will be to question the validity of the doctrine of genetic determinism in the face of strong scientific evidence that there is more to who we are than simply our genes and the environment in which we were raised. Rather surprisingly, the evidence against determinism comes largely from the physical sciences of physics and mathematics, rather than biology. Studying these areas reveals there is a lot happening at the molecular and systems levels which affect who we are, but more on this later.

It is difficult to consider determinism without also looking at reductionism; the two go hand in hand. Reductionism, in brief, is the view that we can best understand something by breaking it in to smaller units and determinism is the view that we can make predictions about that thing based on what we know about the units. This is a very

circular model and it is difficult to know where the starting point is or should be; similar to the fabled chicken and egg scenario. In fact, by some definitions, determinism is actually considered a form of reductionism and referred to as *causal reductionism.*

Reductionism is not easily defined. Ironically, the many scientists and philosophers who have attempted to reduce the concept in order to better explain it have only muddied the water. *Methodological* reductionism refers to the traditional scientific method of acquiring knowledge (Ayala & Dobzhansky, 1974, p. viii) and has been the modern scientific approach since the time of Newton (Chan & Chia, 2003, p. 193). Broadly speaking, methodological reductionism involves "analysing the thing to be studied in terms of its parts" (Murphy & Brown, 2007, p. 47), thereby reducing it to simpler terms. It is widely agreed that while this was the focus of early modern scientists, current scientific investigations may benefit from supplementing this knowledge with an understanding of higher-level entities, including the role of the environment.

Epistemological reductionism is perhaps best explained by Lord Ernest Rutherford who said: "All science is either physics or stamp collecting" (Birks, 1962) –or, in other words, all science can be reduced to physics. Fundamentally, epistemological reductionism aims to explain the scientific world (both animate and inanimate) using the fewest possible concepts and laws. Epistemological reductionists believe that laws relating to higher-level entities (humans, for example) can be reduced to laws relating to lower-level entities (atoms, for example). Therefore, the laws and theories relating to biology are not stand-alone laws; rather they are special cases of laws and theories formulated in another branch of science (Ayala & Dobzhansky, 1974, p.ix). In this case (and, in fact, in all cases) the fundamental branch of science to which all other sciences can be reduced, is physics. The driving force here is the persistent belief that "every problem, however complex, can be modelled in terms of some basic formal rules..." (Chan & Chia, 2003, p.193) and therefore life can be reduced to chemistry, and chemistry can be reduced to physics (Lehrer, 2007, p. xi).

The practice of reductionism creates a "hierarchy of theories" (Dupre, 1993, p.4), with each higher-level theory "derivable from [and nothing more than] a theory of simpler

entities" (Dupre, 1993, p.4). This type of reductionism is often referred to as *ontological* because the higher-level entity is said to be nothing more than the sum of its parts and "there is a chain of causation that runs from the units to the whole" (Lewontin, Rose & Kamin, 1984, p. 6). It is argued that ontological reductionism means "that as one goes up the hierarchy of levels, no new kinds of non-physical "ingredients" need to be added to produce higher-level entities from lower" (Murphy & Brown, 2007, p 47). Simpler entities are said to exist prior to higher-level entities which they comprise. In other words, human beings are nothing more than the adding together of "simpler entities"; no *élan vital* required.

Two other less well known classes of reductionism exist. *Causal* reductionism is the view that the behaviour of the simpler entities determines the behaviour of all entities higher up in the hierarchy (Murphy & Brown, 2007, p. 47). Causal reductionism is, therefore, more commonly referred to as *determinism*. *Atomist* reductionism is the view that only the simplest entities "are *really* real; higher-level entities—molecules, cells, organisms—are only composite structures (temporary aggregates) made of atoms" (Murphy & Brown, 2007, p. 47-48).

The issue with attempting to distinguish so many branches of reductionism is that it makes each branch seem as though it is a stand-alone concept. Rather, the many branches of reductionism should be seen as different phases in the process of acquiring knowledge. First, in order to acquire new knowledge through scientific discovery, one must have a question or a need that the knowledge is to address. In the case of reductionism, the [epistemological] question relates to whether all experimental laws and theories in the universe are simply "special cases" (Ayala & Dobzhansky, 1974, p.viii) of the experimental laws and theories of physics. The second consideration is how this new knowledge will be acquired. A [methodological] reductionist approach here would see the problem reduced to smaller components for further study, for example, a particular biological theory may be reduced to an organ system or its molecular components. Next in the scientific method, one would decide what this newly acquired knowledge could be used for. In the case of [causal] reductionism, one might use the new knowledge of a particular organ system to make predictions about future biological events or behaviours occurring in the whole organism. Finally, having completed the scientific journey to acquire knowledge one may begin to ask

metaphysical [ontological or atomist] questions about the nature of the knowledge itself. Of most significance for this thesis will be the concept of methodological reductionism as well as, of course, determinism (or causal reductionism).

Determinism is the doctrine that things happen because they were predetermined (or destined) to happen. In particular, biological determinism suggests that "human lives and actions are inevitable consequences of the biochemical properties of the cells that make up the individual; and these characteristics are in turn uniquely determined by the constituents of the genes possessed by each individual" (Lewontin, Rose & Kamin, 1984, p.6). For example, a determinist would argue that none of us is choosing to pick up a pencil. Rather, our picking up of the pencil was determined by the genes in our brain. These genes code for particular cell properties and groups of these cells working together to orchestrate a movement pattern that coordinates the muscles in our upper limb and causes us to pick up the pencil. To a determinist, "we" didn't decide to pick up the pencil. It was predetermined by our genes. Lewontin, Rose and Kamin argue all human society is governed by this chain of events and the chain can be extrapolated to determine "the sum of the behaviours of all individuals" (p.6). The issue for this thesis is the extent to which determinism should follow from methodological reductionism. While it may make sense as part of the scientific method to break an organism in to ever smaller categories in order to understand the workings of the whole, is it then valid to make determinist predictions about the behaviours and actions of the whole based on this knowledge of the smaller categories?

I intend to argue that the doctrine of determinism is a fallacy because it is based on unsound reasoning and evidence. Fallacy may be a bold and controversial word but, used here the word "fallacy" should be understood to mean simply a mistaken belief or a belief based on unsound reasoning.

I will argue that in order to be truly deterministic, one must make predictions based on information related to the most fundamental units of the whole. In biology, most deterministic predictions are made using information gleaned from units a few layers down the hierarchy, for example, organs and cells. I will show that when we look as far down the hierarchy as it's possible to go—to the atomic and molecular level—the idea of making accurate deterministic predictions loses credibility.

By way of illustration, imagine you intend to build a house and you're standing at the building site staring at the grassy paddock that will one day bear the weight of your new house. It is unthinkable that any rational person would make predictions about the structural integrity of the yet-to-be-built house based solely on what could be learned by looking at the grass. We all know that there are many, many layers of earth under the grass and the stability of these layers will affect the stability of your new house. For example, if the grass is concealing layers of soft, shifting sand the impact of this sandy layer will have a flow on effect and ultimately, the sand will detrimentally affect the stability of the house. When considering this example, a determinist would scoff at the thought of trying to determine the stability of the house based on surveying the grass alone. It's clearly ridiculous. The grass is not the most fundamental unit, therefore, it is not the most important consideration in determining the stability of the house. One must look below the grass.

Biology—specifically, people—should be considered no differently. In order to make predictions about the whole person, we must look below the level of the grass, below the organs and cells, and look at what occurs a few layers below.

Looking at the layers below, it becomes clear that fundamental laws are not deterministic.

1.1 Issues Raised by Genetic Determinism

The basic doctrines of reductionism and determinism raise two main issues that cast doubt over how clear cut the principle of genetic determinism really is.

The first issue concerns the number of genes in the human body. The human genome contains approximately 20,000 coding genes (Sapolski, 2010) there are simply not enough genes in our genome to code for all of our cells and processes in a one to one manner. In other words, we do not have enough genes for every cell and tissue type, physical and behavioural trait to have its own gene. If every cell, every process and every function we perform required its own separate gene, we would need millions of

genes – not 20,000. For example, there are millions of capillaries in the human body (Sapolski, 2010). If every bifurcation in every capillary required a separate gene, that single process alone would run our genome in to the millions. The impression we get from reading scientific textbooks is that the way we look and behave is a result of our genes; that every facet of our bodies and behaviour has its own miniature "recipe card." But if each physical trait and behavioural attribute is not coded for by its own gene, how does this system work? And, more importantly for this thesis, could this discrepancy in the numbers provide a possible third option; an alternative to nature or nurture that might help to explain how we are who we are?

More commonly, this discrepancy is referred to as "*emergence*" and it is the emergent properties of nonlinear systems which may provide a third option. A nonlinear system is any system where the sum of the parts is greater than each part on its own, that is, the system does not display linearity of additivity. Quite simply, in a linear system, one plus one equals two; in a nonlinear system one plus one might equal three. In a nonlinear system, the emergent property is the result of this "extra" piece that has been added to the system. For example, in the heart, a single cardiomyocyte is nothing special; however, a network of cardiomyocytes creates a beating heart. Similarly, a single neuron is not capable of much, but a network of neurons creates a brain capable of consciousness and creativity. It is these emergent properties that create problems for methodological reductionists. Creativity cannot be found by reducing the brain and its neurons to ever smaller components because creativity does not exist anywhere tangible. Creativity is an emergent property of an interactive network of neurons.

A second issue relates specifically to the way our genes are transcribed and translated to the proteins that make up the way we look and behave. Using traditional point for point reductive logic to explain complex systems does not take into account random chance. The random vibrations of molecules (Brownian motion) will have implications for human biology – from conception all the way up to who we are in our everyday adult lives. For example, every time a cell splits, Brownian motion will result in an unequal distribution of the cell content (Lehrer, 2008, p.49; Sapolski, 2010, 39:50). One cell may end up with a large number of mitochondria; the other cell may get very few. These small instances of random chance have the potential to add up to large differences and explain why chance is a major barrier to applying reductive logic to

human biology. In the scientific literature, chance is referred to as "stochastic gene expression" which is said to result in phenotypic variation among cells that are genetically identical and exposed to the same environment. Stochastic gene expression, therefore, may be another candidate for the third option; an alternative to nature or nurture. Stochasticity (or chaos) could affect any number of cell processes, for example it may alter the level of promoter binding, mRNA translation, protein degradation (Raser & O'Shea, 2005) or result in varied reaction rates (McAdams &Arkin, 1999). This creates a serious conundrum for determinists. If our genome is the "recipe" for making us, why, then, do identical twins have different finger prints? If our genes are faithfully transcribed and translated like clockwork why did the first cloned cat, Cc (for "carbon copy"), look and act nothing like her genetic mother, Rainbow? As Raser and O'Shea (2005) put it, "...even cells or organisms with the same genes, in the same environment, with the same history, display variations in form and behaviour that can be subtle or dramatic." How can a determinist argue that our thoughts and actions are the "inevitable consequences of the biochemical properties" (Lewontin, Rose & Kamin, 1984, p.6) of our cells when these properties of our cells are not fixed, but rather, are chaotic?

This thesis will examine whether there is sufficient evidence to support the idea that chaos or emergence may be the third option. If so, what are the real-world implications of this theory?

1.2 Relevance

Understanding genetic determinism will inform a broader discussion regarding the role of genetic technologies in our society and how we view these advances within bioethical frameworks. Exploring the validity of claims related to genetic determinism will allow sound decisions to be made regarding future healthcare policies, as well as inform both journalists and the public.

Questions related to genetic determinism are part of a broader, ongoing discussion concerning rapidly advancing biotechnologies and how they reconcile with our existing moral and bioethical frameworks. Further, it is important that we give people a very

clear understanding of exactly what genetic determinism is and what it is not. A poor understanding of the role and power of genetic determinism may give people an inadequate perception of the causal role of genes in illness and lead them to believe the predictive power of genetic tests is high. In addition, an inadequate understanding of genetic determinism may lead to the development of unnecessary public policies and result in a lack of individual responsibility.

Media coverage of advancing genetic technologies and the issues these technologies raise goes a long way towards informing public opinion and the public's understanding of the science involved (Lynch et al, 2011). According to Lynch et al, genetic information is often perceived by the public as "definitive" and this view is reinforced by the fact that the role of social and environmental factors in disease pathologies is not often discussed in media coverage (Arnason & Hjörleifsson, 2007). Media coverage reports that genetic technologies will have desirable outcomes, will greatly improve healthcare and that progress is inescapable (Arnason & Hjörleifsson, 2007). Journalists frequently suggest that genetic technology is on the verge of entering "the medical mainstream" (Arnason & Hjörleifsson, 2007) and the validity of the research supplied to journalists is seldom questioned (Arnason & Hjörleifsson, 2007; Ross, 2004). In light of how little journalists question the information they are given, the results of a study by Lynch et al (2011) are troublesome. The group conducted a survey of media coverage to assess how often the concept of genetic determinism (among other things) appeared. Genetic determinism appeared in 42 of 92 news stories. 10 of the news articles were considered "high genetic determinism" which the authors say coded for "stories that presented genes as the sole factor in determining human outcomes." Thirty-one articles were coded "moderate genetic determinism." This study shows the extent to which journalists are perpetuating the idea that our genes play a deterministic role in human development and behaviour.

Genetic determinism is also made relevant by the potential impact of genetic technologies on healthcare in the coming years. An understanding of genetic determinism is required to provide the context for a broader discussion related to the many issues these technologies will raise for our healthcare systems and healthcare providers themselves. Genetic testing is expected to be used widely for predicting disease in healthy people and for the "diagnosis and management of patients... (Bell,

1998).” Further, genetic technologies are expected to reveal the genetic contributions of human diseases and “the development of rational strategies for minimizing or preventing disease phenotypes altogether (Collins, 2000).” But, without an understanding of how genotype relates to, and directs, phenotype it will not be possible to make informed decisions about the information revealed by genetic technologies.

Genetic technologies raise many issues for healthcare systems and clinicians that make an understanding of genetic determinism necessary. For example, genetic testing may lead to a proliferation of unnecessary screening programmes unless the relationship between genotype and phenotype is better understood (both by healthcare professionals and the public, who may drive demand for genetic testing). Secondly, we must understand the role of genetic determinism in order to answer the questions of whether we should test for diseases we cannot treat, whether we should test children and what impact test results may have on family members. Genetic testing is likely to lead to earlier identification of diseases (or the potential for a particular disease to develop). How the results of these tests are interpreted will decide how much of an impact this new information will have on our already strained healthcare system. It will not help hospitals and general practitioners to have their waiting rooms clogged with people who may, at some point, develop a disease, as well as those who are currently sick. Potential also exists for genetic discrimination which may be mitigated significantly by a sound knowledge of the impact of genetic determinism. Finally, we need to understand genetic determinism in order to prevent individuals from deciding it unnecessary to take personal responsibility for their health and lifestyle. If people believe their genes are the sole cause of their disease and there is nothing they can do to prevent getting sick, or that if they do get sick they will simply undergo an enhancement therapy, or have an organ cloned, we will all potentially end up much sicker and much worse off.

In bioethics (and, more broadly, in philosophy), determinism is a concept that is applied widely and ties closely with arguments related to free will. Whether or not humans have free will is a fundamental question in bioethics and philosophy. In short, the argument between determinism and free will goes as follows: in the natural world, all events have causes and identical causes produce the identical effects; humans are part of the natural world, therefore, human behaviour must be deterministic; if humans are to subvert

deterministic laws, we must possess something extraordinary (or "super-natural"), such as consciousness or a soul which is able to make its own decisions. The aim of this thesis is to highlight the implausibility of determinism and, therefore, the outcome of the arguments presented here will have implications for broader discussions related to free will.

1.3 Aims

In closing, the aim of this thesis is to question the validity of genetic determinism with a view to presenting a "third option" in the nature versus nurture debate. This topic has come out of my own attempts to explain why I am an intelligent, well-rounded, successful person when both nature and nurture would have predicted otherwise. I intend to argue that the concept of determinism falls short on two fronts. Firstly, it fails to take in to account the emergent properties of complex systems. Where emergent properties are involved, we cannot make predictions about the mature state by studying the initial conditions. Similarly, we cannot reduce the mature state to its initial conditions. Secondly, determinism does not make any allowances for chance, or stochasticity. The random distribution of molecules within, and between, cells has implication for the mechanics of the cell. All of these minor instances of random chance, or chaos, may translate in to larger implications for the whole organism. The relevance of this thesis can be seen on many fronts. The debate between determinism and free will is a class argument in bioethics and philosophy. The arguments presented here may have significant consequences for this debate. Academics and theory aside, the fallacy of determinism has many practical consequences for how we live our everyday lives. It raises questions around the role of genes in illness and public policy, the future of healthcare and media portrayal of new technologies. This thesis, it is hoped, will provide greater clarity of the limitations of determinism so that future debates may be better informed.

2 Scientific Background

2.1 What is Determinism?

In philosophy, determinism is the doctrine that that things happen because they were predetermined (or destined) to happen. It is often discussed alongside the concept of free will: are we capable of making free decisions, or is every decision we make the result of (or predetermined by) previous circumstances? Resnik and Vorhaus (2006) call this type of determinism "psychological determinism." The authors say there are three basic positions relating to free will and determinism: 1) hard determinism, which says we do not have free will; 2) indeterminism, which says not all events are caused by previous circumstances; 3) compatibilism (or soft determinism), which says both doctrines can co-exist. For the purposes of this thesis, we will not look specifically at free will, but will instead focus on the genetic argument for determinism.

In biology, determinism is the notion that "human lives and actions are inevitable consequences of the biochemical properties of the cells that make up the individual; and these characteristics are in turn uniquely determined by the constituents of the genes possessed by each individual" (Lewontin, et al, 1984, p.6). Genetic determinism is the notion that our genes dictate how we look and behave. In scientific terms, it is the notion that genotype (an organism's genome) causes phenotype (the observable trait or characteristic, such as brown hair or intelligence).

2.2 The Underlying Science

In order to understand where the genetic deterministsare coming from, it is important to know how a genome might determine the physical trait. What is the mechanism that connects the two?

A genome is composed of DNA, deoxyribonucleic acid, which is the hereditary unit of all living material (Watson and Crick, 1953). DNA is effectively a recipe or set of instructions which tells a cell how to create a certain piece of protein. Each gene contains the DNA (or instructions) on how to create a specific protein; a genome is an organism's entire set of DNA and therefore represents all of the potential proteins the cell can make. DNA is made of strings of nucleic acids and each of the four nucleic

acids is represented by a letter (A,T,C,G) (Levene, 1919). When read in groups of three (called codons), these letters tell the cell which amino acid (the building blocks of protein) should be inserted at that particular point in the protein. For example, if the DNA code says "TAT," that tells the cell to insert the amino acid tyrosine at that point. "TCT," however, would tell the cell to insert the amino acid serine (Crick et al, 1961).

To get from DNA to the proteins we're all made of, DNA must first be transcribed in to RNA. DNA and RNA are very similar, with the main differences being that RNA is single stranded and has the nucleic acid uracil (U) rather than thymine (T). In the cell's nucleus, DNA is transcribed in to messenger RNA (mRNA) by an enzyme called RNA polymerase II (Hurwitz, 2005). This process creates a complementary copy of one of the strands of DNA. The enzyme attaches to the strand of DNA in a specific region (the promoter region) and moves along the strand, transcribing as it goes. Other enzymes, called transcription factors, unwind the helical structure of the DNA so that the RNA polymerase II can make its way along. Once finished, the RNA polymerase II has created a single strand of nucleotides called which we now call mRNA (Hurwitz, 2005). DNA cannot be translated directly in to protein because DNA resides in the nucleus of the cell and is too large to pass in to the cytoplasm where proteins are made. RNA is significantly shorter than DNA because RNA contains instructions on how to make just one protein, whereas DNA contains the code for making many, many different proteins.

