A DEVELOPMENTAL SYSTEMS APPROACH TO GENETIC DETERMINISM

by

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(Under the direction of Scott Kleiner)

ABSTRACT

This paper evaluates developmental systems theory for its ability to combat genetic determinism. I describe the major tenets of a developmental systems approach. I argue that three factors contribute to genetic determinism (i) an intentional view of genetic information, (ii) a historically contingent and a DNA-centric gene concept, (iii) a tendency to assign single causes and centralized control to complex events. In contrast, a developmental systems approach provides causal views of information, a more holistic gene concept and a dynamic explanation of complex systems.

INDEX WORDS: Developmental systems theory, Genetic determinism, Genetic information, Gene concept, Dynamical systems

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CHAPTER 1

INTRODUCTION

In this paper I wish to explain and assess developmental systems theory, or DST, for its ability to combat genetic determinism. Genetic determinism might be combated on grounds that it can be proved incorrect through empirical research. It might also be combated on grounds that genetic determinism comes from misunderstanding scientific terminology. To the extent that it is an empirical question, I cannot decide here if a DST or a genetic program approach to development is more scientifically accurate. But I can discuss whether a redescription of development in DST terminology might be beneficial by avoiding genetic determinism. Terms such as 'genetic information', 'coding', 'gene for a given trait', 'transcription', and 'translation', though they look like everyday language, are all scientific terminology. The mechanics of processes such as transcription and translation are fairly well understood yet the interpretation of those processes vary. When they are described using language that has both a technical and colloquial meaning, the meanings overlap and promote a genetic determinist interpretation of humans and human behavior. For this reason, a redescription of scientific work in DST terms may be more accurate and beneficial. I want to see if DST is a beneficial redescription.

In order to assess DST, I examine the uses of the terms 'gene' and 'information' since these are the prime examples of scientific terminology that are problematic. Current gene concepts are ambiguous and do not support a simple gene for trait locution.

The ambiguity of the term has not always shaken the belief that genes control development and produce traits in scientific circles. The ambiguity has reached far less into public knowledge. Simplistic use of an unclear term by either scientists or laypeople promotes genetic determinism. The status of information in biology is extremely unclear. Here a precise term from communications theory has been taken into biology with a loss in precision. Even the status of the term as metaphor or literal description is controversial. Claims of genetic information can also promote genetic determinism. The confusion over these words suggests that a redescription of scientific work in different terms would be helpful. DST claims that sole control over development by genes is incorrect. The disciplines of developmental biology and genetics have each attempted to describe development with genetics taking a genecentric view and developmental biology a more holistic approach. Until recently genetics has had more explanatory success. Instead of joining the battle over whose research is more legitimate, I want to approach the problem from another angle. It is easy to assume that a complex system needs centralized control to produce coherent behavior. Work in AI shows this assumption is untrue. Comparing biology to artificial intelligence does not prove that biology functions the same way that AI does. However, the comparison can show that it is possible to have organization without central control. Genetic control does not need to be clung to because it is the only possible explanation.

CHAPTER 2

GENETIC DETERMINISM

A strict definition of genetic determinism states that a certain gene produces a certain trait no matter what other genes are present in the organism and no matter the environmental conditions (Kitcher 2001, 397). The strict definition does not include the types of traits we wonder about such as diseases, intelligence, aggression, and sexuality. Because we try to answer these questions in terms of genetics, the strict definition is too narrow to encompass what we mean by asking, "Do I have the gene for breast cancer? Will I get this disease if I carry the gene?". We want to know how much and how a gene contributes to the development of a trait. The question is important to protect ourselves from disease and to create a society we consider just. DST may give us an alternate way of answering these questions and achieving self-understanding.

Weaker views of genetic determinism, including the 'gene for specific trait' view, acknowledge interaction between the genes and environmental conditions. These views are still characterized as genetic determinism because of the type of interaction between genes and environment. In this version of interaction the genes and the environment provide very different needs to the developing organism. Genes provide the basic self of the organism, described as a plan, blueprint, recipe or set of instructions. The genes carry information about the phenotype encoded in base pairs of DNA. Their information is intentional and symbolic. DNA is intentional because its form has been selected by

natural selection for its effect, or its information content. DNA is symbolic because there is no necessary connection between a codon and the amino acid it codes for (Maynard Smith 2000). This relationship is an accident of evolution. As a complement to the genes, the environment provides the raw materials needed to realize the organism. The environment includes nutrition for the developing organism which is sometimes provided by the organism's parents, and it provides shelter from harmful aspects of the environment such as weather conditions or predators.

Taken to a genecentric extreme, the environment can include the body of the organism itself. It may appear odd to partition the organism such that one part of the organism is the environment for another. However, if we conceive of the environment as physical location in all its aspects, the immediate environment for the genes is the cell. More broadly it is the organism as a whole. We should notice the consequences of partitioning environment and organism into environment and genes in such a way that the whole body of the organism (minus the genes) is the environment. By relegating the body to the status of environment it makes the genes, and not the rest of the body, the true organism. Remember in this view the genes are the primary cause of the organism and the environment is only the materials out of which the organism is built. In dividing genes as instructions from body as raw materials, the organism is better identified with the genes than it is with the raw materials. Genetic instructions are unique to an organism while physical building blocks are not. When environment includes body, the organism itself must be something other than the body.