A specialised mRNA exporter protein, located in the nuclear membrane, allows movement of the mRNA from the nucleus in to the cell's cytoplasm. In the cytoplasm, the mRNA is translated in to protein by a molecule called a ribosome (Palade, 1955). The ribosome binds to the start of the mRNA and reads the code it contains. Each codon tells the ribosome which amino acid to insert and the appropriate amino acid is "handed" to the ribosome for insertion by a molecule called transfer RNA (tRNA). Stop codons, such as UAA, signal to the ribosome that translation is complete (Brenner et al, 1965).

To complicate matters, following both transcription and translation the mRNA and protein, respectively, undergo further modifications. The details of these post-transcriptional and post-translational modifications are beyond the scope of what is

necessary here. It is worth noting, however, that these additional steps serve as points of regulation, allowing, for example, the cell to destroy a misshapen protein by tagging it with an ubiquitin molecule during post-translational modification. Once tagged, this protein will be recognised by a lysosome and digested (Goldstein, 1975). A variety of post-transcriptional and post-translational modifications serve as checks and balances for the cell, as well as aiding sorting, recognition and many other things.

Why is protein so significant? What is the relationship between the genome, protein and whether or not you have blue eyes?

Proteins are the building blocks of human beings and fall in to two general categories: structural and functional. Structural proteins, as the name suggests, form the structural components of cells, and therefore, the body. Proteins can join together to form molecules and play an important role in the cell membrane. They may also take the form of connective tissues, such as collagen, elastin and keratin, or serve as motor proteins that transport molecules around the cell, such as dynein and myosin.

As with structural proteins, functional proteins may be involved in transport and can bind to each other (as well as other molecules) to form large complexes. Functional proteins work as antibodies and enzymes, regulate the workings of the cell, and are heavily involved in cell signalling. A great example of a cell signalling protein is insulin (a special class of small protein commonly referred to as a hormone), which signals to the body to take up glucose from the blood and store it as glycogen (Banting et al, 1922). A further important role for functional proteins is acting as receptors on the surface of cells. A molecule (or, in fact, another protein) binding to one of these receptors sets off a cascade of activity within the cell. Functional proteins may also form channels across the cell membrane (transmembrane channels) which allow (or deny) the passage of molecules in to or out of the cell.

It is very easy to see that protein is heavily involved in all processes occurring within the cell, but it still isn't exactly clear how proteins (and therefore, DNA) determine how we look or behave. The role of proteins in determining eye colour is a useful example to illustrate this relationship. Many different types of protein serve as the machinery in melanocytes, the cells involved in creating melanin, which is the pigment responsible

for eye colour (as well as hair and skin colour). Melanin is assembled from a chain of tyrosine amino acids within a special compartment of the melanocyte called the melanosome (Swan, 1963). If the melanocyte produces a great deal of melanin, the individual will have dark eyes. If little melanin is produced, the individual will have blue (or light) eyes (Lamb, 2009). It is our genes that code for the proteins the melanocyte needs in order to create melanin. Any mutation in the DNA could result in the absence of a protein necessary to create melanin and therefore, gene mutations reduce the amount of pigment made. These mutations are often inherited from our parents, explaining why eye colour runs in families. In particular, the gene OCA2 can be important in determining how much melanin is produced (although, there are other eye colour genes). In certain individuals, which version of the OCA2 gene they possess they can either produce large amounts of pigment (and have dark eyes), or very little (and have light eyes) (Lamb, 2009).

2.3 Areas where Genetic Determinism Arises: Genetic Testing.

2.3.1 What is Genetic Testing?

A genetic test is a type of medical test designed to determine the presence or absence of a certain gene variant or mutation in order to assess whether a person has a particular disease or is likely to develop the disease. An individual may choose to undergo a genetic test if they are showing symptoms of the disease, if they have a family history of the disease or before having children so that they may assess the risk of passing on a "bad gene."

2.3.2 What can we Test for Now?

There are many different types of genetic testing: preimplantation genetic diagnosis (PGD); prenatal testing; newborn screening; carrier identification; and, testing for late-onset disorders.

PGD (also known as embryo screening) is available to couples using in vitro fertilisation and involves running genetic tests on an embryo before it is implanted in

the woman's uterus (Fragouli, 2007). PGD allows individuals with a genetic disease or chromosomal mutation to select only healthy embryos for implantation (Fragouli, 2007). Many different diseases can be screened for with PGD including cystic fibrosis and α-1-antitrypsin deficiency (Fragouli, 2007). Prenatal testing (such as amniocentesis) is similar to PGD but occurs during pregnancy. As with PGD, prenatal testing can screen for many, many diseases, including cystic fibrosis, Down syndrome and spinal muscular atrophy (Dolan, 2009). Initially, prenatal testing was only carried out to screen for neural tube defects and chromosomal abnormalities but testing has evolved and now "a spectrum of screen and diagnostic tests" is on offer (Dolan, 2009). Newborn screening (NBS) is testing done within 48 to 72 hours after a baby's birth. NBS tests for conditions such as phenylketonuria (PKU), cystic fibrosis and a number of extremely rare diseases (Wilcken and Wiley, 2008). Identification of these diseases allows "appropriate interventions to ameliorate or avoid adverse outcomes (Wilcken and Wiley, 2008)" and has been used widely in New Zealand and Australia since the 1960s (Wilcken and Wiley, 2008). Carrier identification, as the name suggests, is a genetic test designed to test whether a particular individual is a carrier for a genetic disease. The results of this test will allow the couple (and their physicians) to assess how likely it is that their children will have the disease. Whether or not both parents are carriers will affect how likely it is that the child will have the disease or whether the child will be a carrier (Williams, 1997). Genetic testing for late onset disorders involves screening for the predisposition, or likelihood, that an individual will get a disease later in life such as heart disease or cancer (Holtzman, 1999).

In some cases, the genetic test may be diagnostic, for example, an individual with the Huntington's gene will get Huntington's. Other genetic tests indicate only the susceptibility to developing a particular disease, such as Alzheimer's.

2.3.3 What Might We Test for in Future?

Genetic testing is currently used for the purposes of non-medically necessary sex selection—that is, allowing couples to choose whether they have a male or female child for reasons other than the risk of a sex-linked disease (Davis, 2010). Many believe that we may, in future, be able to screen for other traits such as height, beauty, intelligence

and sexual orientation (Fukuyama, 2002; Stock, 2002) although it is fair to argue that these advances are some way off (Robertson, 2002), if at all possible. The genetics of traits such as human intelligence are incredibly complex (Deary et al, 2009) and involve considerable environmental interaction. We may never be able to select for such complicated traits.

2.3.4 How common is Genetic Testing?

In New Zealand (and many other countries such as Australia and the United States), every baby born undergoes NBS. PGD is available in New Zealand to couples looking to start families, although government funding is limited to certain diseases. One cycle of PGD can cost $12,000 so couples ineligible for funding may struggle (Hodgson, 2005). As of mid-2008, there were 1,200 genetic tests available to clinicians in the United States and that number was expected to increase by 25% each year (Hawkins-Allingham, 2008). Already relatively common, it seems reasonable to predict that the prevalence and breadth of genetic testing will only increase.

2.3.5 What Happens with the Test Results?

This depends largely on the test involved. In the case of PGD or prenatal testing, the couple will be given the information and allowed to decide whether to proceed with the implantation or pregnancy (Fragouli, 2007). An individual who learns they carry the gene for a particular disease can then decide whether or not to have children, or perhaps to use PGD. If NBS identifies any diseases the baby can be treated for the disease (if symptoms are already present) or the parents can be prepared for what is to come if the disease presents later (Wilcken and Wiley, 2008). Except in the case of Huntington's, the results of genetic testing for late onset diseases indicate susceptibility rather than a guarantee that the disease will eventuate. If a person finds out they are at an increased risk of developing heart disease, but currently have no symptoms of the disease, they may simply choose to alter their diet and lifestyle. If a person is symptomatic, identifying the specific type of the disease may assist treatment, such as the case with HER2 positive breast cancer (Jackisch, 2008).

2.3.5 How does Genetic Testing Work?

Genetic testing is performed by examining a tissue or fluid sample (such as blood, skin, bone marrow, or amniotic fluid) for abnormalities in the DNA, chromosomes or proteins. Depending on the test, the individual's DNA will be sequenced and specific regions will be compared with those same regions on a piece of DNA known to be disease free. In the case of other diseases, such as Down syndrome, a technique called karyotyping is used. This is a method of organising a person's chromosomes by size and number. In a person with Down syndrome, this process would reveal an extra copy of chromosome 21.

2.4 Areas where Genetic Determinism Arises: Genetic Modification.

2.4.1 What is Genetic Modification?

As it will be used here, the term genetic modification means "any change in the biology or psychology of a person which increases the chances of leading a good life (Savulescu, 2005)." This broad definition addresses concerns related to the use of the word "modification," which some authors believe is misleading. According to Darren Shickle, "The word enhancement is value laden and potentially misleading in the context of genetics. Dictionary definitions of enhance include 'increase in value,' 'improve,' 'appreciate,' and 'inflate' (Shickle, 2000, p.342)," but, of course, not all modifications will be seen as increasing the value of the individual. As A. David Kline explains, genetic modification is an attempt at "the improvement of normal human beings (Kline, 2007, p.16)."

Modification takes many forms and may include using technology to enhance the performance of athletes, or simply increasing the growth rate, memory or intelligence of the average human being. In addition to its many forms, an individual may pursue modification for a variety of reasons. Bayliss and Robert (2004, p.2) argue that these reasons may include, "to be in fashion; to improve performance; to gain a competitive advantage; to secure and exercise power; to promote and protect health and well-being;

to increase the lifespan; to assuage or even overcome existential angst; or, to meet the demands of justice." Naturally, an individual's motivation for seeking modification may affect whether we view their actions to be morally acceptable, or not.

In discussing genetic modification, there are two important distinctions we must make. The first is a distinction of moral significance and relates to the relationship between modification and therapy. Gene therapies are described as those which correct the abnormalities that cause disease—a disease being any "departure from species-typical normal functioning (Holtug, 1999) (some may find this definition of disease controversial). Genetic modification, on the other hand, is any technology that allows one to improve (by genetic means) one's normal functioning. Where gene therapy might be said to bring an individual's functions or abilities back to the baseline enjoyed by the rest of the population, modification takes an individual's functions or abilities beyond the normal baseline. Cosmetic surgery is a useful illustration of this distinction. A burn victim having a facial reconstruction in an attempt to restore their appearance would be considered a therapeutic (although not genetic) intervention, whereas an individual seeking a nose job and brow lift for aesthetic reasons would be considered modification.

A further distinction must be made between somatic (or adult) cell genetic engineering and germ (or embryonic) cell engineering. Somatic cells comprise all of the cells in the body (aside from reproductive cells) and are said to be fully differentiated which means they are fully mature and are not capable of developing in to any other cell type (Wilmut et al, 2002). For example, a neuron cannot further differentiate itself and become a cardiomyocyte or vice versa. Germ cells are reproductive cells (that is, they have the potential to unite with a germ cell of the opposite sex to create a new organism) and are said to be undifferentiated which means they have the potential to develop in to any type of cell present in the body. This makes sense because germ cells divide and multiply to become a fully functional organism (such as a human being) and therefore need the ability to differentiate in to whatever cell type is eventually necessary (Wylie, 1999). For example, at the germ cell stage, a particular germ cell must contain all of the information necessary to create a neuron and a cardiomyocyte (as well as many other cell types) because it may eventually become either cell type.

The distinction between somatic cell genetic engineering and germ cell engineering is important. Somatic cell genetic engineering (such as the deletion, insertion or other modifications to a gene) will affect only the individual subject. Modifications to germ cells, on the other hand, are inheritable (Kline, 2007). As a result, germ cell genetic engineering has implications for future generations and this necessitates special ethical considerations.

2.4.2 How does Genetic Modification Work?

The exact procedure will vary depending on the modification in question but a good example of the use of recombinant DNA technology for genetic modification is the creation of human growth hormone (HGH) using *Escherichia coli* as a bacterial host (Flodh, 1986). To isolate the HGH gene on the human chromosome, specific enzymes are used to "cut" away the rest of the chromosome, leaving only the target gene. The target gene is then inserted in to a plasmid vector, a molecular "vehicle" that can be used to transport genetic material in to another cell (Flodh, 1986). Vectors are used because the target gene (in this case, an HGH production gene) cannot be inserted directly in to the recipient organism (the bacteria). Once the gene has been inserted in to the plasmid vector, the vector is inserted in to the bacteria. Inside the bacterial cell, the recombinant DNA molecule (the plasmid vector and the newly inserted human insulin gene) is replicated along with the bacteria's DNA as part of normal cell division cycles (Flodh, 1986). These cell cycles produce multiple copies of the recombinant DNA molecule and permanently change the DNA of the host bacteria. Once the HGH gene is transcribed and translated by the bacteria, the bacteria will be directed to produce HGH which is then harvested for human use (Flodh, 1986).

Human growth hormone may be prescribed to children who are not growing at the expected rate (Gorman, 2005). But, the drug may also be taken by athletes in an effort to improve their performance (The Telegraph, 2013). For more on the distinction between therapy and enhancement, see chapter 3.

2.4.3 Is Genetic Modification Currently Happening?

Experiments to genetically enhance animals began in the late 80s and early 90s (Pursel et al, 1987; Pinkert et al, 1994; Sutrave et al, 1990) and focused on enhancing growth rate and muscle mass in pigs and cows. In 1999, a group led by Ya-Ping Tang from Princeton University reported they had successfully genetically enhanced mice to "exhibit superior ability in learning and memory in various behavioural tasks" (Tang et al, 1999). Named "Doogie" mice, after the TV show *Doogie Howser, M.D.*, the animals have been engineered to over express the NMDA receptor 2B which is involved in learning and memory formation. According to High (2005), it is now commonplace for diseases in mice to be treated with gene therapy but the practice is uncommon in larger animals as a result of the many obstacles "that hinder the translation strategies for design and delivery of gene therapy vectors...from studies in small to large animals (Baoutina et al, 2007)."

The first human trials began in the early 1990s (Baoutina et al, 2007) with a largely therapeutic focus, rather than a view towards modification. In 2000, the first success was reported by a group aiming to treat children with X-linked severe combined immunodeficiency (Baoutina et al, 2007). Bautina et al state that most clinical trials currently underway are in Phase I or II.

The use of genetic modification technology is being studied extensively in the field of sport science. This technology is intended to aid in enhancing the performance of athletes in a variety of ways such as increasing muscle mass and strength, improving the rate at which injuries heal, reducing pain as well as improving endurance. Many genes with the potential for enhancing performance have already been identified including EPO (endurance), IGF1 transcripts (increased muscle mass and strength), POMC (pain relief) and BMP (bone healing). Research in using genetic modification technologies in humans is very much underway but it should be noted that this still at the developmental stage and it may be many years before any of these products are available for use.

2.5 Areas where Genetic Determinism Arises: Cloning.

2.5.1 What is Cloning?

A clone is an organism, or a single cell, that is the exact replica of its "parent" cell. The cloned organism is created using a copy of the parent organism's genome. This means every cell in the clone contains the same DNA as the parent (Campbell et al, 2005).

2.5.2 How does Cloning Work?

Cloning falls in to two main categories: the type that uses embryonic cells and the type using adult (or somatic) cells. Cloning in either category involves removing the nucleus from an egg cell and replacing it with a donor nucleus from another cell. In true Frankenstein style, the egg and nucleus are fused using an electric shock (Stice et al, 1999). The fused cell begins dividing normally according to the directions given by the genome of the donor nucleus. Eventually, an embryo develops and the embryo is implanted in the uterus of a foster mother. The mother will progress through a normal pregnancy and give birth to a progeny that is a clone of the nuclear donor.

For many, cloning as an achievable scientific outcome only came to light in 1997 when scientists at the Roslin Institute in Scotland introduced the world to Dolly, the six-month old lamb who was cloned from the cell of an adult ewe (Wilmut et al, 2001). In fact, the technique of nuclear transfer was suggested by Spemann in 1938 as a method by which scientists could study cell differentiation (the process by which cells become more specialised) (Campbell et al, 2005). Spemann's technique was first used successfully in 1952 by Briggs and King who were experimenting with amphibians. Work continued with amphibians in to the 1960s and 1970s with Gurdon conducting extensive research. According to Campbell et al (2005) the development of this technology in mammals proceeded more slowly, with early research on rabbits proving unsuccessful (Bromhall, 1975). However, the 1980s saw the birth of the first cloned mice (McGrath and Solter, 1983) and this was followed by the birth of lambs, pigs and cattle (Prather et al, 1989; Willadsen, 1986; Robl et al, 1987).

Up to this point, all cloning was based on the use of embryonic cells but Campbell et al (2005) argue that the limitations of this method forced scientists to look for alternatives. Using embryonic cells to create cloned animals limited the number of potential offspring because there are very few cells available in the embryonic stage. It was this process of searching for an alternative technique that resulted in the birth of Dolly the sheep. According to Pennisi and Williams (1997), until Dolly's birth, a significant body of research indicated that although mammals could be cloned by fusing the nuclei of embryonic cells with an enucleated egg, adult cell types were already fully differentiated, that is, they were no longer capable of dividing and multiplying in to all of the different cell types necessary to develop a mammal. It was believed, according to Pennisi and Williams, that "chemical changes and structural modifications" meant adult cells were no longer capable of the necessary changes. As Diana Lutz (1997) argues, "...what Wilmut [the scientist responsible for breeding Dolly] had achieved was, most of all, the cloning of a differentiated cell" –as opposed to an undifferentiated, embryonic cell.

There are limitations, of course. For example, cloning puts an end to any further evolutionary developments. If a cow that produced an unusually large quantity of milk was cloned, it would not be possible to keep breeding from that cow and continue to see greater milk production. However, this discovery is important because—not only does it signals the potential to clone prizewinning animals or high milk producing cows—the knowledge that a fully differentiated cell can be converted back in to a cell capable of differentiation has numerous applications. DiBerardino (1997, p.386) explains: "It's possible to take a kidney carcinoma cell nucleus from a frog and inject it in to an enucleated egg and get a swimming tadpole. Not more tumour masses, but a swimming tadpole. So the molecular milieu of the [egg] cytoplasm has dictated that the normal genes turn on and the genes causing the [tumour] proliferation turn off." It is not difficult to see that in humans, this kind of regeneration has huge potential.

In the years since the creation of Dolly, there has been little success in the area of mammalian cloning (Check, 2007). Check (2007) reports that scientists are still in the dark, despite claims that after Dolly many other animals would soon be able to be cloned. While advances have been made in recent years, they have not lead to the major breakthroughs in the cloning process (Check, 2007).

2.6 How do these Technologies Relate to Genetic Determinism?

2.6.1 Genetic Testing

The basis of genetic testing is a belief in genetic determinism; we test for certain genetic markers on the basis that these markers indicate the potential development of a disease or disorder (it should be noted that an extensive discussion of this area should typically include mention of genetic heterogeneity, penetrance and expressivity but this is beyond the scope of that is necessary here). The question this assumption poses for this thesis is whether we are making a legitimate prediction and how certain we can ever be of the test results. Further, this thesis seeks to demonstrate the inadequacy of genetic determinism. Therefore, what implications might this have for anyone undergoing a genetic test in the future?

2.6.2 Genetic Modification

As with genetic testing, the entire basis of genetic modification is a belief in genetic determinism. If we did not believe in genetic determinism, there would be no point in trying to make genetic modifications. As far as this thesis is concerned, the assumptions made about genetic determinism in relation to genetic modification raise questions. Firstly, is it ethically acceptable to seek genetic modifications when we cannot be certain of the outcome? In most cases, the answer may be 'yes', depending on the risks and benefits associated with proceeding with the genetic modification versus not proceeding. Which brings us to the crux of the dilemma: if we cannot be certain of the outcome, what are the real world implications of this for an individual considering this technology? We may in fact be 90% certain of the outcome but where do we draw the line? We must tread with caution into this future of new genetic technologies, but at the same time, we must not needlessly delay potentially life-saving progress.