The history of our genetic understanding includes traces of both genetic determinism and anti-genetic determinism. Certain of our gene concepts, including the

classical mendelian, molecular, and evolutionary concepts contain the potential for genetic determinism. Every aspect of these concepts is not deterministic, nor do these concepts have to be used in deterministic ways. Certainly not everyone who uses these gene concepts is a genetic determinist. However, we need to differentiate the deterministic aspects from the non-deterministic aspects. Identifying this history and patterns of thought will make it possible to rethink our gene concepts without implicit genetic determinism.

As our knowledge and understanding of genetics has increased our concept of the gene has changed. Despite changes, the classical or mendelian gene concept, the molecular gene concept, and the evolutionary gene concept have all been used to tell genecentric developmental or evolutionary stories. Although the word 'gene' was thought up in 1909 by Wilhelm Johanssen, our modern understanding of heredity began with Mendel. The classical or mendelian gene concept came to associate the genes with the chromosomes, but had no understanding of a gene on a molecular level. The gene was a factor that created a difference in phenotype. The molecular gene concept understands a gene as a section of DNA. DNA contains a code that specifies a certain protein. The evolutionary gene concept understands genes as the one unit that is faithfully replicated every generation. It is the object of natural selection through its effects on the phenotype.

Early studies of heredity can be understood in light of Darwin's theory of evolution. Darwin's theory explained speciation or the variety of life. Thus an explanation of the constancy of life was also needed. Darwin could explain how one species could give birth over a great period of time to a different species. Yet there was

no satisfactory explanation for why parents give birth to offspring that look remarkably like themselves. A theory of heredity had to explain how traits remained stable and passed between the generations. This need for stability lead to the assumption of "the existence of an inherently stable, potentially immortal unit that could be transferred intact through the generations" (Keller 2000, 14). There were three aspects to this stability. First, the hereditary units themselves must be stable. It was their inherent stability that created stability of inheritance. Second, the traits are stable. In each generation a particular trait, such as the color of a pea, reappears unchanged. It is this stability that requires assumption of the first stability. Finally, the developmental process must be stable. A species' passage from embryo to adult is replicated over and over with very little variation. The third type of stability was also attributed to the first stability of the hereditary units.

Along with the assumption of stability, came the assumption of centralized control. We believed a complex, organized, and stable process needed to be controlled by some central body. Oyama explains the assumption this way "Perhaps because we are creatures whose existence and survival depend on our ability to perceive regularity in our surroundings and in turn to leave our mark-our design-on them, we tend to infer prior design or intent from observed regularity" (Oyama 2000a, 12). Previously we relied on divine design for the regularity we perceived in nature. Now, this is no longer a satisfactory scientific explanation. However, the underlying need for a design has remained. Divine design has been replaced with genetic design through natural selection. The same mechanism that provided stability of development must be the mechanism for

control of development. These assumptions of stability and central control are the beginning of a genetic determinist gene concept.

In favor of Darwin's theory and against Lamarck's theory of evolution, August Weismann provided an early outline for a theory of heredity which included "the existence of particulate, self-reproducing elements that 'determine' the properties of an organism" (Keller 2000, 16). Weismann's determinants were the fundamental units of biology. The belief in a fundamental unit of heredity was also held by botanist Hugo de Vries who explicitly compared hereditary units with atoms in physics and molecules in chemistry. That is, these units were the most basic level of explanation. The rest of biology could be understood in terms of the properties of these fundamental units (Keller 2000, 18). Comparing biology to physics or chemistry is the normal working of reductionist science. On a small enough scale, all biological beings are made of molecules and atoms. Yet, biology needed fundamental explanatory particles of its own. Otherwise it would reduce to chemistry or physics. Any item chosen for the role of 'fundamental theoretical unit of a field of science' would have great power. When hereditary units were chosen as fundamental they became the source of understanding and explanation. To explain in terms of heredity was to explain completely. To explain at the gene level is to explain at the deepest level. This privileging of heredity was another source of genetic determinism.

Weismann also assumed that a full set of these 'determinants' were stored separately from the rest of the organism in what he named the germ-plasm. That way the determinants could be protected from the ups and downs of daily life on the organism and be passed on unharmed to the next generation. This neatly prevented the inheritance of

acquired characteristics, since the germ-plasm was insulated from all such acquisitions. This idea was picked up in 1896 by E. B. Wilson who said "As far as inheritance is concerned, the body is merely the carrier of the germ-cells, which are held in trust for future generations" (Keller 2000, 17). This is the beginning of the evolutionary gene concept which lately blossomed into Dawkins's theory of replicators and vehicles. The evolutionary gene concept holds that the gene is the unit of selection. It explains evolutionary change in terms of two different types of entities: replicators and interactors. Replicators are so-called because unlike other developmental or evolutionary factors, they have the ability to self-replicate. The gene, as replicator, builds the interactor and directs the interactor's physical characteristics and behavior for the replicator's own evolutionary advantage. Interactors are housings for the replicators and the means by which the replicator interacts with the environment and other replicator/interactor complexes. Generally they are the body of the organism.

Many people see the direction of the interactor by the replicator as genetic determinism. In order to combat accusations of genetic determinism proponents of the evolutionary gene concept emphasize the difference between evolution and development. The replicator is important to understanding evolution, but we must recognize that more than genetic factors are important to development. Thus many factors above and beyond genetic factors create an organism. DST claims that evolution and development are not truly separable in this manner (Gray 1992, 187). A factor that directs traits and behavior to evolutionary advantage will also affect development because developmental methods are subject to natural selection. Genes are only visible to natural selection to the extent that they direct and influence their interactors. The further from genetic control of

behavior the evolutionary gene concept goes, the less sense it makes to take genes as the units of selection. Nor will regarding genes merely as recorders of evolutionary change solve the problem. Unless the genes contain the entire organism (in the form of instructions, blueprints, or any other popular metaphor) they will not reflect the sum of evolutionary change in the organism or population as a whole. The evolutionary gene concept is not committed to the view that only genes control the phenotype (Sterelny and Griffiths 1999). However it is committed to a very close connection between genes and traits. It must also view genes as a privileged developmental resource.

Prior to Mendel heredity was explained through blending of characteristics directly transmitted from parents to offspring. That is, offspring are a blend of the characteristics of their parents. Mechanisms for the passage of traits varied, but generally included particles of the traits being passed from parent to offspring (Whitehouse 1973, 1-2). In contrast, Mendel theorized that inheritance was particulate not blended, and that factors for traits, not traits themselves, are passed between generations. Particularity can be seen at the factor and trait level. Of the peas that Mendel examined, each was either green or yellow, wrinkled or smooth not a combination of colors or of shapes. Characteristics at the trait level do not blend. When green and yellow peas were crossed all the offspring were yellow. Yet when these yellow offspring were crossed together one fourth of the offspring were green. Traits disappeared in one generation only to reappear unchanged in the next. By implication, the factors that caused the trait did not disappear or blend with other causal factors. "This is the crucial feature of his theory of heredity, that characters are not 'transmitted' directly from generation to generation as

the classical theory supposed, but that there exist discrete particles responsible for the appearance of particular character" (Whitehouse 1973, 8).

Griffiths and Neumann-Held define the mendelian gene so "the gene is a stretch of chromosome that is associated with a phenotypic difference" (Griffiths and Neumann-Held 1999). This period had little biochemical or molecular understanding of genes. Genes were identified by their phenotypic effects. The classic mendelian period is marked by understanding of genes using Mendel's laws of inheritance. The first law is the law of segregation that states the alleles of a gene separate from each other in meiosis. The second law is the law of independent assortment that states when alleles or genes segregate they do so as individuals. One gene does not influence another gene as to which gamete it ends up in. Both of these laws echo the assumptions of Weismann and his contemporaries of genes as fundamental units. As fundamental units they are particulate, discrete, and do not mix with each other.

This character/factor difference can be interpreted as preventing the inheritance of acquired characteristics or preventing environmental influence. Whitehouse explains "The fundamental difference is that on the classical theory inheritance is direct and hence the extent of transmission of a particular character to the progeny may be influenced by the character itself, whereas on the Mendelian theory the determinant of a character is considered to be in no way modified by its presence in an organism, whether it posses that character or not, and hence the inheritance of a character is thought to be unaffected by the character itself" (Whitehouse 1973, 12). The distinction between acquired and inherited traits was controversial before Mendel's work was rediscovered (Jablonka and Lamb 1995). His work did not settle the controversy, but laid the conceptual groundwork

for identifying a mechanism that explained the distinction. The separation of trait from factor began to explain the separation of acquired and inherited. When Johannsen coined the terms phenotype and genotype these too appeared to map onto trait and factor and suggested that genotypes were inherited and phenotypes acquired. The conflation and confusion of inherited and acquired vs. genotype and phenotype remains with us today. Mendel's theory postulated internal factors that created phenotypes and explained development. These factors were studied because they had causal power and they provided an explanation and predictive power where previous theories had not. However, this focus on Mendel's factors and subsequently the whole field of genetics has neglected the role of the environment and epigenetic inheritance in development. The developmental systems approach seeks to redress this balance.

The next important discovery in the classical mendelian period was the localization of genes to chromosomes. The chromosome theory of inheritance is one transition point between the end of the mendelian theory and the beginning of our molecular understanding of genes. It was important because it changed the unit of inheritance from a theoretical concept to a physical thing within the cell. Even though the gene had made the transition from theoretical unit to physical object, assumptions from its days as a theoretical unit still hung about it. The gene is the basic unit of heredity, indivisible into smaller units. The gene must be stable, individual, and permanent through the generations. These assumptions were codified into theory. Classical mendelian theory stated the gene was the basic unit of transmission, function, recombination, and mutation. This was called the bead on a string theory of the gene. Genes are the basic and indivisible units of structure, function, and mutation. Being a

unit of structure means that genes are not split by recombination into smaller parts. As a unit of function it means that only a complete gene can create a character. Segments smaller than a gene have no function. Finally it is the entire gene that mutates. As a basic unit, it does not contain smaller parts that can change (Griffiths et al. 1993).