2.6.3 Cloning

Copying an organism's entire genome and using it to develop another, separate organism appears to be the most straight forward example of genetic determinism. In principle, it seems clear that the parent organism's genome determines the resulting organism's genome in a very linear way. The main issue raised by cloning for this thesis is whether cloning is even a realistic possibility, given the inadequacy of genetic determinism. If genetic determinism is indeed a fallacy and it is not possible to produce a true clone, should we continue to pursue this technology?

3 Current Discussion

The aim of this chapter is to review and critique the relevant literature with a view to understanding what is currently being written about genetic determinism and the nature versus nurture debate.

3.1 Interdependence between Nature and Nurture

Broadly speaking, all of the authors writing in this area argue strongly that neither our biology nor social influences are sole determinants of who and what we become. The authors' own background, usually biological or social sciences, generally determines whether they believe biological or social influences to be a slightly stronger determinant.

The exception here may be Steven Rose, a neuroscientist who has held roles in biology and neuroscience, as well as the directorship of the Brain and Behaviour Research Group at The Open University in the United Kingdom. Rose (1995) advocates strongly for biological scientists to get away from purely reductionist interpretations of advances in science. Rose argues that we must consider both biological and social factors in order to explain "the phenomena of human existence and experience."

Further, in specific reference to neuroscience, Rose argues the strengthening of our scientific understanding serves to reinforce the reductionist position. This position, he argues, is further supported by what he calls "a social and political environment conducive to such claims." He states it is much easier to believe that people are violent or depressed because of something in their genetic makeup which is beyond their control. If an individual is violent or depressed because of social problems, society would be required to come up with social solutions.

In a thoughtful and articulate manner, Rose identifies a myriad of issues related to purely reductionist interpretations of advances in neuroscience. He believes neurogenetic determinism, in particular, is based on a system of faulty reductive reasoning which includes a belief in the statistical bell curve, misplaced causality and a false believe in the absolute separation of biological and social causes. More important for this thesis than any of the individual steps he identifies in the reductive process is

the way Rose frames the central issue in this whole debate. He argues; "And the issue at stake is not the formal philosophical one, but the question of the appropriate level of organization of matter at which to seek causally effective determinants of the behaviour of individuals and societies (Rose, 1995, p.380." This critical issue is the central question of my thesis and Rose uses this question to frame, and give context to, the rest of his argument. By identifying multiple issues with the system of reductive reasoning and framing the central question of his article so clearly, he positions himself as somewhat of an authority in the field because, surely, an unqualified individual could not have described these issues so eloquently. It comes then as quite a disappointment that he fails to offer solutions to any of the problems he has been so perceptive in identifying. Rose offers no view on the "appropriate level of organization of matter" at which to start making determinist predictions, he merely identifies that this is the most important issue and states that the solution is some combination of biological and social factors. Rose appears to agree with the argument I am putting forward that no discussion of determinism is complete without acknowledging that there are different levels of organisation of matter and making some attempt at deciding how important each of these levels is in the overall scheme. In the end, Rose's key message is that we must get away from the traditional notion of the unidirectional nature of causality (that is, genotype causes phenotype) and appreciate a more integrated view of biological and social factors.

Writing in the British Journal of Psychiatry in 2001, Rose expands on his view that the line between biology and sociology is blurred. While his position in the 1995 article considered only genetic and environmental causes, the 2001 piece suggests the situation is far more complicated. Perhaps in reference to what he suggested is the central question in the 1995 article, Rose argues there is no single level of human existence that provides the complete picture. He states that we are part of a complex ecosystem – "molecular, developmental and evolutionary" – and none of these explanations can be collapsed in to any of the others to provide a single account. Once again, Rose acknowledges that there are many levels of organisation that are important in describing who we are but he goes no further than to simply state that there is interplay between these different levels. He makes reference to the molecular level but does not make any comment on the significance of the molecular level of organisation, let alone the significant events that might occur there.

Further, Rose argues human development does not fit neatly into categories of nature or nurture; rather it is an "autopoeitic process, shaped by the interplay of specificity and plasticity (2001, p.s6)." This sentiment – that nature affects nurture and nurture affects nature – is reflected elsewhere in the literature (see Cooper and Zubek (1958), Liu and Dorio (2000) and Francis, Liu and Dorio (1999)). This interplay, Rose argues, is the reason we cannot predict (or determine) the future. Rose states, "People move from unfavourable conditions; we absorb aspects of our environment and in doing so we constantly change our environment (2001, p.s6)." The circular nature of the relationship between our genes and our environment means we cannot predict future patterns. He contends that we are continually responding to our environment and "in doing so, changing the environment both for themselves and others" which means we cannot track "a continually moving and inherently unpredictable target." Rose argues that our futures are "radically unpredictable" and this means we have the power to determine our own futures. In this regard, it appears Rose would agree with my hypothesis – that the concept of genetic determinism is a fallacy. Where we appear to disagree is the relative contributions of each of the factors identified: molecular, genetic, environmental, personal and historic. The contribution of molecular factors has received barely a mention which implies it is not considered as important, or influential, as genetic and environmental factors. Rose alludes to the molecular but gives this level of organisation of matter no further attention or consideration.

The interdependent relationship between nature and nurture, genes and the environment, is well established and well accepted. Two brilliant examples that illustrate this relationship have come out of the work of Cooper and Zubek (1958), Liu and Dorio (2000) and Francis, Liu and Dorio (1999). Wahlsten and Gottleib (1997) describe Cooper and Zubek's groundbreaking work with selectively bred "maze bright" and "maze dull" mice. The mice were bred at McGill University in the 1950s and over some generations, two different genetic strains emerged. The maze bright mice, as the name implies, were particularly adept at negotiating mazes while the maze dull mice performed poorly. In their study, Cooper and Zubek raised the young of the bright and dull mice on either impoverished, enriched or a controlled diet. As adults, the mice were tested in a maze and stunning results emerged. Mice raised on an impoverished diet – both bright and dull – performed poorly in the test compared with the bright and

dull mice raised on an enriched diet. Further, the genetic advantages each mouse started out with were levelled by their diets. Bright and dull mice on the impoverished diet performed equally poorly. Similarly, bright and dull mice on the enriched diet performed equally well. In other words, the performance of genetically similar mice was dramatically affected by diet. As expected, bright mice in the control group performed better than their genetically dull counterparts. This study demonstrates convincingly the interdependence of genes and the environment.

The work of Liu, Diorio and Francis (Lui and Dorio (2000); Francis, Lui and Dorio (1999)) is similarly compelling. In their experiments, the trio used BALB/c and C57 mice. BALB/c mice are known to be "highly fearful and maze dull in the extreme" (Meaney, 2002, p.57). In contrast, C57 mice show "increased...responses to stress..., are hyperactive and show profound learning and memory deficits" (Meaney, 2002, p. 57). The major difference between the two species, however, relates to how they raise their young. On average, a C57 mouse will lick and groom her offspring significantly more often than a BALB/c mother. The frequency with which a mother licks and grooms her offspring contributes to the mouse's intellectual capacity as an adult. Not unlike human babies, perhaps, the more post-natal licking and grooming a mouse receives, the more emotionally stable and intelligent it will become. When the group switched BALB/c and C57 babies to a mother from the other species at birth, as adults, the mice behaved more like their surrogate mother than their genetic mother. As observed by Meaney, "Dramatic alterations in the phenotype of the BALB/c mouse occur if it is reared in the care of a C57 mother (2002, p.58)." If the offspring of mothers who lick and groom infrequently are raised by mothers who lick and groom frequently, "there is a reversal in patterns of neural synapse formation and behaviour" (Meaney, 2002, p.58). These findings support those by Cooper and Zubek. Despite the genetic predisposition of all four species of mice involved, environmental factors altered the way the mice behaved later in life.

These studies provide striking evidence of the interdependence of genes and the environment in mouse development, which may reflect human development. Of course, while these studies are compelling, there is no suggestion by any of the authors that there may well be other factors involved. The dilemma is very much presented as scientists trying to unravel the relationship between genes and the environment, as if an

absolute understanding of these two factors will entirely explain human behaviour. In fact, in explaining the implications of the nature and nurture debate for heath science researchers, Meaney (2002) argues that the reward for understanding the relevant genes and how the environment influences those and other genes, "will be the ability to assemble an integrated explanation of individual differences in vulnerability/resistance to disease than spans *from the level of gene activity to socioeconomic factors* [my italics] (2002, p.60)." Meaney speaks of the necessity for an integrated, multidisciplinary approach to understanding human development but "multidisciplinary" in this case merely means the inclusion of genetic and social scientists. An integrated, multidisciplinary approach is generally the best way to solve any problem but consulting only genetic and social scientists is bi-disciplinary, at best. A truly multidisciplinary approach would lead to the consideration of factors other than nature or nurture. It might include, for example, the search for a third option.

While it seems most scientists have known for many years that explaining human behaviour would require some consideration of both genes and the environment, there are, of course, scientists who seem to be surprised by this fact. Writing a few years after the release of the first draft of the human genome sequence in 2001, Paul Silverman was surprised to be writing that "the gene may not be central to phenotype at all..." (Silverman, 2004, p.32). Silverman contends that funding was secured for the human genome project (HGP) based on a belief in the "deterministic nature of the gene (p.32)" and describes a number of scientific factors that "confound the gene's predictive value (p.32)". These include, he believes, alternative splicing, micro-RNA, tissue and cytoplasmic factors. In the briefest of references to the possibility of influential factors outside of genes and the environment, Silverman considers that the factors which confound the gene's predictive value "may be affected by a variety...*of unpredictable protein-interaction events* [my italics] (p.32)." Despite his acknowledgement that the environment may play a role in development, Silverman continues to argue against genetic determinism on the basis of our genetic complexity – not because of an interaction between genes and the environment. He states that the current model of DNA transcription and translation is too simplistic and what is required is a model that takes in to account the many post-transcriptional and post-translational modifications. He alludes to the importance of the environment – diet, exercise, antioxidants – and suggests that other scientists working in the area of molecular biology are reluctant to

transition to a multidisciplinary way of thinking. Yet, in a subtle way, his own writing suggests he too belongs in this camp. He mentions environmental factors, but only in passing and concludes that the solution is simply a better understanding of the science. He mentions "unorganized, stochastic events (p.33)" but reasons that once we have a deeper scientific understanding we will be able to explain these events (Silverman, 2004). It seems greater reductionism is Silverman's solution.

What, then, of a third option? A possible third option has been suggested by some and that is free will. Daniel Dennett (2003) argues that our free will enables us to ameliorate any of our genetic traits we do not like through education and culture. As an example, he cites his myopia which he has chosen to address by wearing glasses. He argues that people with certain genetic diseases can often live their whole lives without experiencing symptoms by following a particular diet or taking medication. Dennett argues strongly that determinism is not the same as inevitability; what is important is whether an undesirable event or effect can be avoided by taking a different course of action. For example, if a child is rejected by its mother in its first year of life, there is a greater chance of the child committing a violent crime later in life (Dennett, 2003). But, Dennett argues, it is not determined that the individual will commit a violent act because the individual can choose a different path.

Dennett raises an interesting point, but free will does not emerge out of nowhere. As with all aspects of who we are as humans, our ability to display and act on our free will – like our ability to be confident or aggressive – must come from somewhere, namely, some combination of our genes and environment (and, possibly, a third factor). In his example of the child who is rejected by his or her mother early in life, it is theoretically possible for the child to choose a different path – as it is theoretically possible for all of us to be more confident or aggressive – but the child's genetic makeup and environmental factors are likely to determine whether the child is capable of making this decision. A child raised in a poor environment may be unlikely to know that there are other options for him or her in their lifetime and will continue to follow the path their family has set down for them. This is, after all, the basis of what is commonly referred to as the "cycle of violence" (Maxfield & Spatz Widom, 1996). Even if the child is aware he or she possess the freedom to make their own choices, whether they can act on that freedom is a function of their genetic and environmental makeup.

Similarly, the child may be aware he or she has the ability to be a more confident individual, but this does not mean the child is capable of becoming more confident. Free will cannot be considered an independent or overriding factor in the arguments against genetic or environmental determinism because it is a function of our genetics and environment (and, of course, the third option!).

Dennett (2003) does not support the notion of there being a third option besides free will. He suggests chance or luck may play a role as both are "all around us in the causeless coin-flipping of our noisy world, automatically filling in the gaps of specification left unfixed by our genes and unfixed by salient causes in our environment (p. B9)." But he goes on to say that this is "pseudo-random, of course (p. B9)" which heavily implies that he believes it is not actually randomness and one day, following further scientific reductionism, we will understand the randomness.

3.2 Genetic testing

A persistent fear in the literature relating to genetic testing is that genetic tests can deliver certainty; others argue that the tests may be able to deliver certainty but it is our ability to interpret the data lags behind. The fear is, writes Lucassen (2012), that genetic testing will lead to the oversimplification of disease processes—that is, talk of 'having the gene' for a particular disease or condition—when in fact the risk of developing the disease is small "or only exists in the presence of another variant or group variants, perhaps in turn in combination with environmental exposures during fetal or adult life (p.196)." Although research is progressing well, Lucassen writes, and providing insight in to disease processes, "their predictive powers are not always as good as predicted (p.196)." As an example, Lucassen cites a study by Paynter et al (2010) which found that examining more than 100 variants related to coronary heart disease had no greater predictive power than a detailed assessment of family history. Lucassen cautions, "Interpreting the output of...genomes with millions or billions of base pairs requires complex bioinformatic analysis (p.196)," and we do not currently possess these skills.

Lucassen's assumption (or optimism) that we do not yet understand, or do not yet have the right technology, is common and this assumption is based, somewhat, on a belief in

genetic determinism. The assumption is that there is a rhyme and reason to human biology; that human biology is based on a logical system; that human biology is fundamentally understandable—all we need is the right technology. This belief is pervasive and is an example of the way in which genetic determinism indirectly underpins so much of the thinking presented in the literature. Asked directly, it is unlikely Lucassen would say she believes in genetic determinism—most scientists know better—despite this, still she argues from a position of genetic determinism, that is to say, her argument relies on the principle of genetic determinism being fact. This kind of reasoning is why the fallacy of genetic determinism persists.

In a similar vein, Lippman (1999) also fears the oversimplification of disease processes. She writes that genetics is increasingly being seen as "*the* way to reveal and explain health and disease, normality and abnormality (p.47)." Pre-natal genetic testing, she avers, is a prime example of using high-tech resources to solve health problems, at the expense of low-tech approaches—such as social, environmental and political measures—which would have greater benefit for the underprivileged. Social, environmental and political contributors to health are disregarded in favour of a more individualised approach to health. This view is supported by Rothman who argues that focusing on the genetic components of disease—and thereby individualising medicine—fails to take in to account "the role of the social and political world in causing disease (Rothman 2001, P.104)."

A further concern raised by Rothman (2001) is that genetic testing may be used "in attempts to gain control over our future through manipulating human procreation... (p.104)" To some extent—perhaps even a large extent—Rothman may be right in this line of thinking. Embryos can already undergo screening for certain diseases and those embryos with the disease are likely to be discarded in favour of healthier ones. In some cases, couples are selecting embryos on the basis of sex to eliminate the chance of sex-linked diseases or to "balance" their families. However, as with Lucassen's argument, this idea is fundamentally based on the factual nature of the principle of genetic determinism. If genetic determinism is a fallacy, we could not use genetics to "attempt to gain control over our future (p.104)." Rothman appears to be imagining a future replete with "designer babies" but even our current knowledge of mechanisms of inheritance suggests this future is unrealistic. Add to this a possible third option, in

addition to nature and nurture (to be discussed in the next chapter), and it seems highly unlikely that we will ever have the technology to "manipulate human procreation" in the monumental way Rothman alludes to.

The media representation of the predictive power of genetic testing appears frequently in the literature. Arnason and Hjörleifsson (2007) report on a wide range of studies by others and say that genetic testing is generally portrayed as a positive move because "the technologies hold great potential for alleviating and preventing suffering and are likely to yield financial benefits (p.423)." These authors note that a range of concerns is reported, such as playing God; discrimination; and, "revoking eugenic atrocities of the past (p.423)." But, they state, journalists are not likely to question "research findings or predictions about benefits (p.423)," which ultimately feeds any concerns they or the public may have. They argue that this failure by journalists to question the evidence allows scientists to claim that they are conducting the "right" kind of research, subject to appropriate ethical scrutiny and that their research will be beneficial. Lynch and colleagues (2011) also examine the media coverage of genetic testing technologies. They report that consumers get their information about genetic tests from "the company offering the test, the internet and the news media (p.487)." This finding reinforces the importance of the accuracy of media coverage because it forms a large part of public education. Lynch et al found that the media presentation of the scientific concepts involved has a "significant impact on public understanding of science (p.487)." They report that "the main themes and frameworks presented in the media become the building blocks on which the public constructs their own understanding of the scientific issues (p.487)." Other researchers such as Clayton (2003); Hubbard and Wald (1993); Nelkin and Lindee (1995); Silva (2005); and, McCabe and McCabe (2008) have found that the media contribute significantly to the public's belief in genetic determinism and the view that our genetic makeup is authoritative. This view of genetics creates fear of genetic discrimination (Lynch et al (2011).

Concerns about genetic discrimination bring health insurance companies in to the picture. Lynch et al (2011) refer to concerns about individuals being able to keep their genetic information private, while Hübner (2006) writes about the issues individual's may face once their genetic information is known. He writes that individuals with "unfavourable genetic structures (p.44)" may encounter difficulty in obtaining

affordable health insurance coverage. He also examines the counter-argument—that individuals revealed to have "favourable" genetic structures in genetic tests may be able to secure insurance "under more advantageous conditions (p.44)."

Of course, the issue here is a belief by medical insurance companies in genetic determinism. What is unique, however, is that this may be one of the few instances where there is financial motivation to sustain a belief in outdated science. In most cases, such as where individuals are concerned about their own health or scientists are interpreting the results of a study, a belief in genetic determinism is detrimental because it leads to false conclusions. Those conclusions generally involve an assumption that gene X leads to disease X when in fact, gene X may only be a contributor or gene X may be inactive unless a certain environmental stimulus is encountered. For medical insurance companies, however, believing that the presence of gene X leads to disease X is grounds for refusing someone coverage or, better yet, charging them greater premiums than someone without gene X. Not only is this line of thinking beneficial to medical insurance companies for their own bottom line, but it is beneficial to them that the public continues to buy in to this thinking. For similar reasons, it is in the interests of companies making genetic tests—whether they be at home tests or those only available through clinicians—that the public continues to believe in genetic determinism. Anyone who stands to profit from genetic testing (or genetic modification or cloning) has strong motivation to continue to perpetuate the fallacy of genetic determinism.

To reiterate, concerns presented in the literature relating to genetic testing and genetic determinism centre around five main areas: the oversimplification of disease processes; genes being portrayed as *the* way to understand who we are; playing God and controlling the future; the media's role; and, those with the financial motivation to continue a belief in bad science. The first three of these concerns are all based on a belief in strong genetic determinism. They all fail to take in to account the role of the environment and, as we shall see in the following chapter, they fail to recognise that genetic determinism is not straight forward. Once it is clear that genetic determinism is a myth, these three areas of concern largely take care of themselves.

As for the fourth concern, the media's role, it is clear that further education is needed. Journalists need to question the scientific evidence presented before writing an article or preparing a news piece. Public reliance on news media for their understanding of science and genetic technologies is well established (Lynch et al, 2001). It is, therefore, critical that the fallacy of genetic determinism becomes more widely known.

Companies with a financial motivation to support a belief in genetic determinism present quite a challenge. It may be that this is one corner where a convenient view of genetic determinism reigns, while the rest of the view catches up to reality. In time, as a strongly deterministic view becomes less acceptable, it is likely that these companies—particularly medical insurance companies—will have to change their practices.

3.3 Genetic modification

For the authors writing in this area, the concerns related to the genetic modification of humans are related to ensuring equal access to the technology, its effect on human evolution and the philosophical debate over "playing God." Among these concerns are fears related to the implications of genetic determinism.