Interestingly, the chromosome theory was controversial at first precisely because it allowed recombination. (Whitehouse 1973, 79). Stable, particulate genes must not undergo recombination. However, there are more genes than chromosomes which implies that there must be more than one gene per chromosome. As de Vries pointed out, if all the genes are to be passed on independently of one another the chromosomes must break up and freely exchange genes with one another. This recombination preserves Mendel's law of independent assortment. (Whitehouse 1973, 80).

Ironically, recombination also provides an exception to the law of independent assortment, so that the law is no longer considered true. When the chromosome theory gave genes a physical reality, it took away their theoretical perfection. Statistically speaking, genes that are further from one another on a chromosome are more likely to recombine. The closer one gene is to another the less likely it is that a recombination will happen to occur between them. Genes that are sufficiently close almost never recombine and for this reason are called linked genes. Linked genes break the law of independent assortment. This recombination theory was shown to be true with *drosophila* crosses on sex linked traits. The chromosomes do not behave as permanent stable units during meiosis in the production of gametes. Instead they break apart and exchange segments with one another. The bead on a string gene theory recovered from this recombinational

insecurity. It was assumed that only the chromosomes broke up and recombined. The genes themselves remained whole, stable and particulate just as Mendel described.

Griffiths and Neumann-Held identify the start of the molecular gene concept with the publication of T.H. Morgan's *The Theory of the Gene* in 1917. The molecular gene concept includes the discovery of DNA as the hereditary material and holds that a segment of DNA codes for a single polypeptide (Griffiths and Neumann-Held 1999). The molecular gene concept has undergone many changes between 1917 and our current conception of the gene. Early versions are reminiscent of both Mendel's theory of particulate factors and the bead on a string theory. At first the molecular concept held that a gene began in a particular location on the chromosome and continued in an unbroken sequence until it stopped. It was surrounded, but not overlapped by, other genes. Each gene produced one protein.

In 1941 Beadle and Tatum did an experiment showing that one gene produced one enzyme. They worked with mutants of the mold *Neurospora crassa*. The mutants were unable to produce all the amino acids they needed to survive and had to be supplied with supplemental nutrition to keep them alive. Beadle and Tatum found that each of three mutants was lacking a particular enzyme. Ordinarily these enzymes converted precursors into necessary amino acids. They inferred that the genetic mutations corresponded to the missing enzymes and that therefore each gene produces its own enzyme.

Beadle and Tatum's 1941 experiment was an example of two trends in genetic thinking at the time. First, it was an example of the 'gene action' interpretation of genes and development. Keller explains that 'gene action' is a way of attributing causal power

to the genes. She says "this phrase was the primary locution by which geneticists between the mid 1920s and the 1960s referred to 'the causal processes that connect the gene and the characters' and it was a way of talking that at least tacitly granted to the genes the power to act" (Keller 2000, 46). At least at first, geneticists had no idea how genes exerted their effects or even what genes were. Sturtevant proposed that there is a simple linear chain of cause and effect between the gene and its associated character. His proposition lead to Beadle and Tatum's experimental success. The picture of gene action produced after Beadle and Tatum's experiment was of each gene producing a protein and the sum of these proteins producing an organism. This interpretation encouraged geneticists to study the simplest organisms where the chain of cause and effect would be clearer and more easily discovered. The thinking of the time was exemplified by Jaques Monod saying "What's true for *E. coli* is true for the elephant" (quoted in Keller 1995, 24). This thinking ignores the context sensitivity of genes and the complexity of organisms.

Second, Beadle and Tatum's experiment was an example of the struggle between genetics and embryology for legitimacy and explanatory power in the early days of genetics as an independent field. The primary question of embryology was "how *does* a germ cell develop into a multicelled organism?" (Keller 1995, 12). Phrased this way, the question was too broad and complex to answer. Geneticists were successful in rephrasing the question into one more easily answerable. In 1932 Sturtevant illustrates the change from embryology to genetics "One of the central questions in biology is one of differentiation-how does an egg develop into a complex multicelled organism? That is, of course, the traditional major problem of embryology; but it also appears in genetics

in the form of the question, 'How do genes produce their effects?'" (quoted in Keller 1995, 14). Rephrasing the question of embryology not only made it answerable; it made it answerable in terms of gene action. With this question Sturtevant reduces all of developmental biology to a matter of genetics. The implicit assumption is that development is under the control of the genes. Beadle and Tatum's experiment bolstered geneticists' claim on embryology by demonstrating how genes might act. Although later scientists undermined the one gene-one enzyme hypothesis, we see its descendants in projects that look for the genes for behavior such as the gene for aggression. A lingering hope for simplicity makes current work more difficult to understand and interpret correctly.

Gene concepts have had to explain both heredity and development. To explain heredity, the gene needed to provide a mechanism for the passage of traits from parent to offspring. To explain development, the gene needed to provide a mechanism for the growth of an embryo into an adult. Part of the history of genetics and genetic determinism concerns the growth of the second problem out of the first. 'Gene action' talk came about before there was any notion of how genes created characters or organisms. In the absence of knowledge, geneticists had faith that gene action would explain development. They also believed that the rest of the cell would prove largely irrelevant to development. A student of Morgan's, R.A. Brink stated "the Mendelian theory postulates discrete, self-perpetuating, stable bodies-the genes-resident in the chromosomes, as the hereditary materials. This means, of course, that the genes are the primary internal agents controlling development" (quoted in Keller 1995, 7). Griffiths and Neumann-Held call this the "black boxing" of development by geneticists. Black-

boxing allowed geneticists to make great strides in understanding heredity. At the same time, it allowed geneticists to believe that development was explainable in terms of genetics without addressing the problem specifically (Griffiths and Neumann-Held 1999). When we state today that a phenotype is encoded in the genes it is a modern form of black-boxing. Thus the potential for genetic determinism was born.

The most important discoveries in the molecular period were Avery, MacLeod, and McCarty's demonstration that genes were made of DNA and Watson and Crick's discovery of the structure of DNA. The discovery of the structure of DNA was so influential because so much depended on that structure. DNA structure was supposed to explain "autoreplication, specificity, and information content" (Portin 1993). The very assumptions of stability that Johannsen wished to avoid by coining the new word gene appeared to have been proved correct. Before the structure of DNA was identified, Erwin Schroedinger suggested the structure of the genetic material must be responsible for its stability and the ability to persist through the generations. He felt this stability explained life's ability to resist entropy and retain organization. It was the difference between life and non-life. (Keller 2000, 21). Watson and Crick recognized this ability in the structure of DNA they found "It has not escaped our notice that the specific pairing we have postulated immediately suggests a possible copying mechanism" (Watson and Crick 1953a). The cause of all the excitement was complementary base pairing in the DNA. DNA has been compared to a ladder in shape, albeit a twisted one. The rungs of this ladder are made of two molecules called bases. There are four of these different bases in all; adenine, thymine, cytosine and guanine. Each base will only pair with one other specific base. A will only pair with T and C will only pair with G. This is because

of the chemical structure of the base and it also ensures that all the rungs are the same width. This type of pairing also means that if the ladder were sliced in half down the middle of the rungs, the missing half could be correctly rebuilt from the remaining half. Biologists immediately saw the potential for accurate replication.

The second consequence of the DNA structure was what came to be called the Central Dogma of genetics. It states DNA coded for RNA which was transcribed into a protein. The sequence of the nucleotides in the DNA dictated the sequence of nucleotides in the RNA and through the RNA to the protein. The process was unidirectional and once information had passed from DNA to protein it did not pass back from protein to DNA. The information or meaning of the DNA was contained in the sequence. The sequence of the nucleotides in the DNA is recapitulated in the sequence of nucleotides in the RNA that is transcribed from it. The RNA sequence in turn specifies the sequence of amino acids in the protein. The Central Dogma provides another method of separating acquired from inherited characteristics. DNA is the repository of inherited characteristics. Because information once translated from DNA to RNA cannot return to DNA, characteristics acquired after translation cannot be inherited. Although the DNA code was not broken until the early sixties (Portin 1993) it was assumed to contain information from the first.

The last milestone in a history of genetic determinism is work done by Francois Jacob and Jaques Monod in 1959 to 1961. They introduced the concept of structural genes and regulator genes. Structural genes are genes that actually code for proteins that build the body of an organism, and regulator genes that control the rate at which the structural genes do their work. Sturtevant's conception of "gene action" changed to a

new conception of genes described as a “genetic program”. Instead of each gene producing an effect through a simple chain of reaction, genes interacted with each other and their products in a coordinated network. Some genes turned other genes on and off. Despite evidence to the contrary “genetic program” came to mean a program run by genes, not on them. The instruction set for building a cell was not so simple as it had at first appeared, but the control remained firmly in the hands of the genes.

CHAPTER 3

DEVELOPMENTAL SYSTEMS THEORY

Developmental systems theory, or DST, developed in response to a genecentric, preformationistic view or development, heredity, and evolution. The genecentric view holds that genes contain information, often thought to be intentional, encoded in the bases of the DNA through natural selection. The information is in the form of instructions, blueprints, programs, or plans for the development of an adult organism from a zygote. DST has been motivated both by scientific discoveries and social concerns. The scientific discoveries include RNA splicing and editing, the behavior of introns and exons in protein production, DNA proofreading and repair mechanisms, and overlapping or nested genes. These discoveries belie the concept of an inherently stable gene whose unique function can be read from its sequence. The social concerns are about the effects of genetic knowledge and scientific language on our society. Genetic knowledge, including recombinant DNA technology, has given us great power over our genes. Designer genes are on the way to becoming science fact, not science fiction. DST warns us against believing we can find a gene for intelligence or a gene for aggression. The 'gene for' view of genetics is both scientifically problematic and may lead to morally problematic methods for solving society's ills.

There are six major tenets of the developmental systems approach. I explain them in detail below with the exception of tenet six which will only receive brief treatment in the interests of keeping the discussion to a manageable size. Even though DST holds that

development and evolution are not truly separable I am only going to address development here. The tenets are:

1. Joint determination of the phenotype by multiple causes,

2. The contingency and context sensitivity of any cause on the rest of the system,

3. Extended inheritance of each organism to include many developmental resources beyond its genetic inheritance,

4. The process of development from embryo to adult is a process of construction of traits afresh in each generation, rather than a process of transmission of traits from one generation to the next,

5. Distributed control of development instead of control by just one factor such as the genes,

6. Just as development is construction so is evolution. Organisms and environments affect each other instead of the environment forming the organism. (Oyama, Griffiths and Gray 2001).