Resnik and Vorhaus (2006) describe genetic modification as the "process of intentionally altering human genes for the purpose of producing offspring with those genetic changes (p.2)." Or, to paraphrase, genetic modification can be thought of as the intentional alteration of human genes for the purposes of creating designer babies. Certainly the first part of this definition ("...intentionally altering...") is widely supported in the literature, however, the later part of their definition ("...for the purpose of producing offspring...") is not universal. In many cases, genetic modification is described with the intention of helping the individual whose genes are being modified, for example, when discussing enhancing professional athletes to improve their performance.

An important point raised in the literature is the need to distinguish between genetic enhancement and genetic therapy. This distinction is morally relevant for some as "there may be a duty to treat or cure disease to relieve attendant suffering, while a

similar obligation to improve lives that are being lived within the normal range of human functioning does not exist (Rosoff, 2012, p.164).” For example, should laser eye surgery for the repair of common myopia be considered treating an individual’s poor eyesight so they may enjoy the same vision as everyone else, or is that individual being enhanced beyond their natural state? In this case, it seems relatively clear that this individual is simply being treated for a defect in their genetic makeup. It seems difficult to argue that this individual is being enhanced where they are merely being given the same standard of vision the rest of us enjoy. But it is not hard to wander in to murky territory. Consider an individual with a low IQ. If it became possible to improve IQ through genetic enhancement, could that ever be considered treatment? Logically, you might reason that this is an obvious case of enhancement because having a low IQ, while unfortunate, is not a disease or deformity.

But how is having a low IQ all that different from myopia? An individual with poor eyesight should be given the option of enjoying the same level of vision as the rest of us, but could the same argument not be applied to IQ? Having a low IQ is not normal and it limits an individual’s life options as much as poor vision. Gordon (1999) argues that it is clear that the concern over genetic enhancement relates to the “improvement of functions that without intervention would be considered entirely normal (p.2023).” The difficulty with this position is temporal. The technologies we are debating and discussing are, largely, not yet available, particularly where genetic enhancement is concerned. It will be years—perhaps decades—before these technologies are used on people. But, writers like Gordon are making their assessments using today’s definition of normal. It is not currently abnormal for an individual to have a low IQ because there is a spectrum of intelligence so naturally some people will fall at the lower end. However, in 20 years’ time, will the distribution along this spectrum be the same? At present it is accepted that some people will have a low IQ, but what if this becomes abnormal in future? If this eventuates, Gordon’s (1999) argument, that public concern over genetic modification “does not relate to improvement of traits for alleviation of deficiencies...”, becomes weakened because a low IQ will be considered a deficiency to be alleviated.

The focus of this thesis is not the distinction between genetic enhancement and genetic therapy. The line between enhancement and therapy, as we have seen, can be difficult

to define and may not hold the moral significance it is often assigned (Resnik, 2000). To this point, Darren Shickle writes: "The word 'enhancement' is value laden and potentially misleading in the context of genetics. Perhaps the term 'genetic enhancement' would be better replaced with a more neutral term such as 'genetic manipulation' to reflect the fact that the consequences of as yet largely untried technology may be beneficial, balanced or harmful (2000, (p.342))." Consequently, to avoid becoming overly focused on this argument, and instead, concentrate on the arguments around genetic determinism—which apply equally to both genetic enhancement and therapy—the term "genetic modification" will be used.

A further concern relates to the distinction between modifications that are genetically heritable and those which are not (Gordon, 1999). This issue relates very closely to the issue of genetic determinism because if our genes are a significant contributor to who we become, any modification to those genes has the potential to affect, not just the individual in question, but also future generations. Taken to its extreme, this may even raise issues for human evolution, although Gordon (1999) argues that the impact of genetic modification on human evolution would be negligible.

A further application of genetic modifications that are potentially heritable is in professional sport. The most likely uses of these technologies in sport are to increase endurance or muscle capacity by targeting growth factors such as IGF-1, recombinant EO and the ACE gene (Miah, 2006). Unsurprisingly, perhaps, the concerns related to the use of genetic modification in sport are largely the same as concerns for its use elsewhere. Perhaps most pressing for professional athletes, however, is the need to be certain that any modifications produce the intended outcomes (Culbertson, 2009). How disastrous would it be if an otherwise perfectly healthy athlete underwent a modification procedure, say to increase muscle capacity, only to be worse off afterwards as a result of reduced muscle capacity, or even permanent muscle damage? The question of human biological systems being non-linear appears here, too. As McFee (2000) argues, "being slightly adrift in one's grasp of initial conditions can lead to being hugely wrong in one's predictions (p.155)." Culbertson (2009) suggests that if this is the case, namely, that biological systems are nonlinear, it would be unwise "to make changes because we could have absolutely no idea of the outcome because of the degree to which our calculations can be inaccurate in nonlinear equations (p.144)." He

adds that genetic modifications at the germ-line level "do not fit a physics-based model, so any significant degree of predictability is impossible (p.144)" and furthering our understanding of the science involved may not increase predictability.

Resnik and Vorhaus argue that strong genetic determinism (which contends that a particular gene leads to the development of a particular phenotype in greater than 95% of cases) is rare and they cite several reasons for this. Firstly, they acknowledge the role environment plays in genetic expression and describe the relationship between the two as complex and interdependent. Further, they argue, most phenotypes are caused by more than one gene; most likely dozens, or perhaps even hundreds, of genes. In addition, the pair point out that the developmental process itself plays a significant role in gene expression, that is, how the organism actually converts genetic information into phenotype. In a brief reference to the subject of this thesis—the possible third option—Resnik and Vorhaus argue that the actual patterns and processes of development (that is, the order in which tasks are carried out, the frequency at which certain tasks are carried out, the quantities of certain enzymes) have an impact on gene expression. This is why, according to them, twins can have identical genomes and be raised in near identical environments and still have subtle variations in "hair, skin pigmentation, facial shape, fingerprints and dental impressions. Even among cloned animals there may also be phenotypic differences (Resnik and Vorhaus, 2006, p.4)"

Rosoff (2012) continues in this vein, arguing that strong genetic determinism is rare because of issues with reification—in other words, actually describing the trait in question in genetic terms—and epigenetics. It makes sense that in order to genetically modify an individual in order to increase their intelligence or decrease aggression we must be able to adequately define the trait in question; the trait must be translated in to the scientific language of genes, proteins and enzymes. According to Rosoff, we are no better off today than we were more than a century ago in terms of our ability to define or describe complex traits such as intelligence or aggression. For him, "Even if one is confident of what a trait is...codifying that knowledge in to an unambiguous and coherent 'thing' is another matter altogether, mainly because it needs to be measured in a clear, repetitive manner by a variety of different techniques and methods. To do so requires agreement on a physical definition (p.166)." He considers that our inability to define traits may be an insurmountable barrier to genetic modification. Rosoff points to

the enormous effort researchers have put in to understanding the genetic causes of diseases such as cancer, diabetes and heart disease in the last several years. For him these are all diseases we can define in a "straightforward, understandable, and reproducible manner (p.172)," and yet we know very little about the genes involved in their aetiology. How then, do we expect to fare with traits such as intelligence or aggression which are much harder to define?

A further concern identified by Resnik and Vorhaus (2006) is that despite the reality that strong genetic determinism is rare, "popular culture, the media and politicians (p.4)" continue to propagate the myth and "journalists continue to speak of 'genes for obesity,' 'genes for alcoholism,' and 'cancer genes,' as if genes exist that, once discovered, will give individuals the ability to simply 'shut off' obesity, alcoholism, or cancer with a simple snip to the genome." Hindmarsh (2000) supports this view and adds that research in this area has been positioned as a technology which assumes it is a predictable, 'closed system.' Further to this, Birch (2005) argues that genetic modification is presented "as a *fait accompli* (p.17)" and the word 'genes' is taken to mean "predictor of phenotype and behaviour (p.17)."

3.3.1 Genetic Modification—Freedom, Giftedness and Authenticity

Resnik and Vorhaus (2006) describe a number of other, more philosophical, objections to genetic modification on the basis that it interferes with the future of the individual being modified. These include the argument from freedom (Kass, 1997); the argument from giftedness; and, the argument from authenticity.

The argument from *freedom* (or *autonomy*) suggests that genetic modification interferes with the modified individual's ability to make free choices in relation to the modified trait. This is because the person choosing which genes to modify is in control and, if genetic determinism holds, the modified individual will have no choice but to follow the life path chosen by the modifier. Kass (1997) argues strongly that controlling a child's genotype gives one tyrannical control to shape "their future according to one's will (p.5)." For example, creating a child with athletic talents is the equivalent of condemning the child to life as an athlete, irrespective of what the child may have

otherwise chosen for themselves. As Resnik and Vorhaus (2006) have already shown, this type of strong genetic (and behavioural) determinism is very rare. To truly shape a child in to some ideal image "will require a lifelong commitment (p.6)" because there are many, many factors involved—not just genes. For others, the freedom argument relates more to the modifier limiting the modified individual's future life choices. This is very closely linked to the previous argument (that the modifier is controlling the modified individual), however, this argument can be taken to be more from the modified individual's perspective.

Rather than suggesting genetic and behavioural determinism (that is, the child must become an athlete), the life choices argument views the child's other options as limited (that is, the child is now less likely to choose to become a musician). Of course, this is still the argument from freedom, albeit, from a slightly different perspective. As such, Resnik and Vorhaus' (2006) observations about the rarity of strong genetic determinism and the multitude of other developmental factors involved are relevant here, too. Michael Sandel (2004) also argues against the freedom objection but from a philosophical, rather than scientific perspective. Sandel argues that none of us chooses our genetic endowment. A child does not choose his or her genetic makeup, so why does it matter—within the context of this line of thinking—whether the child's genes came from his parents or genetic modification? A child's autonomy is neither increased nor decreased whether his genes come from genetic modification or the genetic lottery.

The argument against genetic modification from *giftedness* views modification as wrong because it treats children as commodities. Writers such as Sandel (2004) and Kass (1997) argue that parents are trying to "play God", designing their children to be exactly as they want them to be, rather than appreciating the miracle of creation and birth. Sandel writes of the hubris of parents in their "drive to mastery (p.51)" over their children and believes this drive causes parents to miss "an appreciation of the gifted character of human powers and achievements." For him, life is a gift and genetic modification fails to respect and appreciate that gift. As such, genetic modification is a moral failing. Parents should not, the argument goes, be trying to control the traits of their children; rather, they should appreciate their children for who they are. Of course, this is yet another example of an objection to genetic modification which assumes strong genetic determinism. Images portrayed in the media of parents being able to go

to a "supermarket" of sorts and select the traits they would like for their children are greatly flawed. There are many limitations to this model. Resnik and Vorhaus (2006) argue that the relationship between genotype and phenotype exhibits limited causality. According to them, our lack of "scientific or technological mastery (p.6)" means "our ability to exert control via genetic modification will *necessarily* fall far short of anything that could be construed as mastery (p.6)."

The argument from *authenticity* contends that if an individual is genetically modified, these modifications are seen as an advantage and will undermine the authenticity of any of the individual's achievements—the achievements are not the individual's own, they are the product of genetic modification (Resnik and Vorhaus, 2006). For Sandel (2004), this argument is flawed because without genetic modifications there are significant differences between the genetic endowments of people that lead to remarkable differences in ability. When watching a brilliant musician or athlete perform, we do not think less of their performance because of their obvious genetic gifts so why, Sandel argues, would we view the achievements of the individual any differently if they were, in part, the product of genetic modification? It appears to be that it is the issue of authenticity that creates tension when discussion genetic modification in relation to athletes (Miah, 2006). Our problem, Miah argues, is "the demise of the natural human" and our ambivalence over this demise.

However, this argument is also flawed because it fails to take in to account the hard work the individual must put in to develop their skill. In his 2011 book, *Outliers*, Gladwell argues that it takes 10,000 hours of dedicated practice at one's art in order to truly master the skill. Even if strong genetic determinism were possible, an individual could not sit passively by and expect to become a talented musician or athlete based on genes alone. The same comparison could be made about individuals who work to become more intelligent, through challenging work or study, and those who may be similarly intellectually gifted but choose to work in manual labour. From a genetic perspective, both individuals may start in the same place but only one of them works on their "craft", so to speak. With so many complex factors involved in the development of who we are as individuals, a genetically modified individual will never be able to rely on genes alone.

3.3.2 Genetic Modification—Epigenetics

Epigenetics—ways other than DNA, chromosomes or the environment by which phenotype can be influenced—is, Rosoff (2012) argues, another reason why strong genetic determinism is unlikely. Epigenetic effects are the post-transcriptional and post-translational modifications that do not alter the DNA sequence, but alter the likelihood of the gene being activated. Rosoff concedes there is still much to be discovered about the capabilities of epigenetics, but what we currently know "...only reveals the ... challenges facing anyone who would try for an end run around the complex genetic engineering that would be required for enhancements to succeed." Even if those attempting genetic modification were successful in locating the appropriate target genes and make the necessary changes, there is no reason to believe that the modified genes themselves would not be the subject of epigenetic revision. Rosoff believes there are those who would argue that epigenomics simply represents our incomplete knowledge of human biology and that once we have a complete understanding we will be able to explain everything we would currently refer to as the result of epigenetic factors. He concedes this position may be true, but believes this assumption is based on the false belief that biological systems are linear when in fact biological systems are nonlinear which means there may be a disproportionate relationship between cause and effect. This nonlinear relationship means small causes may have large effects and vice versa, all of which makes it very hard to believe, Rosoff states, that we will ever have a complete understanding of human biology. Even more challenging, he argues, is the reality that epigenetic factors appear to have a mind of their own, acting completely under their own control—that is, if they are under any control at all.

Following the insight of these authors, particularly Resnik and Vorhaus (2006), Rosoff (2012), and, Culbertson (2009), the question becomes: is the third option just epigenetics, an area that already has a reasonable definition and a small, but growing, body of research to support it? The answer, unfortunately, is maybe. Like everything in this area, the waters are murky and it is difficult to tell exactly how far, or, across how many organisational levels of an organism, the definition of epigenetics spans. In the literature reviewed above, the word 'epigenetics' is used broadly to refer to anything affecting development that is not related to genes or the environment but a clear explanation of the boundaries of the concept is not given. The vague sense in which

this term is described may just be a symptom of our vague understanding of this area of science.

Of all of the articles surveyed, Rosoff's (2012) is the most in-depth in terms of defining epigenetics. By looking critically at his article, it is possible to make a sound judgement call about whether epigenetics really is the third option that is the subject of this thesis. To quote Rosoff, "epigenetic input appears to be under completely independent, if not autonomous, control (in truth, it acts as if it is not under any control whatsoever) (p.173)" but he does not specifically mention stochasticity nor does he cite any sources that refer to stochasticity. Similarly, he mentions the nonlinear nature of biological systems and the disproportionate relationship between cause and effect, but does not refer to emergence; in fact, he does not even use the word 'emerge' as a verb, for example, to describe the way a disproportionate relationship between cause and effect might cause unexpected traits to emerge. Rosoff's article includes 83 references and would, therefore, be considered thoroughly referenced by virtually every definition. In light of the obvious depth of research undertaken to prepare his article, it seems likely that Rosoff will have come across these terms and concepts. It may be that Rosoff does not like or agree with the terms chaos or emergence, but when writing in this field it seems remiss that he would not at least make reference to the terms, if appropriate, and explain why he is choosing not to use them. Therefore, we must assume, that if Rosoff wanted to specifically refer to stochasticity or emergence he would have. Through an entirely reductive process, we can assume based on Rosoff's definition of epigenetics and his exclusion of references to chaos and emergence, that epigenetics is not the third option. It may be *a third option* to be considered alongside nature and nurture in its own right, but epigenetics is not the third option this thesis serves to highlight.

Genetic modification raises concerns on many fronts—the line between enhancement and therapy; heritability of modifications; uses in professional sport; media involvement; arguments from freedom, giftedness and authenticity—but, most of these are based on antiquated notions of genetic determinism. Once we account for the fact that strong genetic determinism is rare, many of these concerns lose their steam, so to speak.

As with genetic testing, what remains an issue is the media portrayal of what genetic modification can realistically achieve and what the timeframe for these advances might really look like. Further education is called for to ensure journalists, and, therefore, the public who are reading and viewing news media, are better informed about the capabilities of scientific technologies.

3.4 Cloning

A number of ethical concerns are raised in the literature when it comes to human cloning. Would a clone be more susceptible to disease? Would the clone's "older" genetic material mean they would age more rapidly? How would research in to human cloning be conducted ethically (Caulfield, 2001)?

There appears to be some agreement in the literature on cloning that an individual can never really be cloned because we are the product of more than just genetic material (Lutz, 1997). However, the extent to which the genetic determinism argument is ingrained is evident. At the highest levels, there is support for the notion that we are our genes. Take, for example, the [United States'] President's Council on Bioethics (2004) which argues that cloning would interfere with the development of the cloned individual's identity and individuality. They argue that "genetic uniqueness is an important source of our sense of who we are and how we regard ourselves (p. 102)." Cloning, they contend, would clearly "present a unique and possibly disabling challenge to the formation of individual identity (p. 102)." The issue with this line of thinking is that it relies heavily on the principle of genetic determinism. These fears would be realistic if our genes were the sole dictators of "who we are and how we regard ourselves," but our personal identities and sense of individuality are the result of the interplay between innumerable complex factors. As Resnik and Vorhaus (2006) argue, this belief is based on the "unsupportable conclusion that two individuals with identical genomes will exhibit identical phenotypic expression. Not so. ...it seems a near certainty that even genetically identical clones would exhibit very different traits (p.9)." These traits, of course, would leave the two individuals, progenitor and clone, to behave differently, to pursue different life paths, to seek different experiences. These

differences add up to very different lives and, therefore, different people with different experiences.

In this day and age, it is not possible to conduct a literature review of cloning without coming across Dolly the Sheep. News of Dolly's birth hit the media waves in early 1997 when the lamb was six months old. Dolly was not the first animal to be cloned—sheep and cattle had been being cloned for a decade prior to Dolly's birth (Pennisi and Williams, 1997)—but Dolly's birth was of monumental significance because she was cloned using an adult cell. Up until her birth, it had been assumed that the DNA of adult cells was irreversible, meaning an adult cell was not "capable of supporting the development of all the different cell types needed to build an animal (Pennisi and Williams, 1997, p.1415)." Adult cells, scientists believed, could not be turned back because of "chemical changes and structural modifications (Pennisi and Williams, 1997, p.1415)." With Dolly's birth came proof that adult cells could be "reprogrammed" and a slew of histrionic headlines hit the media waves igniting immediate concerns that humans would surely be next. Within days (Pennisi and Williams, 1997), countries around the world called for increased, or introduced, legislation banning the cloning of humans. President Clinton called for an urgent inquiry by the National Bioethics Advisory Commission in the United States. He also banned the use of federal government funding for human cloning research. Dolly's creation uncovered potential loopholes in the existing legislation of many European countries, including the United Kingdom and Germany, and lead to the development of new policy to specifically cover the cloning of humans. The panic caused by Dolly was such that the 40-nation Council of Europe (which includes countries both within and outside of the European Union) immediately began developing a convention on human rights and bioethics (Pennisi and Williams, 1997).

It took Ian Wilmut and Keith Campbell at the Roslin Institute in Edinburgh, Scotland, 277 attempts to create Dolly (Pennisi and Williams, 1997), indicating just how complicated and difficult it is to clone a sheep. Still, those writing at the time, believed Dolly's birth represented a "tantalizing possibility (Pennisi and Williams, 1997, p.1415)".The hope was that cloning animals would enable the creation of copies of prized livestock and animals specifically for research purposes. Taken a step further—or a step too far, some might say—animal cloning technology may enable us to clone

genetically “modified animals that can produce drugs or better milk, meat or wool (James Rohl, quoted in Pennisi and Williams, 1997, p.1415).”

Animals aside, Lutz (1997) observes three barriers in the quest to clone a human parent cell. First, she argues that a cloned cell, borne from nuclear transfer, will not have the influence of mitochondrial DNA as would a cell borne from standard reproductive processes. Mitochondrial DNA comes from the egg so, believes Lutz, “unless the nuclear DNA from the parent were transplanted in to an egg from the parent’s mother, a clone would not genetically match the parent at every level (p.10).” Secondly, Lutz argues the DNA in an adult cell has undergone a lifetime’s worth of damage whereas a foetal cell has not. Lutz’s third point is that even identical twins are not identical so a clone would not be identical to its parent because of the influence of the environment.

Many of the epigenetic issues presented by the prospect of genetic modification also present barriers to cloning. With specific reference to the cloning of animals, Campbell et al (2005) seem adamant in their belief that these barriers can be overcome with further knowledge. They report success with the embryos and foetuses of a range of animal species in terms of applying the scientific techniques used to create Dolly the sheep. However, the birth rate of healthy offspring, of any species, remains low. These experiments have shed light on the “epigenetic mechanisms involved in nuclear reprogramming (p.263),” but the group acknowledges that “other cytoplasmic factors...such as the transmission of somatic mitochondria (p.263)” play an important role in development. Unlike the authors writing on the subject of genetic modification, Campbell and colleagues seem more optimistic that they will one day unravel the mysteries of epigenetics and be able to create a clone. They argue that with continued research they will improve the birth rate of live offspring by gaining a better understanding of the “control of cell differentiation and maintenance of the undifferentiated cell (p.263).” So confident are they in their abilities to unravel the mystery of epigenetics, they say that this ongoing research will “...inevitably lead... (p.263)” to an increase in live offspring and provide insight in to other methods of cloning. The applications for such technology—that is, improved live birth rates among cloned animals—are numerous: the repopulation of endangered species; cloning elite animals (such as superb milk-producing cows); the development of transgenic animals

for research, agriculture and biopharmaceuticals (Campbell, 2005); and, improving processes of animal farming by enabling superior animals to be bred more efficiently.

As with the other areas related to genetic technology examined here, there is a prevailing view that our genes define who we are and are essential for our sense of individuality and identity. Caulfield (2001) notes that the media's constant misrepresentation of the capabilities of genetic technology does nothing to dispel the myth. He argues strongly that our genes do not determine our future. They are essential, but there is "incredible complexity [in] the interaction between genes and other genes and between genes and the environment (p.403)." It is these myths that contribute to notions of parents being able to create a "replacement" child. Media misrepresentation of cloning caused top British scientists to make a public plea for better reporting in 2004 (Ross, 2004). The plea came a month after an American fertility specialist, Panos Zavos, claimed he had implanted a woman with a cloned embryo (Ross, 2004). Aghast at continually having to respond to such claims, scientists wrote an open letter to media editors pleading journalists to look more closely at the evidence before running with a story (Ross, 2004). "Over the past 2 years," they wrote, "such announcements have grabbed the headlines, despite the fact that none of those involved have produced a shred of evidence to substantiate their claims.... Despite the lack of evidence forthcoming on each occasion, we are still expected to respond every time a bogus claim is made (Ross, 2004, p.1)." They argue that those involved in spinning these stories "are more interested in publicity than advancing science," and they appealed to editors to closely assess the evidence before deciding the "priority given to these stories (Ross, 2004, p.1)." The letter was attributed to "several fertility and genetics pioneers; a scientist involved with the cloning of Dolly the Sheep; a former chief British government scientific adviser; the head of the government's medical research unity; and the president of the Royal Society, Britain's academy of science (Ross, 2004, p.1)."

Caulfield (2001) goes on to say that legislators may be as guilty as journalists of perpetuating the myth of genetic determinism. Banning human cloning on the grounds that it threatens individuality and identity suggests that genetic determinism is a realistic fear. This sentiment is echoed by Tranter and Statham (2007) who argue that legislators who remain committed to genetic determinism are only reinforcing the hysteria that surrounds cloning. Caulfield (2001) goes one step further to suggest that

the prevalence of deterministic thinking may lead the clone itself to undervalue its individuality and identity. The solution, according to Caulfield (2001), is that, rather than legislation, governments need to invest in educating the public about the real limitations of genetic technology in an effort at "cutting through the "genohype" (p.403)" that pervades much of the current popular culture.

The news media's portrayal of the sophistication of genetic technologies and the extent to which the results of any attempt at cloning are likely to be as predicted is also cause for concern. Hopkins (1998) argues that the media has lead the entire debate around cloning—from dispensing scientific knowledge, to shaping the relevant ethical discussion, to informing "social, religious and psychological significance" (Hopkins, 1998, p.6)—for both Joe Public and legislators. He charges that the media is responsible for "both revealing and creating (p.6)" the public moral debate that surrounds the possibility of human cloning. In Hopkins' survey of top-level United States media reporting of the issue, he focused on three main areas: "The loss of human uniqueness and individuality, the pathological motivations of anyone who would want a clone, and the fear of "out-of-control" science creating a "brave new world." (p.6)" Overwhelmingly, concerns related to cost, medical risk, and equal access have been ignored in favour of a focus on the risk to human individuality. Countless magazine covers have been plastered with images of "cookie cutter (p.12)" children and ask searching questions such as, "where do we draw the line (*Time,* 8 Nov. 1993)?" or "will there ever be another you (*Time,* 10 Mar. 1997)? (p.12)" Hopkins argues this portrayal of cloning as "mass production (p.7)" perpetuates the myth of genetic determinism. In another context, such as in reference to someone who has committed a violent crime, the general public would normally look to arguments based on genetic determinism (as in "my genes made me do it") as suspicious, provided, of course, the claims were not rejected outright (Hopkins, 1998).

What Hopkin's work clearly demonstrates is the need for greater education about the limitations of genetic determinism. He highlights why theses such as this are so important in crossing the barrier between science and the general public. A secondary aim of this thesis is an attempt to bridge this gap and ensure that better science is communicated to the public. With better information, the public can engage more

thoughtfully in ethical debate and, hopefully, the results will be legislation and policy that truly reflects the situation at hand.

Behind the sensationalist headlines, most news media go some way to explain that a clone would not be identical to its progenitor because there is no way the clone can have the progenitor's same life experiences, accidents or luck. It is communicated to the public (and legislators) that a clone would not necessarily be without a sense of self or identity, but somehow, this message always appears hidden or, in some cases, it is just not communicated clearly. Take, for example, Charles Krauthammer writing in *Time* who states, "[What] Dolly...promises is not quite a second chance at life (you don't reproduce yourself; you just reproduce a twin), but another soul's chance at *your* life (*Time*, 10 Mar. 1997)." Cloning as another soul's chance at your life? That is a very liberal interpretation of the scientific evidence!

Julian Savulescu (2005) believes others might try to make a case against cloning on the grounds that it would be an "affront to human dignity (p.18)"—human dignity being, according to the US President's Council on Bioethics, "the acceptance of a child whose genome is mysterious in origin (p.102)." According to this argument, parents should accept that their child comes in to the world as a mysterious gift—not a product to be purchased or modified. The argument here, according to Caulfield (2001), is that the autonomy of the clone would be compromised because the clone would not have genetic individuality. In an earlier article, Savulescu (1999) even states that other writers claim human cloning "crosses a significant boundary in removing the single most important feature of autonomy: the fact that each of us is genetically unique and individual (p.19)." The philosophical mistake made by so many is the assumption that there is a "significant relationship (p.4)" between our genetic makeup and our sense of individuality and identity (Resnik and Vorhaus, 2006). Once again, these writers cite identical twins as the perfect example. Few identical twins would say they feel any less unique or individual because of their genetic similarity with another person. Genetic uniqueness, they say, matters very little. For most people, a sense of self comes from living a life—from experiences, relationships, career and interests. Here, we have a philosophical view point serving to reinforce the scientific argument. Our sense of self and individuality is not the mere result of our genetic makeup; it comes from the

interplay of many complex factors—biological, environmental, personal, cultural and temporal.

An additional ethical concern related to human cloning is the fear that cloning promotes a form of eugenics—a process whereby those with desirable traits are cloned to perpetuate the desirable traits, in an effort to eliminate less desirable traits. Savulescu (2005) argues that we currently have technology available, and in use, that could achieve similar results: contraception, sterilization, abortion. These technologies are not currently the target of eugenic labels and there is no reason to believe human cloning would be any different.

There are a handful of other objections related to cloning. These include the issue that the clone may be born with the expectation that it will look and behave in an identical manner to its progenitor. The clone will, in effect, be born with a pre-determined life path. Cloning may also reduce the genetic diversity of the human population which has implications for human evolution. Of course, all of these ethical dilemmas are based on a notion of strong genetic determinism. Few of the articles surveyed go as far as to assess the basis of these claims; if they had, they would have discovered that strong genetic determinism is very rare and, therefore, these fears are unfounded.

In the literature, cloning is presented as having very strong ties to the principle of genetic determinism. It is clear those writing in this area have a number of concerns about cloning which are all based on this fear. These include the inability of a clone to develop its own identity and sense of self; the intrinsic value in our genetic uniqueness; and, fears related to eugenic selection of desirable traits. As we have seen with genetic testing and genetic modification, once it becomes clear that genetic determinism is a fallacy, these issues become less significant.

3.5 Understanding the Science

Let it be clear that I am not advocating for or against genetic testing, genetic modification or cloning. My position is simply that one cannot argue for or against any of these technologies from a position of genetic determinism. Genetic determinism is

not clear cut; therefore, any argument based on the principle weakens under close scrutiny. It may still be possible to argue that cloning is unethical because it is an affront to human dignity but this argument cannot be launched from a position of genetic determinism if it is to be taken seriously. In fact, it may be possible that all of the arguments presented in the literature represent legitimate fears, but if we are ever to stem the prevalence of the principle of genetic determinism, these arguments must be framed more appropriately and based on a better understanding of the science involved.

4 The Third Option

4.1 Limitations of Reductionism

It is evident from the preceding chapter that nature and nurture—biology and the environment—provide a rudimentary picture of how we come to be who we are. Consideration of these two factors alone makes it clear that a third option is necessary. This is largely because the arguments presented rely heavily on the factual nature of genetic determinism. What remains is to examine why the principle of genetic determinism may not be quite so straight-forward and explore a possible third component that might help to create a clearer picture of how we are who we are. At the outset, it should be understood that I am not intending to minimise the significance of nature and nurture. The influences discussed in this chapter, if legitimate at all, are minor in their influence. It is my intention in this chapter to examine the legitimacy of these other influences so that I may draw conclusions about how significant they really are and what the real-world implications may be.

When we consider complex systems, it becomes apparent that genetic determinism is not as straight forward as we might hope. As outlined in the introduction to this thesis, the concept of determinism is tied closely to reductionism; we reduce systems to their component parts in an effort to make predictions about how the system as a whole might behave. The strength of genetic determinism weakens when we consider the legitimacy of reductionism as a scientific method. Anderson (1972) explains, "The behaviour of large and complex aggregates of elementary particles, it turns out, it not to be understood in terms of the properties of a few particles. Instead, at each level of complexity, entirely new properties appear... (p.393_"

Reductionism assumes there is a point for point relationship between the constituent parts and the resulting complex system (Sapolski, 2010). In simple systems, there may indeed be a linear relationship between the parts and the system they add up to become and in this case, the linear relationship allows predictions to be made about the whole system. Similarly, if you know the simple system, you can make predictions about its initial conditions or parts. However, a complex system does not display linearity of additivity which means that adding up all of the component parts in a traditional deterministic manner does not give you a complete picture of the whole system. In

other words, input is not linearly related to output; "a small (large) change in some variable or family of variables will not necessarily result in a small (large) change in the system, (Rickles et al, 2007)." According to Anderson (1972), the main issue with this kind of scientific method is the assumption that a "reductionist hypothesis...[implies] a constructionist one. The ability to reduce everything to simple fundamental laws does not imply the ability to start from those laws and reconstruct the universe." Complex systems can be found everywhere in nature; weather systems are a prime example. Most biological systems are also complex and these systems illustrate a number of areas where the reductionist approach fails. One of these areas is visual processing.

Two neurobiologists, Hubel and Wiesel, conducted pioneering research in to the visual cortex and how we obtain visual information from the world around us (Hubel & Wiesel, 1959; Hubel & Wiesel, 1962). They discovered a simple, point-for-point relationship between certain photoreceptors in the retina and neurons in the first layer of the visual cortex. When the receptors were stimulated, the corresponding neuron in the cortex was stimulated (Hubel & Wiesel, 1959). They discovered that each of these neurons recognised a single dot in a particular area of the visual field. Hubel and Wiesel (1962) then discovered that if neurons in the first layer were stimulated in a particular order, a neuron in the second layer would fire. They concluded that neurons in the second layer recognised straight lines (Hubel & Wiesel, 1962). Further, if the appropriate neurons fire in the first and second layers of the visual cortex, a neuron fires in the third layer which recognises curves (Hubel & Wiesel, 1962). Sapolski argues that this approach is classic reductionism where knowledge of what is happening at one layer of the visual cortex gives 100% knowledge of what is happening in the other three layers (Sapolski, 2010). Extrapolating from this knowledge, it was assumed (Sapolski, 2010), that if one dug far enough in to the layers of the visual cortex one would eventually find the "grandmother neuron;" a neuron whose sole purpose was to recognise your grandmother's face at a particular angle. An adjacent neuron would recognise your grandmother's face at a slightly different angle. Further along, there would be a set of neurons whose sole purpose was to recognise your grandfather's face at particular angles, and so on until a neuron was discovered that would recognise every person and object we might ever encounter.

Elsewhere, neurons were discovered in the auditory cortex that recognised single notes from input provided by one cochlear cell. Layers and layers in to the auditory cortex, it was postulated that neurons would eventually be found that processed complex auditory information (Hubel & Wiesel, 1959) if the reductive process was followed.

Sapolski argues that researchers continue to search for grandmother neurons—the neurons that process and crystallise complex sensory input—but there are very few of them (Sapolski, 2010). Sapolski considers that there are a few instances of neurons recognising one, and only one, face (such as the Jennifer Anniston neuron which also, strangely, responds to images of the Sydney Opera House (Quain Quiroga et al, 2005)) but this is the exception, rather than the norm. Sapolski adds, "The vast, vast majority of attempts to find grandmother neurons failed dismally for a very simple reason." The reason, according to Sapolski, is because there are not enough neurons in the visual cortex—indeed, in the entire brain—"to do face recognition in a point for point reductive manner... You can't solve the problem of recognising faces by using reductive component part neurobiology (Sapolski, 2010)."

A further example of the limitations of the reductive process is in bifurcating systems which are found throughout nature, most notably in circulatory systems. The issue arises when we ask questions about how the body codes for a bifurcating system. The walls of arteries and veins are composed of cells. As cells are being laid down in order to create an aorta, for example, reductive scientific thinking says there must be a gene (or genes) which tells the system the point at which to bifurcate in to the left and right iliac arteries. Further down the arterial line, there must be another couple of genes which tell each iliac artery to bifurcate again. This process must repeat all the way down to the level of capillaries and it also occurs in the venous and lymphatic systems. Bifurcation also occurs in the brain and is responsible for the branching patterns seen in the dendrites of neurons. Sapolski argues the genes responsible for bifurcation in the circulatory system are vastly different from those guiding the development of neurons because the walls of the circulatory system are made of thousands of cells, whereas a neuron is a single cell unto itself (Sapolski, 2010). The two systems are operating on completely different scales; one involves bifurcation within a single cell, one involves creating a bifurcating pattern using thousands of cells as building blocks. As with processing sensory information, the issue here is that there are not enough genes in the

genome to code in this manner. “You can’t code for bifurcating systems in a living organism that covers completely different scales... You can’t code for it in a point for point reductive way where the points down at the bottom, the component parts, are individual genes. ...You can’t code for the bifurcating systems in the body because [components of the circulatory system] will bifurcate out to millions of capillaries and there’s only 20,000 genes. The reductive process breaks down here...,” Sapolski notes (2010). Sapolski may have a legitimate point to make about the genetic coding of bifurcating systems, however, he does not cite any references in his argument. He may be right on this point, but it has not been possible to support his theory with the work of any other writers.

Reductive logic also breaks down in behavioural or social systems which are very complex. Chase et al (2002) studied the formation of dominance hierarchies in cichlid fish, a species of fish which is known to be especially dominant. In one of their experiments, the fish were spilt in to pairs and each pair was placed in a separate tank. The pair of fish were separated by a partition for two hours and allowed to acclimatise to the tank before the partition was removed. Once the partition was removed, the fish were observed until it was established which of the two was the dominant fish and then they were returned to their isolation tanks. This process was repeated in a “round-robin” tournament style, until each of the ten fish had been paired with every other fish. The results of the tournament were used to determine a dominance hierarchy for the group. The most dominant fish was the one who had dominated all nine of the other fish in the one-to-one interactions. The second most dominant fish had dominated eight of the others, and so on. Once the predicted dominance hierarchy was established, all of the fish were placed in to a single tank and, once again, the researchers observed the behaviour of the fish. Surprisingly, the dominance hierarchy predicted from the individual pairings had no bearing on the behaviour of the fish in a social situation. An entirely different dominance hierarchy was observed. Sapolski argues that this is because chance plays an important role. “Chance interactions wind up driving the system. Random movement of the animals and such winds up meaning knowing the starting states of the dominance relations of every single dyad gives you zero predictability of what the complex system is going to look like,” he says. Chase et al (2002) and Sapolski (2010) both concluded the results of this study were due to chance but, of course, there may be other interpretations. I cannot find fault in the study’s

methodology but the group's observations may be due to the complex nature of social interactions, rather than chance.

Complex systems do not display linearity of additivity; what we see is the emergence of unexpected properties and, to further complicate matters, Sapolski argues chance (or chaos) also plays a role. Chaos, he believes, throws off our ability to predict the initial conditions of a complex system—in other words, our ability to reduce the system to its component parts. In a reductive world, it is assumed that the embryos of identical twins divide evenly and therefore the resulting cells are identical. This process is repeated over and over again through countless cell divisions and the result is the birth of two identical twin babies. Sapolski argues that this is not the case because even after the first cell division, the distribution of the molecular cell components—mitochondria, transcription factors and the like—is uneven (Neumüller and Knoblich, 2009; McMurray and Thorner, 2009; Koch and Schaechter, 1962; Kœrn et al, 2005).

Similarly, Raser and O'Shea (2005) point to the consequences of "random processes such as the partitioning of mitochondria during cell division" on gene expression. They state that chance may play a role in many biological events, including identical human twins having finger prints that are "readily distinguishable on close examination." The effects of chaos are also seen in density and distribution of sensory hairs on the bodies of fruit flies (Lehrer, 2008, p.49). The population of hairs differs between the two sides of the fly due to "random atomic jostling inside [the fly's] cells, (Lehrer, 2008, p.49)." Some even claim our individuality is a result of the chaotic actions of retrotransposons in the brain (See Lehrer, 2008, p.50) while Kœrn et al (2005) refer to chaos (or stochasticity) as playing a role in "non-genetic individuality." Following the first cell division, Sapolski says chance throws off our ability to look at the initial conditions and know what the complex system is going to be (Sapolski, 2010).

One interpretation of these examples is that they illustrate the limitations of a reductive system. From the behaviour of animal groups down to the activities of single genes, reductive systems break down "because there simply aren't enough pieces in there to explain complex function in a point-for-point...way... and there is no way to deal with the fact that chance plays a role in biological systems," (Sapolski, 2010). Sapolski argues that these examples point to the fact that the most interesting parts of science

such as brain function and genetic regulation "...can't be regulated in a classical, reductive way... so it's got to be something else," (Sapolski, 2010).