1. Joint determination of the phenotype means that both genes and non-genetic factors combine to produce a phenotype. Both genetic and non-genetic factors are necessary and neither is sufficient by itself to construct a phenotype. The controversial point is how the organism needs both genes and the rest of the world. Instead of dividing genes and environment into separate causes of form and matter, DST asserts that form is contained in both genes and environment. The biological example often given to support this claim is the phenomena of phenocopying. A phenocopy is a phenotype that is the same as the phenotype brought about by a genetic mutation, hence the 'copy'. However, the phenocopy is not brought about by the genetic mutation, but by an environmental

manipulation. In other words, the phenocopy organism has a normal genotype with a mutant phenotype. An extremely commonly cited example is the bithorax phenotype in drosophila. This phenotype is characterized by an extra pair of rudimentary wings. It can be brought about either through a genetic mutation in a normal environment or exposing a genotypically normal fly embryo to ether three hours after fertilization (Gray 1992, 176; Gray 1996, 393; Oyama, Griffiths and Gray 2001). When we think of a genotype producing a phenotype, we think of a given genotype as typically producing a particular phenotype. There is a particular phenotype that follows from a given genotype so often, we think of the genotype as 'supposed' to make this phenotype. We identify the genotype as the cause of the phenotype because we see them together so often. In scientific terminology, we say this genotype codes for this phenotype. This type of thinking is supported by experimental work where a scientist alters a DNA sequence and an organism with an altered phenotype is the result. In other words as Oyama explains "Phenotypes, then, can 'match' (Lindzey, Hall & Thompson, 1978, p. 42) or 'correspond with' (Nash, 1970, p. 29) the genotype or not; by extension a phenotype can also match a genotype other than its 'own'" (Oyama 1981). Sometimes we see a phenotype without the genotype that is most commonly associated with it. Instead of recognizing this as an alternative developmental pathway, we label it as a copy of something it is not. That is, even though the genotype 'should' have produced a different phenotype, instead it has copied the phenotype of another genotype. Developmental systems theory asks us to see the arbitrariness of assigning a genotype to a phenotype without assigning an environment to a phenotype. In the drosophila example, both the mutation and the ether produced a bithorax fly. The precise causal process in each case was different, but each

was equally a cause of the bithorax phenotype. Dubbing one fly a phenotype and the other a phenocopy implies there is something unequal in the causation by ether or causation by genes.

2. Contingency and context sensitivity of causes is also illustrated by the phenocopy example. This is another way of emphasizing the interconnectedness of genes and environment. The issue is whether or not the genes and environment interact in such a way that they can be separated or not. The conventional view claims they can be separated while the developmental systems view denies it. Oyama et al. say "The demands of interactionism in its conventional form can often be satisfied by merely admitting that every organism must have some genes and some environment. With that out of the way, the real business of settling what is due to nature and what is due to nurture can continue" (Oyama et al. 2001). While the first tenet states that both genes and environments are equal causes of development, the second tenet speaks to their effects. The effect of any cause, like genes or environment, depends on the effects of the other causes at work in development. The interconnectedness of causes also makes it impossible to separate the causes from one another. In one environment the genes may have one effect and in a different environment they may have a different effect. In *drosophila* a normal genotype causes a normal phenotype in a normal environment. But in an environment with ether the normal genotype causes a bithorax phenotype. This means we cannot speak of the effect of the genes without specifying the environment or vice versa.

One way to deal with this problem is to assume that genes have a proper effect which a normal environment supports. We might assume the proper effect of the

drosophila genes is to produce a normal fly. When the genes' correct action is disrupted by ether a bithorax phenotype ensues. However, talk of proper effects and normal environments assumes the genes carry intentional information. When we assume a proper effect for genes we assume that genes are a different type of cause from environment. We assume they are a plan, blueprint, or contain information while the environment does not. Alternatively, we could see the genes as working together with the environment to cause of the organism's development. Genes and environment are just two aspects of the developmental system. There are also problems with identifying a normal phenotype. A good way to define a normal phenotype as one that is adaptive. Adaptiveness or lack of adaptiveness is dependent on the environment, so a normal phenotype cannot be defined independently. This negates the attempt to find an independent criterion for normality. Finally, when we speak of a normal environment we do not mean the most common environment. Griffiths and Gray explain the problem in relation to oak tree development. Most acorns rot instead of growing into trees. The normal environment, in the sense of common or usual, does not cause development. However, if we speak of a normal environment for acorn development we mean the environment that causes maturation not rotting (Griffiths and Gray 1994). Normal environments cannot be defined independently of phenotypes any more than normal phenotypes can be defined independently of environments.