The assertions made by these scientists may be extreme and, to some extent, based on speculation. However, aside from Lehrer, these are well established, extensively published researchers (Lehrer has made his conclusions based on consideration of the work of other well established and extensively published researchers). In particular reference to Sapolski, it is worth noting that while he is extensively published and teaches a course on this topic at Stanford University, he is not published in the fields of chaos or emergence. While there are limitations to these researchers' claims (namely, the modest body of research in this area), these limitations are not sufficient to detract from the potential legitimacy of their claims. The conclusions reached by these scientists should be viewed critically, but it would be misguided to write them off entirely based on the fact that these are novel claims.

4.2 Terminology

Many scientists choose to use the word "noise" (or sometimes "stochasticity" or "chance") rather than chaos (Lehrer, 2008, p.48). Rickles et al (2007) refer to this as "looseness" in how the terminology has been taken from the physical sciences and applied to biology. It is worth asking ourselves whether this is really necessary. Is it simply because the idea of there being chaos present in biological systems—indeed, practically everywhere in the natural world—is too much of a challenge to our traditional ways of thinking? The notion that we will never have a complete understanding of human biology or human nature is unsettling and, potentially, disheartening for scientists who devote their lives to understanding and unravelling the human condition. For further discussion of this point, refer to Chapter Five.

Chaos does not refer to randomness in the sense of having no idea what the outcome in a particular situation may be or which behaviour a certain system may display. Rather, chaos means knowing that the outcome will be one of three or four variables, but not being able to predict from the initial conditions which of the three or four variables it will be (Laughlin, 2005, p.130)." Take, for example, a dice. When you roll a standard

dice you know ahead of time that you will roll one of six numbers but the principle of chaos makes it impossible to predict ahead of time which number you will roll (Laughlin, 2005, p.130).” This is sometimes referred to as complexity. When a system is referred to as chaotic (or complex) what is really meant is “that the physical process by which it is formed is unstable and with a slight nudge could have generated one of many different [variables] (Laughlin, 2005, p.130).” Conversely, Laughlin notes (2005, p.130), a system is said to be simple (or non-chaotic) when the same variables are produced every time, “even when nudged fairly violently.” This is an important point for two reasons.

Firstly, few systems are chaotic in the absolute sense. In every situation, there are a finite number of outcomes; the principle of chaos enters the picture in trying to determine which of the outcomes will triumph. For example, imagine you sow a garden with carrots and sunflowers. Assuming there are no remnants from other gardening projects left behind [other seeds or weeds], all that is going to grow in this garden is carrots and sunflowers; you will never grow a watermelon, or a tulip, or a grizzly bear. The chaotic (or random or complex) part of the garden is whether all of the seeds will grow, only some, or none; how many of each will grow; will they grow to the same size.

This example leads nicely in to a further reason why it is important to understand that the principle of chaos does not refer to chaos in the absolute sense, but rather it refers to chaos within the boundaries of selecting between a number of possible outcomes. The second reason relates to principles of order which also operate within the system. Take the garden example once again. Without order in the system, you just might end up growing a watermelon or grizzly bear. The system has structures and processes in place to ensure that only a finite number of variables are possible outcomes. In the human body, these processes ensure that humans (in the majority of cases) end up with two eyes that are brown, blue, green, grey or hazel. Once the possible choices of eye colour are narrowed by genetics, an individual may be destined to have brown or blue eyes. It is the order within the system which ensures the individual ends up with brown or blue eyes—not pink or yellow. This phenomenon is often referred to as self-organisation (or collective organisation for Laughlin, 2005, p.31) and describes the pattern of order emerging from chaotic systems. Self-organisation is itself a fascinating principle but it

is beyond the scope of this thesis. De Wolf & Holvoet write informatively on the subject and its relationship to emergence. Briefly, they describe self-organisation as "an increase in the structure or order of a system's behaviour (2005)" to promote specific functions without the guidance of external controls.

These observations lead to a further important point. Despite common vernacular—and perhaps, common sense—a chaotic system is deterministic (Monti, 2010). In the context of physics and mathematics, a deterministic system is any system that is obeying a set of rules; the behaviour of [the] system appears random, but is generated by simple, non-random, deterministic processes (Rickles et al, 2007)". These systems are entirely determined by their initial conditions (Kellert, 1993)—that is, the initial environment within the system determines the system's available options. For example, in the garden scenario above, the initial conditions are soil that is ripe for growth, as well as carrot and sunflower seeds. These initial conditions determine (or limit) how the behaviour of the system will unfold. In the garden example, the system will behave in such a way that some ratio of carrots and sunflowers will grow. Despite the system being deterministic (in that it obeys a set of rules), it is still not possible to predict exactly how the system will behave (with our current knowledge, at least). As previously discussed, we cannot tell how many of each seed will grow, or even if they will grow. The idea that a system is determined by its initial conditions is called sensitive dependence on initial conditions (Gleick, 2008, p.23) and may be more commonly referred to as "The Butterfly Effect."

4.3 Chaos in Nature

In popular culture, the Butterfly Effect is said to be responsible for "the notion that a butterfly stirring the air today in Peking can transform storm systems next month in New York (Gleick, 2008, p.8)." Weather systems are a brilliant example of sensitive dependence on initial conditions. This phenomenon was uncovered by Edward Lorenz in the 1960s (Gleick, 2008, p.11). Lorenz created a model weather system on his computer which allowed him to programme a set of initial conditions and watch the weather unfold. His machine produced a print out of numbers and these numbers signified the activities of the weather system, such as westerly winds or cyclones

(Gleick, 2008, p.11). The graphics used by Lorenz's computer system, displayed the output as a wavy line printed on a roll of paper. As the weather system moved, so too did the wavy line on the paper and those in the know could interpret the line to figure out what was happening with the weather system at any given time (Gleick, 2008, p.16). Lorenz assumed that once he settled upon a set of laws—he chose twelve equations that described various relationships—and programmed them in to his computer that the "determinism of physical law" (Gleick, 2008, p.12) would render further interventions unnecessary. According to Gleick, the philosophy of those who made computer models was that understanding the laws meant understanding the universe (p.12). In theory, computer models were supposed to give meteorologists the same power astronomers had: to "reckon the future of their universe from its initial conditions, (Gleick, 2008, p.14). Gleick notes one small flaw in the assumptions of working scientists: the acceptance that measurements would never be perfect, and therefore the predictions would never be perfect, but a small error in measurements would only ever lead to a small error in the results (p.15). For example, a small error in the measurement of Comet Halley location in 1910 would only result in a small error in predicting its location in 1986 (Gleick, 2008, p.15).

According to Gleick (p.16), on one occasion in 1961 Lorenz decided he wanted to examine one segment of the print out in greater detail so, rather than starting the model running from the beginning, he started closer to the relevant part. Using an earlier print out of the same sequence, he typed the numbers that represented the sequence he was interested in—.506 — and set the computer to work. If previous theories were correct—that small errors in measuring the initial conditions would only result in small errors in predicting the results—Lorenz should have seen virtually the same print out. But he did not. When Lorenz looked at the original print out and compared it with the new he thought "he might well have chosen two random [weather printouts] from a hat (Gleick, 2008, p.16)." The weather pattern in the new printout "diverged so rapidly from the pattern of the last run that, within just a few months, all resemblance had disappeared (Gleick, 2008, p.16)."

Lorenz realised the problem lay with the numbers he had typed in to the computer (Gleick, 2008, p.16)." In the computer's memory, the code that represented the original pattern was stored as .506127, whereas Lorenz had run the system again using the code

.506. According to Gleick, Lorenz made a reasonable decision in assuming that the difference of one part in a thousand between the two codes was insignificant (p.16). Based on all previous theories, such a small deviation in the initial conditions of the two systems should only have resulted in a small deviation in the results. Surely, Gleick reasons, small numerical errors "should [fade] or [cancel] each other out before they could change important, large-scale features of the weather, (p.17)." Instead, however, the small numerical error resulted in the creations of two weather systems that were wildly different. Lorenz eventually reasoned that the Butterfly Effect was necessary (Gleick, 2008, p.22). "To produce the rich repertoire of real earthly weather, the beautiful multiplicity of it, you could hardly wish for anything better than the Butterfly Effect, (Gleick, 2008, p.23)." Lorenz had discovered a system displaying chaos. As previously discussed, this is not the same as a random system. A chaotic system is following a set of rules, but the outcome of these rules is not predictable.

Lorenz expanded his search and found systems displaying similar, chaotic properties elsewhere. Fluid systems display chaos. If water is heated from below, the hot water will rise, causing the relatively cooler water on top to fall to the bottom. This heating and cooling process causes the water to form cylindrical rolls. If the heat is increased, instability appears in the system and smooth cylindrical rolls give way to a wobble that travels along the length of the cylinder. If the heat is increased further still, the wobble turns in to "wild and turbulent" flow (Gleick, 2008, p.25).

This behaviour is also observed in a particular kind of waterwheel model and is precisely described by Lorenz's equations. Imagine a waterwheel that looks exactly like the side of a Ferris wheel. The waterwheel has a number of evenly spaced buckets attached to it and each bucket has a small hole in the base for drainage. Water is steadily poured on the top of waterwheel. If the water flows fast enough, the top bucket fills enough to overcome friction and the wheel begins to move. If the flow of the water remains steady, the second bucket fills enough and eventually the wheel starts to move in a steady circular motion around a central point of attachment. If this continues, the wheel will reach equilibrium and rotation will continue at a steady state. If however, the flow of the water is sped up, the system becomes chaotic. As the water speeds up, so too will the wheel—initially, at least. The first couple of buckets to pass under the water will be filled more than the other buckets on the wheel, simply because more

water is entering the buckets per unit of time. These heavier buckets will cause the wheel to speed up, but, as a consequence, of the wheel speeding up, there is less time for each of the remaining buckets to fill. A second consequence is that the heavier buckets have less time to empty before reaching the top of the wheel again. These two factors cause the wheel to begin spinning in the opposite direction. Lorenz's intuition was that the wheel would eventually reach a steady state and begin to "rotate steadily or it would oscillate steadily back and forth, turning first in one direction and then the other at constant intervals, (Gleick, 2008, .p30)." It was expected that the system would repeat itself periodically. Instead, the system was aperiodic and behaved chaotically.

4.4 Chaos at the Genetic Level

Chaos is found throughout biological systems (Swain et al, 2002; Arkin et al, 1998). Spudich and Koshland (1976) write that "biological systems are constantly confronted with [chaotic] occurrences in their environmental conditions and internal processes." They go on to argue chaos may actually be used as "an elaborate biological apparatus" to help the survival of the species. The role of chaos in biology was posited by Motoo Kimura in 1968. In his paper "*Evolutionary Rate at the Molecular Level,"* Kimura argues that the human genome changes at a rate between 100 and 1000 times greater than would be predicted based on evolution alone. In other words, if evolution—or natural selection or adaptation—was the only driving force, our DNA would change much, much less than it does. Kimura's explanation for this discrepancy was chaos (Kimura, 1968; Kimura, 1989). He argued that we must "recognise the great importance of random genetic drift...in forming the genetic structure of biological populations, (Kimura, 1968)." Ignoring the role of chaos (in favour of purely evolutionary arguments), he states, is "rather similar to assuming a great flood to explain the formation of deep valleys but rejecting a gradual but long lasting process of erosion by water as insufficient to produce such a result, (Kimura, 1968)." In direct reference to Kimura's work, Lehrer (2008) says: "At the level of our DNA, evolution works mostly by accident. Your genome is a record of random mistakes. ... Inside our cells, shards and scraps of nucleic acid and protein float around aimlessly, waiting to interact. There is no guiding hand, no guarantee of exactness, (p.48)." Lehrer's view is certainly interesting, although this should be considered an extreme view.

A beautiful illustration of the presence of chaos in biology comes from Elowitz et al (2002) who studied the effects on protein expression of the low concentration of many important cellular components. They reason that the expression of each gene is limited by "the concentrations, states, and locations of molecules such as regulatory proteins and polymerases (p.1183)," therefore any fluctuation in the presence of any of these molecules will result in fluctuations in gene output (Elowitz et al, 2002). These fluctuations affect the output of a particular gene within the cell, and the fluctuations will undoubtedly vary between cells. But, they caution, even in a cell where the concentrations and states of these molecules are identical, gene expression will still vary because of stochasticity—or chaos. In these systems, chaos causes "microscopic events that govern which reactions occur and in what order," (Elowitz et al, 2002, p.1183). The group argue, "No matter how accurately the levels of regulatory proteins are controlled, [chaos] fundamentally limits the precisions of gene regulation (Elowitz et al, 2002, p.1183)." In an effort to measure the levels of intracellular stochasticity (or chaos), Elowtiz et al designed an experiment using cyan and yellow strains of the green fluorescent protein (GFP) inserted in to *Escherichia coli* (*E. coli*). GFP is a reporter gene which is simply a gene whose expression is easy to monitor. In the case of GFP, when the gene is expressed the cell will change colour—usually to green, but in Elowitz's experiment cyan and yellow strains were used so the colours cyan and yellow will be expressed. The two different strains of GFP were introduced in to *E. coli* cells in equal numbers. If equal amounts of the two proteins are expressed—as would be expected given that each cell as the same amount of each protein—the cells will appear yellow. However, if more of the yellow strain of GFP is expressed, the *E. coli* cell will appear red and vice versa, more cyan means the cell will appear green. One would expect an excess of yellow GFP to produce a yellow cell (and similarly with an excess of cyan). Fortunately, understanding this element of the study is not critical to grasping the main outcome of the study—that is, the presence of chaos within the cell.

In a startling illustration of the presence of chaos in gene regulation, Elowitz's group saw that the expression of the two different genes was wildly different in all of the cells. Rather than producing a population of yellow cells (indicating that the two proteins had been expressed at the same rate), they produced a population of different coloured cells. The different coloured cells indicate that gene expression within each

cell was subject to the influences of the "concentrations, states and molecules, (Elowitz, 2002, p.1183)." Where it was expected that gene expression would operate like clockwork and produce a population of yellow cells, chaos was shown to be present, thus "giving rise to a population in which some cells express more of one fluorescent protein than the other, (Elowitz, 2002, p.1183)."

Ozbudak et al (2002) go as far as to claim chaos (or stochasticity) is "ubiquitous in biological systems (p.69)." The group conducted an experiment very similar to Elowitz et al (2002) in which they sought to understand whether variation in gene expression between identical cell populations could be explained by stochasticity. To ensure their experiment measured only fluctuations attributable to stochasticity, the group carried out a number of differential measurements. Similar to Elowitz et al, their experiment compared the rate of expression of green fluorescent protein (GFP) in *B. subtilis* and they concluded that increased translational efficiency is the predominant source of stochasticity. The group reason that this is because low rates of translation lead to "reduced fluctuations in protein concentration (p.69)," rather than higher rates of translation where proteins are produced in "random and sharp bursts (p.69)." If stochasticity in biochemical reactions is due, in some part, to the low concentration of some intracellular molecules, reducing the fluctuations in protein concentration (by lowering the rate of translation) may offer the cell some sort of control mechanism.

Blake et al (2003) also demonstrated the presence of stochasticity in biological systems by studying *Saccaromyces cerevisiae*, a species of yeast. The group concluded that stochasticity arises due to transcription, contributing "significantly to the level of heterogeneity within a eukaryotic clonal population (p.633)," and, similar to Ozbudak et al (2002), that this stochasticity can be altered at the translational level. Blake et al observed a 20-40% increase (compared with base line) stochasticity at higher rates of transcriptional efficiency (or, the number of protein molecules produced from a single mRNA transcript). Increased stochasticity in the transcription of regulatory proteins leads to increased variability between cells which, they claim has "implications for the role of noise in phenotypic variation and cellular differentiation (p.633)."
Stochasticity has also been implicated in the development of tumours. Kemkemer et al (2002) reports that the number of copies of a gene is critical to ensuring "a predictable outcome of gene products (p.13783)." The group worked with human melanocytes

(melanin producing cells) that were haploinsufficient (where mutation causes an individual to have only one active allele for a particular gene instead of two) for the tumour-suppressor gene, neurofibromatosis type 1 (NF1). They compared the dendrite formation of normal-type melanocytes and melanocytes haploinsufficient for NF1. The group observed increased variation in dendrite formation in the haploinsufficient cells. They concluded that in cases of haploinsufficiency, there is a notable increase in stochastic activity which may interrupt gene expression and they argue that haploinsufficiency may have similar effects in other tumour-suppressor genes.

Despite considerable effort, alternate explanations for stochasticity could not be located. Article upon article demonstrates its presence within the cell (in addition to those above, Becskei et al (2005), Kœrn et al (2005), Raser and O'Shea (2005), Kepler and Elston (2001) and McAdams and Arkin (1999)) but there appear to opposing views to contrast with the affirmative perspective. All of the experiments cited have controlled for instrument and human error (through multiple repeats of their experiments. Therefore, the only alternate explanation remaining may simply be that we do not yet possess the knowledge or the technology to explain these processes. There may, in fact, be a set of predictable rules operating that we do not yet understand. In future, we may come to understand these rules, however, the evidence for stochasticity is considerable.

4.5 Emergence

The interactions between the components of a nonlinear system—including interactions that are the result of chaos—give the whole system properties which cannot be reduced to the components (Rickles et al, 2007). These properties are known as "emergent properties" and it is these properties that represent the extra piece in the equation; the extra piece that is present within the system but which cannot be identified if the system is broken down in to its component parts. Chaos, on the other hand, is an identifiable component whose effects can be seen within the component parts of the system; emergent properties only exist when the components are added together.

To truly understand the best example of emergence—Newton's laws—and, therefore, the magnitude of the concept, it is necessary to have a basic understanding of the history of physics. In the 1600s, Galileo, Kepler and others observed and documented regularity in the natural world. It was Galileo who initially observed the basis of Newton's First Law of Motion; that an object will remain moving at a constant speed in a straight line unless an external force is applied. Kepler, on the other hand, made observations about the planarity, size and period of planetary orbits. In 1687, well after the passing of both Galileo and Kepler, Isaac Newton published his historic *Principia Mathematica* (Hawking, 2005, p.61) which built on his predecessors' work by applying mathematical principles to describe their observations. Newton's mathematical equations were straightforward, could be applied consistently and connected seemingly unrelated behaviours (Laughlin, 2005, p. 23). In 1905, building on Newton's work, Einstein released his *Special Theory of Relativity* (Laughlin, 2005, p.117). Between the two of them, Newton and Einstein developed the laws that explain the movement of the world we see before us—from the planetary down to the human scale. While Einstein's theory offers more precise predictions, Newton's laws (also known as Newton's theory of gravity) are used for most experimental purposes because the difference in accuracy between the two theories is small and Newton's laws are much simpler to work with (Hawking, 2005, p.46).

According to Laughlin (2005, p.30), over time, Newton's laws of motion were applied more and more widely, and eventually it was assumed Newton's laws applied in experiments where the validity of the laws could not actually be directly verified. Calculations were made based on this assumption (that Newton's laws were correct under the experimental conditions) and then the experimenters would "argue from agreement with experiment that the initial assumptions were correct" (Laughlin, 2005, p.30). This circular line of thinking meant it was assumed Newton's laws applied to situations where the laws were actually irrelevant. For example, Newton's laws predicted "a beam of helium atoms projected onto an atomically perfect solid surface" (Laughlin, 2005, p.31) would bounce off in all directions when, in fact, the atoms "diffract into rainbows as a beam of light would do" (Laughlin, 2005, p.31). Atoms do not behave like billiard balls as Newton's law suggest but rather they behave like waves (Laughlin, 2005, p.31). Discovered in the early twentieth century, the laws describing the movement of subatomic particles, atoms and molecules are referred to as quantum

mechanics. It is worth acknowledging here that these are physics-based examples because no genetic or even neuroscience examples of the application of this principle could be found. The question of the extent to which an idea in physics applies to biology is a valid concern and will be addressed in Chapter Five.

The discovery of quantum mechanics by Bohr (Gleick, 2008, p.7), Schrödinger, Heisenberg (Laughlin, 2005, p.4) and others unveils another deeply puzzling phenomenon. How can it be that the behaviour of planets and atoms must be described by different laws? The laws of quantum mechanics fail when the number of atoms involved exceeds around ten (Laughlin & Pines, 2000). But planets are made of atoms; planets can be reduced to atoms. How can the movement of planets be described by Newton's laws and the planet's constituent parts—atoms—are described by a set of laws "so different from Newton's that scientists struggled to find proper words to describe them" (Laughlin, 2005, p.31)? The answer is that Newton's laws are the largest—in terms of both scale and significance—example of emergence (Laughlin, 2005, p.31; Verlinde, 2010; Dhar & Giuliani, 2009). Laughlin (2005, p.31) writes, "They are not fundamental at all but a consequence of the aggregation of quantum matter into macroscopic fluids and solids—a collective organizational phenomena." Emergence may be seen to be a result of the fact that "[universal] laws hold well only in a certain range" (Dhar & Guliani, 2009) and the emergence of new laws at different levels of hierarchical organisation is actually critical to our existence (Dhar & Guliani, 2009). Laughlin (2005, p.31) believes many physicists remain in denial about the roots of Newton's laws and insist they are an "approximation" for quantum mechanics; "valid when the system is large enough." But, he argues, "...no legitimate approximation scheme has ever been found." Elsewhere, Laughlin & Pines (2000) write that many working in the physical sciences believe the idea of higher organising principles is "dangerous and ludicrous" because it is "fundamentally at odds with the reductionist beliefs central to much of physics."

As a brief aside, Hawking writes that the eventual goal of science is to "provide a single theory that describes the whole universe (Hawking, 2005, p.46)"—a quantum theory of gravity, encompassing the general theory of relativity and quantum mechanics (Laughlin and Pines (2000) also briefly discuss the search for this "Theory of Everything."). To date, the search for the quantum theory of gravity has involved

breaking the problem up in to smaller problems, true reductionist style, and developing a series of "partial theories." Hawking concedes that this approach may be wrong if "everything in the universe depends on everything else in a fundamental way (Hawking, 2005, p.348)."

A system can be said to display emergent properties when "there are coherent emergents at the macro-level that dynamically arise from the interactions between the parts at the micro-level (De Wolf & Holvoet, 2005)" The emergents, or emergent properties, are new—that is, they were not readily understood from individual system components at the micro-level (De Wolf & Holvoet, 2005). De Wolf & Holvoet (2005) report they settled upon this definition after an extensive literature review to identify the most important and most often discussed characteristics. One of these important characteristics is the micro-macro effect which refers to "properties, behaviours, structures, or patterns that are situated at a higher macro-level and arise from the (inter)actions at the lower micro-level of the system. (De Wolf & Holvoet, 2005)" In short, this effect produces emergent properties. Also critical to understanding emergence is accepting that the emergent properties (behaviours, structures or patterns) are not explicitly represented at the micro-level (De Wolf & Holvoet, 2005). They write that this phenomenon is, by definition, non-reductionist because "the macro-level emergents are not reducible to the micro-level parts of the system," but the emergents can be studied within the context of the entire system. The pair cautions that attention must be paid to how emergent properties are described. It would be incorrect to state that the "emergents are not captured by the behaviour of the parts (De Wolf & Holvoet, 2005)" because the emergent property is "implicitly contained (De Wolf & Holvoet, 2005)" within the properties of the parts—the property had to have come from somewhere, after all. The emergent property appears radically novel, they argue, because it is *not readily understood from* the properties of the parts.

4.6 Emergence in Biology

As we have already seen, emergence is prevalent within biology. Sapolski (2010) and Chase et al (2002) offered many examples: visual processing; auditory processing; bifurcation in the circulatory system, as well as within neurons; and, social systems. For

Laughlin and Pines (2000) the prevalence and essential nature of emergent properties is obvious but, they write, the idea is resisted by many scientists because "it is fundamentally at odds with the reductionist beliefs central to much of physics." Of course, the reluctance of many scientists to except emergence (and chaos) as central to our understanding of the natural world does not make these concepts any less real.

Much of foundation of knowledge relating to emergence comes from physics and has simply been applied to biology. This has been done based on the assumption that physics naturally transitions in to biology (via chemistry, one might argue). No real literature exists on this topic that would make it possible to definitively argue that this assumption is true. While it may be possible to argue that an idea in physics does not apply to biology, recent trends in both physics and biology would suggest this is not true. Physicists are increasingly turning their hands (and heads!) to the questions of biology (Wolgemuth, 2011; Lee, 2006; Knight, 2002; Pastore, 2012) which suggests the ideas and principles of physics do indeed apply to the biological sciences.

4.7 Putting it all together

In light of the evidence for the presence of chaos and emergence in biological systems, what can be made of the views expressed in chapter three by authors writing in the areas of genetic testing, genetic modification and cloning?

4.8 Genetic testing

Lucassen (2012) contends that the issues with genetic testing are the certainty of test results and our ability to accurately interpret the data. She states we do not currently possess the skills to undertake "complex bioinformatic analysis (p.196)." An additional fear put forth by Lucassen is that genetic testing may lead to an "oversimplification of [the] disease process (p.196)" with talk of an individual having 'the gene' for a particular disease. While it may be uncommon for people to speak of 'the gene' for a disease in serious discussion, Lucassen's concerns are based strongly on a belief in genetic determinism and, unfortunately, this view underpins many significant bioethical discussions. When we add chaos and emergence to the picture, it seems reasonable to

believe we may never have the knowledge to understand the complex nature of all biological systems and, therefore, we may be severely limited in which diseases we are able to make accurate tests for. That is, we may only be able to create genetic tests for those diseases where one, perhaps two genes, are responsible, such as cystic fibrosis (Karem et al, 1989), Huntington's Disease (Sermon et al, 2002) or glycogen storage disease type 1A (Lei et al, 1993). Once we accept the limitations of the reductionist/determinist model of science, individuals will stop expecting genetic tests that predict for anything but the most straightforward diseases. Gradually, this will lead to a greater understanding of our genetic complexity.

Lynch et al (2001) (and many others: Clayton, 2003; Hubbard and Wald, 1993; Nelkin and Lindee, 1995; Silva, 2005; McCabe and McCabe, 2008) support the view that genetic testing may lead to an oversimplification of disease processes and add that the media contribute substantially to the proliferation of this belief. They report that the media's explanation of genetic testing "...[becomes] the building blocks on which the public constructs their own understanding of the scientific issues (Lynch et al, 2011, p.487)." Lynch and colleagues (2011) state that this is of concern because the media is a significant source of information for consumers of genetic tests (along with the Internet and the company conducting the test). Arnason and Hjörleifsson (2007) made similar observations about the media's role, adding that genetic testing tends to be portrayed positively because of its power to "... [alleviate] and [prevent] suffering and [the likelihood it will] yield financial benefits (p.423)." They also noted that journalists are unlikely to question the research provided by scientists or the predictions they make.

Misreporting of the facts pertaining to genetic testing is a significant concern because, as we have seen, media sources are a considerable resource for the public. The public's misunderstanding of genetic determinism becomes a concern when individuals start weighing in on bioethical discussions. For media, the issues are twofold: 1. The ingrained belief in genetic determinism which leads to an exaggeration of the impact of our genes, 2. The inability (or unwillingness) or journalists to question the research they are given by scientists and the conclusions those scientists draw. Perhaps part of the solution here is the training of more journalists from a scientific background or, an insistence by the public that news editors and journalists examine scientific claims with

more scrutiny before they go to print—although, this is not to say that all journalists and media outlets are guilty of misrepresenting scientific achievements.

The amount of research that has gone in to the media misreporting of science and, subsequently, the extent to which this affects public perception, raises the question of whether the science presented by the news media accurately reflects the discussion occurring within the scientific literature. This issue was addressed in Chapter Three as a result of an extensive analysis of the scientific literature.

Hubner (2006) argues that medical insurance companies have a strong case for ensuring the myth of genetic determinism remains alive and well. If the public continues to believe that the discovery of gene X as a result of a genetic test means the individual will develop disease X, the insurance company can determine its premiums accordingly. If, however, the public were to understand that gene X may only be a contributor to the disease or that the gene may be inactive unless a certain environmental stimulus is encountered, the insurance company would struggle to get away with refusing coverage or charging higher premiums. The case for genetic determinism here is strong but, hopefully, as understanding of the complexity of biological systems gains greater momentum practices like this will be phased out.

Lippman (1999) and Rothman (2001) both argue that genetic testing reinforces the importance of medical interventions at the expense of social, environmental or political solutions. Rothman (2001) goes as far as to contend that genetic testing may be used "in attempts to gain control over our future through manipulating human procreation... (p.104)" Rothman's view is hugely misguided; this line of thinking represents very outdated science. Once we reduce biological systems to their component parts, we have seen that adding these parts back together creates higher levels of organisation and within these new levels of organisation, new sets of laws may emerge (Anderson, 1972). The emergence of new laws at each level of organisational hierarchy makes it difficult to believe that we will have a complete understanding of the workings of biological systems in the near future. Add to this the presence of chaos within biology (Elowitz (2002), Ozbudak (2002), Blake (2003), Kemkemer (2002) and Chase (2002)) and we may never be able to accurately describe human development and behaviour. This knowledge helps to put claims such as those by Rothman (2001) in to perspective.

Chaos, and to some extent, emergence means it is very unlikely that humans will ever have the scientific knowledge and technology to "gain control over our future... (p.104)" in the way Rothman suggests. What is more likely is a future where there will always be the possibility that technologies or procedures will not have the intended results 100% of the time. This will be discussed further in Chapter Five.

4.9 Genetic Modification

A major concern in relation to genetic modification is whether we should be working towards the improvement of traits or characteristics that would be considered normal (Gordon, 1999). Rosoff (2012) argues that we may have a duty to treat someone who is ill by any means we have available, but we do not face the same obligation to help someone who is living "within the normal range of human functioning (p.164)." This thesis has done little to sway the argument either way on this issue, that is, whether or not we should use genetic modification technologies to enhance or treat humans. What this thesis has done, however, is helped to put this bioethical conundrum in a better scientific context. Before we start concerning ourselves with the extent to which we should genetically modify humans, we should pay closer attention to the science—that is, to what extent do chaos and emergence have an effect at the genetic level? The complexity of human development is already well established. But, when we add the possibilities of chaos and emergence to the discussion, it raises questions about whether we will ever be able to modify humans to any significant, predictable extent. Chaos and emergence may represent insurmountable barriers—at least for the next few decades, anyway. Questions related to whether and to what extent we should modify humans are important bioethical concerns and these discussions should continue to take place but the arguments presented for and against must be grounded in scientific reality.

To their credit, the authors writing in this area appear to be the most open to the idea that strong genetic determinism is uncommon and that there are other factors involved. Resnik and Vorhaus (2006) write that strong genetic determinism is rare and that the interplay between nature and nurture is complex and interdependent. They state that it is unlikely we will ever achieve anything through genetic modification that could be "construed as mastery (p.51)" because there is limited causality between genotype and

phenotype. Culbertson (2009) takes this view even further by suggesting that biological systems may be nonlinear, in which case, it would be unwise to make modifications because "we could have absolutely no idea of the outcome (p.144)." He continues on to state that for germline modifications any "significant degree of predictability is impossible (p.144)." Culbertson's view is extreme. While we may never be able to predict the effects of chaos—which are, by definition, unpredictable—it is not unreasonable to believe we may come to understand emergence more fully.

McFee (2000) also acknowledges the nonlinear nature of biological systems. In reference to the significance of sensitive dependence on initial conditions, McFee notes: "... being slightly adrift in one's grasp of initial conditions can lead to being hugely wrong in one's predictions (p.155)." To some extent, Rosoff (2012) may also be alluding to the role of factors other than nature and nurture. He argues that one of the main issues with genetic modification is reification, that is, describing the trait to be modified in terms of genes, proteins and enzymes. Culbertson and McFee, in particular, seem aware that other factors are at play but perhaps do not have the appropriate terms to describe what they have witnessed. This thesis has aimed to demonstrate that the concepts Culbertson and McFee describe are emergence and chaos. Rosoff's concerns with reification may also have something to do with emergence and chaos. Culbertson alludes to the emergence of new principles at higher organisational levels and both allude to the presence of chaos. It is the work of authors such as these that may help to give the concept of genetic determinism greater context and ensure that bioethical discussions related to genetic modification are based on strong scientific knowledge.

As with genetic testing, the role of the media is also relevant to the issue of genetic modification. Lucassen's concern for the media's portrayal of 'the gene for' features in the writing of Resnik and Vorhaus (2006), Birch (2005) and Hindmarch (2000). Resnik and Vorhaus state that strong genetic determinism is commonplace in "popular culture, the media and [politics]" with talk of 'the gene for obesity' or 'the gene for alcoholism.' This point has been illustrated very recently with a study indicating a 'gene for leadership' (De Neve et al, 2013). Birch (2005) argues that genetic modification is presented in the media "as a *fait accompli*," while Hindmarsh (2000) states that biology is presented as a closed, predictable system. The issues and solutions in regards to the media's reporting of genetic modification are the same as for genetic

testing. It is critical that the reality of genetic determinism is reported accurately because the news media provide an important source of information for the public. Without accurate information, we cannot have informed discussion about the bioethical concerns related to genetic modification.

Kass' (1997) argument from freedom states that genetic modification interferes with an individual's ability to make free choices in relation to the modified gene. In a similar vein, the argument from giftedness put forth by Kass (1997) and Sandel (2004) states that genetic modification fails to appreciate the gift of life. Further, Sandel's (2004) argument from authenticity states that a genetically modified individual cannot take credit for their achievements because they are not their own, rather, they are the product of a modified gene or genes. All three of these arguments are launched from a position of the factual nature of genetic determinism. As we have seen, modifying a gene is not necessarily going to have the intended outcome. Therefore, a case could be made that it would be morally irresponsible to modify an individual when there may be limitations to how certain we can be of the outcome. If this is the case, Kass and Sandel's concerns are moot. These may be interesting thought exercises or philosophical discussions, but we must be careful about ensuring these discussions are conducted in the appropriate scientific context. We cannot, and likely never will, be able to modify an individual to any significant extent because of the complexity of biological systems. This complexity is due to a number of factors, among which, are chaos and emergence. Kass and Sandel may have legitimate concerns, but they are vastly premature and potentially alarmist.

4.10 Cloning

Lutz (1997) acknowledges there are still a number of barriers in the pursuit of cloning. She states that a clone will not have the influence of mitochondrial DNA, nor will it be subject to the same environmental influences or damaged DNA as its progenitor. Despite these issues, there are authors writing in this area who believe the barriers to cloning can be overcome. Campbell and colleagues (2005) argue that epigenetic challenges can be overcome with further knowledge and, while they acknowledge the role of "other cytoplasmic factors (p.263)" in development, the group remain optimistic that we will unravel the mystery. For Campbell and colleagues (2005), further

understanding “the control of cell differentiation and maintenance of the undifferentiated cell (p.263)” will “inevitably lead to (p.263)” greater success in the pursuit of cloning. Although Campbell and his team have demonstrated awareness that there are technical limitations in our pursuit of cloning, the group has underestimated the complexity of the problem. As Neumüller and Knoblich (2009) and others (McMurray and Thorner, 2009; Koch and Schaechter, 1962; Kœrn et al, 2005) argue, the first cell division results in unequal partitioning of the cell contents and, therefore, the resulting cells will not be identical. This view is supported by Raser and O’Shea (2005) who argue that the chaotic distribution of cell components obviously has an effect on gene expression. It is unlikely that any amount of additional technical skill and knowledge will allow us to overcome the effects of chaos in cell division. In the context of this thesis, Campbell and colleagues’ (2005) assertion that greater success in cloning is “inevitable” seems ill informed.

There is great support in the literature related to cloning for the principle of genetic determinism. At the highest level, The President’s Council Report on Bioethics argues that cloning would interfere with individuality because genetic uniqueness is important to identity. Caulfield (2001) notes that the media’s misrepresentation of the capabilities of genetic technologies does nothing to quell the myth of genetic determinism and argues that banning cloning on grounds that it threatens individual identity would only serve to further entrench genetic determinism. Hopkins (1998) states that the media has led the entire debate surrounding cloning, from dispensing scientific knowledge to shaping ethical discussion for both the public and legislators. The critical issue here is the extent to which the notion of genetic determinism underpins the views of the general public and, more importantly, policy decision makers. Policy, legislation and bioethical discussion must all be based on strong science in order to fulfil their purpose, that is, to ensure individuals live the safest, healthiest, most fruitful lives possible. This thesis has demonstrated that much of the literature surrounding cloning (and, in fact, all of the genetic technologies discussed) is not based on strong science because it fails to take in to account the significance of chaos and emergence, therefore, the public, legislators and bioethicists are at risk of making ill-informed decisions in this area.

4.11 Summary

What this thesis has aimed to demonstrate is that nature and nurture provide an incomplete picture of how we come to be who we are. This is largely because the arguments presented rely heavily on the principle of genetic determinism. As we have seen, applying the principle of genetic determinism is not straight forward when one considers complex systems, specifically, complex biological systems such as the human body. In simple systems, there is a linear relationship between input and output which makes deterministic predictions possible. However, in complex systems, at each level of complexity, new properties and laws of governance appear to emerge. This nonlinear relationship means knowing the initial conditions, such as the components of a cell or the role of a particular neuron, does not necessarily give you the ability to make predictions about the end state. As Anderson (1972) put it, "The ability to reduce everything to simple fundamental laws does not imply the ability to start from those laws and reconstruct the universe (p.393)."

Emergence is one of the reasons why genetic determinism is not clear-cut. The emergence of new properties at each level of hierarchical organisation is demonstrated on the grandest level by the emergence of Newton's laws "at the macroscopic limit (Laughlin, 2005, p.32)." Applying the principle of genetic determinism is further complicated by the presence of chaos (or stochasticity) in complex systems. In light of the growing evidence for emergence and chaos, it is evident that a new model of scientific discovery is necessary. The reductionist approach may continue to be a necessary and logical starting point because, of course, when unravelling any mystery it is important to have an understanding of the individual components. However, difficulty arises when we try to make predictions based on our knowledge of the components. What is required to manage the challenges associated with emergence is a new way of thinking. The nature of this new way of thinking is likely to come in the form of self-organisation and systems biology. Systems biology, as the name suggests, takes a system-based approach in an effort to recognise the principles of emergence and the changes in dynamics that are witnessed at different levels of the organisations hierarchy. This may involve a more holistic approach or collaboration between specialties, such as biology and genetics.

This thesis has examined three specific types of genetic technology in an effort to demonstrate the pervasive and wide-reaching consequences of a long-held belief in the factual nature of genetic determinism. However, the implications of this thesis should not be considered to be limited to these three technologies. The fallacy of genetic determinism has wide reaching implications for bioethical discussion. Take, for example, personal responsibility and free will which are critical to many bioethical debates (artificial life, brain-computer interface, informed consent, eugenics, neuroethics, psychosurgery and transhumanism, to name a few). A belief in genetic determinism, as it exists in the literature and media today, is reinforcing the idea that there may be "genes for" certain diseases or conditions—even though this view may be in the minority in the bioethics literature. This view has the potential to limit the extent to which individuals feel responsible for their own health or empowered to protect themselves from certain health outcomes. Genetic determinism also has implications for legal cases where an individual, or their lawyer, may try to mitigate their offenses by claiming a genetic predisposition. Perhaps my own story is the best illustration of the fallacy of genetic determinism. It would have been very easy for me to forgo personal responsibility and assume that because none of my predecessors have amounted to anything, nor would I. I could have believed that because I come from a poor genetic pool that there weren't many options available to me. I imagine there are a great many other individuals in a similar situation, looking at their families, and believing they will never amount to much. This is why the fallacy of genetic determinism must be discussed. We are greatly influenced and restricted by our genes, this is undeniable, but we are not entirely at their mercy.

In closing, it should be noted that I am not intending to overstate the significance of emergence and chaos. My belief is that nature and nurture are, by far, the most important indicators, and predictors, of who we are. What is important, however, is to recognise that two factors alone to not give a complete picture of human biology and behaviour. However small the influence of emergence and chaos may be, it is an influence and must be given due consideration, nonetheless. Moving forward, it will be important to find a way to take concepts such as chaos and emergence in to account in a scientific manner.