Oyama asks "who is to say which is the original and which is the imitation? Is one more or less an expression of genetic potential than the other? ... And when a given genotype gives rise to dissimilar phenotypes, how do we decide which is the one that is genetically coded?" (Oyama 1981). Oyama's questions remind us that we can only see

the genotype at work through its outcome in the phenotype. The phenotype is dependent not only on the genes, but on the environment as well. This means there is no way to identify the effects of genotype alone. The effects of variations in genotype against a constant background can be specified, but that does not show the effect of the genes alone. It shows the effects of the genotype *in that environment*. Imagine that two new species of beetle are discovered. They are morphologically quite different, yet they share the same environmental niche. Surprised scientists discover that these two species interbreed and subsequently decide they are really the same species. Despite having similar genotypes, this species produces two distinct phenotypes. Both phenotypes are adaptive in their environmental niche and are replicated each generation. Griffiths and Gray give an example of a beetle species that produces two different morphologies depending on its environmental interactions (Griffiths and Gray 1998). How are the scientists to decide which phenotype the genotype codes for? Picking one or the other seems arbitrary. Assuming the genes code for a particular phenotype means that any other phenotype the genotype produces is interpreted as a mistake. If we let go this assumption we recognize that both phenotypes are a product of interaction between environment and genes.

A common statistical method for attempting to separate environmental from genetic causes of phenotype is the analysis of variance or ANOVA. The ANOVA method is designed for separating environmental from genetic causes of population variation. For instance, the average height of a field of corn might be five and a half feet tall. Not every plant in the field will be of average height. ANOVA can be used to quantify genetic and environmental contributions to explain why these plants differ from

the average. In order to calculate the percentage of genetic and environmental contributions, the individual corn plant is compared to the population average as a whole. The individual's height is also compared to the average height of plants that share the same environment but do not share a genotype. Then the individual's height is compared to average height of plants that share the same genotype but do not share an environment. The individual's difference from the population average as a whole is the sum of the individual's differences from the average genotype and the average environment.

A concrete example may help. Imagine a corn field that is planted with two varieties of corn. Half of each corn variety is given fertilizer and half is not. Blue corn with fertilizer grows 7 feet tall, while without fertilizer it grows only 6 feet tall. Yellow corn grows 5 feet with fertilizer and 4 feet without. The field average overall is 5.5 feet tall. The farmer wishes to know how much of the difference is due to the type of corn and how much to the fertilizer. The average overall height, 5.5 feet, is compared to the average height of blue corn with fertilizer, 7 feet. The 1.5 foot difference from overall average must be a sum of environmental and genetic differences. To find the genetic component of the difference, the average height of the blue corn, 6.5 feet, is compared to the overall average, 5.5 feet. Blue corn is a foot taller than the overall average. To find the environmental component of the difference, the average height of fertilized corn, 6 feet, is compared to overall average, 5.5 feet. Fertilized corn is half a foot taller then the overall average. This means the 1.5 foot difference between fertilized, blue corn and the overall average is 1 unit genetic and half a unit environmental. That is, it is 66% genetic and 33% environmental.

ANOVA has limitations and must be thoughtfully applied. The first limitation is that ANOVA is designed to separate additive, interacting causes, not non-additive interacting causes (Lewontin 1974). Additive causes are ones where the effects are uniform in different conditions. In the above example, adding fertilizer always increases the height of the corn by one foot. Both genotypes respond to the same environmental manipulation in the same way. Concomitantly, the blue corn is always two feet higher than the yellow corn. Adding fertilizer changes the heights but does not change the ratio. Non-additive causes do not have uniform effects in different conditions. If the fertilizer had caused the yellow corn to grow two feet taller and the blue corn to grow one foot taller there is a non-additive interaction. Different genotypes respond to the same environmental manipulations in different ways. DST holds that gene environment interactions are non-additive and are therefore inappropriate for ANOVA analysis.

The second limitation of ANOVA is that it is a context dependent analysis. It does not produce globally applicable results. The analysis proceeds by comparing population averages. If the population average changes, assessment of the individual's variation will change even though the individual did not. An ANOVA analysis comparing the height of blue corn to oak trees will show a mainly genetic difference. An ANOVA analysis of two identical strains of blue corn will show a mainly environmental difference. In each case the causes of the height of the blue corn are the same. ANOVA does not reflect the similarity of causes because it compares contextual differences. That is, ANOVA quantifies genetic and environmental components of differences between populations. It does not explain the causes of the populations' characteristics directly. For this reason Lewontin says that ANOVA is not a functional analysis in that it does not

necessarily explain how genes and environment interact to produce a phenotype. It is a historical analysis in that it is affected by changes in population and environment.

In non-additive situations, the specificity of ANOVA means that it cannot be used to predict outcomes in different environments. The authors of *From Chance to Choice* point out that differences in intelligence are mainly caused by genetic factors. Yet intelligence has been increasing for decades (Buchanon et al. 2000). These odd results do not mean that humans have been evolving quite rapidly in the past few decades. A phenotypic difference caused mainly by genes in one situation may be caused by environment in another. If other factors relevant to intelligence, such as adequate nutrition and instruction, were altered the main factors affecting intelligence might cease to be genetic. Holding the environment constant does not demonstrate the limits to genetic variation or show that all variation under these conditions is genetic. This approach demonstrates the variation created by both that environment and that genotype, not the genotype alone. Nor does it predict genetic variation in alternate environments. Because environmental variation effects genetic causation, a large genetic component to a trait does not mean that the trait is unchangeable. Ultimately, completely separating genetic causes from environmental causes is impossible for ANOVA or any other method.