5 Conclusion

The aim of this thesis was to question the validity of the doctrine of genetic determinism in light of the observation that my own life story suggests the nature versus nurture dichotomy may be inadequate. By the criteria of nature and nurture alone, it would be fair to say that I have exceeded any predictions that might have been made about me and what I would become. My aim, therefore, was to ask whether there is a third option (or options) that should be included in the debate and in doing so, question the validity of genetic determinism which is so critical to the "nature" component of the nature versus nurture argument.

5.1 Limitations of this Thesis

The limitations of this research are twofold. The first limitation concerns the issue of whether the presence of chaos can ever really be proven. If someone were to claim that a seemingly chaotic system, such as Elowitz's (2002) population of *E. coli* or the distribution of flowers in a garden, was actually operating based on a set of rules we just do not understand or do not yet have the technical capability to see, I could not prove they were wrong for one cannot prove a negative. However, based on the evidence presented in this thesis, the idea that chaos is present in all systems stands up to intellectual scrutiny. Elowitz (2002), Kimura (1968, 1989) and Lorenz (Gleick, 1987), all demonstrated phenomena which, it is reasonable to conclude, are due to the presence of chaos. Similar issues arise when considering emergence. This is a very new and speculative field of inquiry and, so far, the evidence is scarce. While the evidence for emergence may be very interesting, it is likely too early to be drawing any significant conclusions about its role. We can therefore conclude that emergence is not the third option.

A further limitation of this thesis is its relative brevity. The presence of chaos and emergence in biology is a relatively new concept and very little research exists on these subjects. This, of course, does not mean that these concepts are wrong, simply that this is a budding area of research. As with any new idea, one must work harder to prove its relevance than might be necessary when working with knowledge that is commonly accepted as fact. It remains to be seen whether the goals of this thesis, exploring such a new area of biology and applying it to bioethics, could be achieved in such a modest

research project.

This thesis has shown chaos and emergence to be interesting and exciting fields of study that warrant further research. However, additional research in to these areas is likely beyond the scope of traditional bioethics. A useful extension of this research would be to examine further areas of bioethical study that rely on genetic determinism (such as those explored briefly below). An ideal starting point for this research would be to make contact with the researchers cited herein and put to them questions about whether their results are based too greatly on speculation or whether there could be any other interpretations. In addition, more evidence could be sought from a greater variety of sources to support the claim that genetic determinism is inadequate. The goal of this additional research would be to create a more complete picture of the extent of misinformation relating to genetic determinism and how this misinformation is affecting the legitimacy of associated bioethical discussion. An additional extension of this research would be to apply the same model of inquiry to other bioethical principles or doctrines, in other words, to question whether any of our other bioethical tools are based on poor science. This research, too, would help to build a more robust and informed information base from which bioethical discussions and assessments could be launched. The topics examined in this thesis may also benefit from examination in relation to the concept autopoiesis. This may offer an alternate way of making sense of self-organisation.

5.2 Significance for Bioethics

As this thesis has aimed to demonstrate, many arguments, for or against, particular opinions or technologies are launched from a position of the factual basis of genetic determinism. In chapter three, these arguments were analysed in detail and the extent to which a belief in genetic determinism continues to pervade modern thinking was shown. What is alarming is that this belief is wide reaching from scientific literature to government commissioned documents (President's Council on Bioethics report, 2004) to the general public. The research provided by this thesis is significant for bioethics because it demonstrates that one of the tenets of many bioethical discussions—from genetic technologies to neuroethics to vulnerable populations—is based on a poor

understanding of the relevant science. This is of particular concern in regards to genetic technologies because our technical capabilities in this area are only going to improve. We cannot allow people—whether they be legislators regulating the use of a technology or an individual deciding whether to undergo a particular procedure—to make decisions about genetic technologies, based on incorrect assumptions about the nature of genetic determinism. The goal of bioethics is to decide what is morally acceptable or, in other words, what we do and do not want for ourselves in regards to the biomedical field. It is dangerous to make appropriate assessments to this end based on misinformation; therefore, the fallacy of genetic determinism must be addressed. We cannot have informed discussions about a particular technology or decide whether it is morally acceptable based on misunderstandings about science.

5.2.1 Neuroimaging

Increasingly, neuroimaging techniques are being used for a wider range of purposes. Alongside the traditional use of the medial diagnosis of disease, functional magnetic resonance imaging (fMRI) is gradually being seen as a technique that may reveal more about humans than originally thought, namely, predicting behaviour. Researchers at the University of California, Los Angeles (Falk et al, 2010) studied participants' "neural response to persuasive messages (p.8421)" in an effort to understand whether neural response is an indicator of "complex real world behaviour (p.8421)." Participants were exposed to persuasive messages about the benefits of sunscreen use and the researchers used data collected from their fMRI results (taken from the medial prefrontal cortex and precuneus) to predict their behaviour over the coming days, that is, whether they would increase their use of sunscreen. The group reported that using this technique they were able to successfully predict behaviour "spanning weeks (p.8421)" in advance. Soon et al (2008) report similar results using techniques to measure brain activity. The group found they were able to predict motor movements (when a subject intended to press a button) ten seconds before the subject made the conscious decision to move. Using these techniques to predict behaviour relies heavily on a belief in determinism and the technology is already being applied more widely to make predictions about human obedience (Cheetham et al, 2009) and psychopathy (Sato et al, 2011). These studies raise questions about what should be done with this information once we can make

predictions about an individual's behaviour. It seems most likely that the clinician or scientist administering the test would tread carefully here. If an individual is discovered (by testing alone) to have aggressive tendencies, for example, the clinician may recommend counselling or education on strategies to cope with stress. In some cases, treatment with medication may be indicated. Taken to the extreme, one could ask whether the state should make decisions about that individual (and their future) based on this information. While this is an interesting thought experiment, we should be cautious in our estimates of how far away this future is—if it ever arrives. If, somehow, we do end up here, it is unlikely the state would have access to this information in any capacity that would allow anything stronger than recommendations being made to the individual or the individual's parents. To think that the state might begin locking up children on the grounds of an aggressive future having been predicted by neuroimaging technology is fanciful. Results of such tests should always be interpreted with caution, especially in light of the complexity of genetic determinism.

5.2.2 Violent or Criminal Behaviour

Determinism also plays a role in recent investigations into violent and criminal behaviour. Brower and Price (2001) performed a meta-analysis of studies associating frontal lobe damage or dysfunction with violence or crime. After examining 69 reports, they conclude that there is a strong association between "focal prefrontal damage and an impulsive subtype of aggressive behaviour (p.720)." Studies looking at the levels of expression of monoamine oxidase A (MAOA) have found correlations between low levels of the protein and a higher incidence of antisocial behaviour (conduct disorders, antisocial personality symptoms and violent offending) (Caspi et al, McDermott et al, 2009). These studies have resulted in the MAOA gene being given the colloquial name "the warrior gene." Barnes et al (2011) used data from 4,000 people taken from the National Longitudinal Study of Adolescent Health to consider whether "genetic factors could be a strong predictor of which path you end up on (p.944)." The group concluded that "genetic influences in life-course [persistent] offending were larger than environmental influences (p.944)." The issue with this line of research is that is raises questions about what this information will be used for. If we know an individual has an inclination towards violence, what should we do about it? We could provide

interventions—counselling, medication—but where does it end? Should a child with low levels of MAOA expression be allowed in the same classroom as children without the gene? If you have low levels of MAOA expression, should your career choices be limited by the state? If there is a chance we are wrong, however small, can we afford to ever write anyone off entirely?

This thesis has sought to argue that there is always a chance, however small, that we may be wrong about our predictions made on the basis of genetic information. In relation to violence and criminal behaviour, the issue becomes the extent to which our inability to be 100% about certain facts is relevant. The line might be drawn where the consequences of a decision are extreme. For example, if an individual has low levels of MAOA expression, it would do no harm to offer counselling or medication. But, limiting an individual's career options is an extreme intervention. In this case, this thesis suggests we must weigh the decision carefully. At some point in the future, we may decide it is still worth limiting the individual's career choices, just in case, but this decision should be weighed carefully in light of the scientific evidence presented here. If we develop more evidence to support the threat low levels of MAOA supposedly pose, it may be more important to prevent an affected individual from becoming a teacher than it is to allow them the freedom to choose their own career because of the impact of chaos on genetic determinism. In any case, the grounds on which we make the decision must be clear and take into account an accurate understanding of the science involved.

5.2.3 Chemical Castration for Sex Offenders

In 2009, Polish President, Lech Kaczynski, gave the green light on a law that allows the mandatory chemical castration of some sex offenders (Grubin and Beecg, 2010). In 2012, mandatory chemical castration was introduced in Moldova for anyone convicted of offending against a child under the age of 15 years (BBC, 2012). Forced chemical castration has also taken place in South Korea on an individual convicted of four counts of rape or attempted rape on young girls (CNN, 2012). These crimes may evoke a visceral reaction and prompt immediate calls for severe punishment, but chemical castration as a punishment assumes genetic determinism. The assumption made by

decision makers in these cases is that these offenders have offended in the past and will continue to do so without drastic intervention. In many cases, this assumption may be correct, but the argument is launched from a position of genetic determinism. If genetic determinism is not as straight-forward as these decision makers might hope, it would be highly unethical to chemically castrate even the most vile of offenders. This example suggests genetic determinism may feature more strongly in some cases than others. Using the measurement established in the previous example—that the presence of chaos may be more significant when the consequences of a decision are extreme—it seems clear that chemical castration would constitute extreme. The risk here is that an innocent person is irreparably damaged for the rest of their life. This thesis has aimed to demonstrate that genetic determinism is not straightforward, due in part to the effects of chaos. Chemical castration may be one example where the risk that genetic determinism is not well understood outweighs any potential benefits to going ahead with the procedure.

5.2.4 Mental Illness

A considerable body of research exists on the extent to which mental illness (depression, anxiety, schizophrenia) can be predicted based on genetic factors. Kendler et al (1995) studied the lives and family histories of female twins. Based on the results of interviews and questionnaires, the group concluded "genetic liability ... had a significant impact on the risk of onset of depression." Wong et al (2012) examined the interactions of multiple genetic variants and developed a predictive framework that may be used to predict susceptibility to major depressive disorder. In an effort to describe a method of prediction of schizophrenia, Ayalew et al (2012) applied a convergent functional genomics (CFG) approach "to identify and prioritise genes involved in" the disease. The group report significant genetic overlap with other mental illnesses and claim to demonstrate how "the top candidate genes [identified by their study] can be used to generate a genetic risk prediction score." The claims made by all of these researchers rely heavily on genetic determinism. The issue here is on what grounds should we make predictions about the likelihood of an individual developing a mental illness? Should we ever attempt to predict whether someone will become mentally ill and what would this information be used for? There seem to be obvious benefits to

knowing whether or not you may become mentally ill. For example, this knowledge may give an individual an increased appreciation of the importance of good diet, regular exercise, the importance of work/life balance and implementing a good support network so they may be better placed to deal with the disease should they become unwell. In addition, the individual and their family will have the opportunity to become better educated about the disease and what to expect. In this sense, predicting mental illness (or any disease) may be a good thing. In the context of this thesis, the problem is when those predictions are taken too far. Clinicians working in this area must be careful (and likely already are) to fully inform their patients of the likelihood that a particular disease will develop. Until we understand the effects of chaos, the chances of a disease developing may never be 100%.

5.2.5 Synthetic Biology and Artificial Life

In 2010, Craig Venter announced to the world that his group had successfully created synthetic life—"the design, synthesis and assembly of the 1.08-mega-base pair *Mycoplasma* mycoides JCVI-syn1.0 genome starting from digitized genome sequence information (Gibson et al, 2010, p.52)." While not quite "creating life" per se, the group did manage to successfully create a self-replicating bacterium with a completely synthetic genome. Scientists at MIT and Stanford are working to create libraries of standardised biological components which can be added to a bacterial chassis in different combinations to create novel bacteria (The Economist, 2010). George Church, at Harvard University, is trying to create ribosomes which are critical cell components responsible for making protein (The Economist, 2010). Elsewhere, Floyd Romesberg of the Scripps Research Institute has expanded the DNA alphabet by creating two additional bases which can be incorporated alongside the four naturally occurring ones (The Economist, 2010). It is hoped that techniques developed in this field may be able to be used to develop antimalarial drugs and vaccines more cost effectively and help with the clean-up of toxins from the environment (Venter, 2008). The creation of life (even if only on the scale of bacteria) relies clearly on a belief in genetic determinism, that is, if we develop a bacterium with certain genes it will function in a certain way. But what are the implications if that assumption is incorrect? Where healthcare (and, to

a lesser extent, the environment) is concerned, the results of this assumption could be troublesome.

5.2.6 Real World Implications of this Thesis

The real world implications of this thesis may be thought of as small, but potentially significant. The overarching issue, which affects all of the technologies and procedures discussed, is that we cannot make informed decisions about whether something is ethically acceptable based on misunderstandings about the science. Individuals making decisions about whether to undergo a particular genetic test or figuring out how to interpret the results of such a test, must be informed about the limitations of genetic determinism. In this situation, the results of this thesis suggest the physician or genetic counsellor should set clear expectations with the individual about the certainty of the information revealed. It should be made clear to these people—patients, families, students, the public, colleagues—that test results indicate a likelihood (potentially, a very strong likelihood) of a particular outcome eventuating but, in all but a few situations, it is not a certainty.

In the cases of genetic modification or cloning, anyone claiming to be able to improve the traits of an individual should tread carefully. It is likely (due to the already well-established influence of the environment in human development) that these individuals would proceed carefully anyway and take considerable caution in explaining the limitations of any procedure. However, acknowledging the presence of chaos in biological systems means these cautions should continue to be taken seriously, even when we think we understand the technology completely.

Similar cautions apply for anyone attempting to predict behaviour whether through genetic screening to reveal particular genes for aggression and mental illness or with the use of neuroimaging technology. These technologies may yield useful insight in to the individuals involved—both for the individuals themselves and those around them—but the risk that we are wrong in our predictions must be weighed very seriously. The clinician delivering the results of these tests must frame the data carefully. Individuals who are shown to be inclined towards aggression, for example, may be offered

counselling and medication but we must not label these people and consign them to society's margins.

Mandatory chemical castration is a very challenging topic from an ethical standpoint. We each have a right to feel safe in our homes and out in the community and there is immense cost—financial and emotional—associated with recidivist offending. However, I believe the risk that we are wrong about an individual's future behaviour in even a few cases is too great. The situation would be very different if the prisoner was a willing participant, but state imposed chemical castration is a step too far. The fact that an individual has committed a crime does not give the state the right to interfere with that individual's body, especially when, as this thesis has sought to demonstrate, there is a chance we may be wrong about how that individual will behave in future.

Our assumption that genes will function as intended is well demonstrated by synthetic biology and the intentions of those working in the field to develop healthcare and environmental solutions. The risk that we may introduce a bacterium in to the environment that does more harm than good is very real. Similarly, it would be disastrous if a vaccine did not function as intended. Fortunately, these technologies are very much in their infancy. It is likely to be years before any serious results are achieved. But, when we are capable of synthesising our own genetic products, testing and containment procedures will undoubtedly be rigorous and chaos should be a small part of the reasoning for this decision.

Realistically, in most instances, the likelihood of chaos playing any meaningful role in an individual's development or behaviour is very small. However, if chaos were to play a meaningful role, the consequences could be immense—as my own story may illustrate—and it is for this reason that we cannot ignore chaos. This is very similar to the caution anyone about to undergo surgery receives. Even if the patient is only to have a tooth removed, they are always cautioned that there is a risk something may go wrong. Without fail, the surgeon cautions that there is always a risk, especially with general anaesthetic (I know this from personal experience of having a tooth out under general anaesthetic!). It is rare, however, that anything does go wrong and for a vast majority of people the benefit of the surgery outweighs the risk. A further comparison can be found in our legal system. When someone is convicted of a crime and sent to

prison, we know there is always a risk that they may be innocent. In most case, we like to think the risk is small, but it is still a risk. As a moral society, we have decided to accept this risk because the good outweighs the bad. These principles can also be applied to the results of this thesis. There may be legitimate benefit to genetic testing, chemical castration or the prediction of major depressive disorder. When deciding to go ahead with one of these tests or procedures, it may be that the benefits—of knowing whether you will develop breast cancer, of having one less sex offender in the community, of knowing whether you will develop depression—significantly outweigh any potential risks, but we must acknowledge, nonetheless, that the risk that we are wrong about a particular genetic outcome does exist. Of course, unlike in the case of a medical procedure or legal trial, the risk that chaos will have a detrimental impact is considerably smaller.

5.3 So, is there a Third Option?

In closing, it is important to consider whether this thesis has actually achieved the aims it set out to satisfy. It is clear from this thesis that genetic determinism is not as straight forward as we might like; the work of most of the authors surveyed in this thesis supports this assessment. The doctrine of genetic determinism ignores the interdependent relationship between our genes and the environment. This complex relationship means that our genetic makeup affects our environment and our environment, in turn, affects our genes.

What none of the authors surveyed in chapter three have taken into account in their arguments is the influence of chaos and emergence. Where these authors consider only genes and the environment to be relevant, this thesis has sought to ask whether chaos and emergence should also be considered. It would be ignorant to claim that this thesis has proved, beyond doubt, that the presence of chaos and emergence in nature render the doctrine of genetic determinism a fallacy; misleading because there is obviously much more at play here. How we come to be who we are is obviously the result of complex interplay between a number of factors and it is not chaos and emergence alone that prove genetic determinism to be a fallacy. What this thesis has demonstrated, however, is that there is enough evidence in support of the presence of chaos, at least,

to warrant further investigation. This is a very new areas of research which means there is only a small body of evidence to draw on, however, the evidence that does exist is compelling and stands up to scientific scrutiny.

As to the second aim of this thesis—to assess whether a third option should be added to the nature versus nurture debate—the answer is quite simple. Yes, the nature versus nurture dichotomy is inadequate—but, perhaps not significantly. The third option is not emergence. In my view, for all intents and purposes, emergence should be considered an element of both nature and nurture. In this sense, emergence should be thought of as exerting its influence within the system; it is a part of the system, like gravity or any one of the other laws affecting how the universe is governed. While we do not currently understand emergence, there may come a time when we are able to further quantify this aspect of our biological makeup—and, in fact, the universe as a whole. Emergence is one of the reasons we cannot currently look at the initial conditions within a system and predict its end state, but we may overcome this obstacle with further study in the areas of systems biology and self-organisation. As our understanding in these areas increases, it is likely that we will begin to see how the laws of emergence relate to, and influence, nature and nurture.

Where emergence should be considered part of nature and nurture, chaos should be thought of as the parts in between; the forces acting between and around nature and nurture. Even so, chaos is not the third option, either. It is separate from nature and nurture, but the evidence presented here does not go as far as I would have liked towards establishing chaos as the third option. It is an influence and, in some cases, it is a very important influence, but, on the whole, the role of chaos is small and we manage to work around it. Chaos, by definition, cannot be quantified or anticipated. With further study, we may learn more about the prevalence of chaos but, unlike emergence, we will never be able to foresee or plan for its effects. The direct actions of chaos may be small—affecting the distribution of mitochondria between cells during cell division or the order in which reactions occur within the cell—but, as this thesis has demonstrated, sensitive dependence on initial conditions means the results of chaos can be disproportionate. These small chaotic actions may add up to small, inconsequential results or they may add up to an entire human being having defied the odds at every juncture.

This leads, of course, to consideration of my personal aim in writing this thesis: to try and explain how I turned out to be an above average human being when both nature and nurture gave me so little. To chalk a particular result up to chaos may be scientifically unsatisfying but, personally, it is a lot closer to an answer than I was twelve months ago. If my story illustrates anything it is that while the role of chaos may be small, small does not mean insignificant.

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