3. Extended inheritance is the third tenet of developmental systems. Developmental systems theory recognizes that organisms inherit much more than genes from their parents. Jablonka recognizes four types of inheritance systems. These are the genetic and epigenetic inheritance systems as well as systems for inheriting behavior and inheritance systems for symbol systems like languages (Jablonka 2001, 100). Jablonka

asserts that all of these systems carry information which she defines as "*transmissible organization of an actual or potential state of a system*" (Jablonka 2001, 100).

The genetic system comprises the DNA, RNA, and proteins. Parents transmit DNA to their offspring through the gametes. All the DNA the organism will receive from its parents is transmitted during fertilization. After this point the DNA must be replicated by the organism itself. Replication introduces the possibility of mistakes. DNA partly encodes the structure of RNA and proteins. It is also causally involved in their production. Here 'encoded' means that one set of elements reliably produces another set of elements. The DNA codons produce their respective amino acids. Not all of an organism's genetic inheritance is in the form of coding regions or structural genes. DNA also includes regulatory regions that are involved in the turning on or off of the structural genes. DNA also includes so-called junk DNA, which does not appear to have a regulatory or a coding function. The function of junk DNA is as yet undetermined.

Epigenetic inheritance systems tend to control cellular inheritance. In other words, these systems ensure that lung cells reproduce other lung cells and liver cells reproduce other liver cells. There are three varieties of epigenetic inheritance systems. They are steady state, structural, and chromatin marking systems. Steady state systems use feedback to control gene expression. An autoregulatory gene whose gene product acts to promote its own production is a steady state system. As long as the concentration of gene product is high enough the product will continue to be produced. When it falls below a threshold level production stops. The system is triggered originally by an environmental stimulus other than the gene product. The feedback mechanism keeps the system functioning even when the original stimulus is gone. This system can be inherited

when cells divide. Each daughter cell receives not only the genes from the mother cell, but also the appropriate concentration of regulatory proteins that control the genes. Thus the state of gene activation is passed from cell to cell through the protein. This system can have horizontal as well as vertical transmission if the regulatory products are diffusible from cell to cell (Jablonka 2001, 103-105).

The second system is the structural system that directs construction of cell structures such as cilia. Cilia are hair-like structures used by some one-celled organisms for locomotion. They are arranged in parallel, longitudinal rows from the front to the back of one-celled organisms. Cilia are asymmetrical such that the front, back, right, and left of the cilia can be distinguished. When a cell divides to form two daughter cells the orientation of the ciliary row is preserved (Frankel 1989, 19-26). Ciliates with the same genotype can have different cilia patterns. These patterns are stably inherited. Experiments have been done where a ciliary row is rotated 180° by scientists. The rotation is inherited by the cell's descendants. This suggests that ciliary patterns are used as templates to reconstruct the cilia pattern in the daughter cell. DNA does not appear to be involved in specifying ciliary patterns. Jablonka says of structural inheritance "There is no general, autonomous system of transmission independent of the structural properties of the particular complex"(Jablonka 2001, 105). This sort of structural inheritance probably happens in multicelled animals as well as one-celled organisms. Jablonka says it is more likely that cellular structures influence the construction of new cellular structures rather than complex structures self-assembling without a guide (Jablonka 1995, 88).

The third epigenetic inheritance system is chromatin marking. The most well known of these systems is the DNA methylation system. In this system, a methyl group is attached to the bases of the DNA. A methyl group is a carbon atom with three hydrogen atoms. Addition of a methyl group prevents gene expression, so that a methylated gene is inactivated. Methylation is one method of gene regulation. Sexually reproducing organisms receive two copies of each gene; one from the mother and one from the father. However, both genes are not necessarily expressed. In some cases a gene is only expressed if it comes from the mother. In others a gene is only expressed if it comes from the father. This is a phenomenon called genomic imprinting, because each parent appears to imprint his or her mark on the DNA. Genomic imprinting and differential expression appear to be necessary for normal development. Scientists have tried to create mouse embryos with the DNA from two egg cells or two sperm cells instead of an egg and a sperm. Even though they have the full complement of genetic material, these experimental embryos do not develop because they lack the correct imprints. DNA methylation patterns and the gene expression patterns they produce are inherited in cell lines.

Behavioral inheritance systems are the systems by which behaviors are passed from one generation to the next. These can involve learning, but need not. Learning styles can be imitative or non-imitative. Non-imitative behavioral learning transmits a general behavior, but not a specific sequence of actions to achieve a goal. Organisms learn to identify an environmental stimulus and the general behavioral response that is called for by the stimulus. Blue tits and great tits demonstrate non-imitative learning of behavior. These birds have learned to tear open the tops of milk bottles to reach the

cream at the top of the milk. Naive birds learn by observing more experienced birds. However, a naive bird does not learn to mimic a specific bottle opening technique. Rather naive birds learn from the behavior of experienced birds that milk bottles are potential food sources. Each individual bird develops its own bottle opening technique through trial and error. Unlike the genetic and some of the epigenetic systems of inheritance, behavioral inheritance can be transmitted between genetically unrelated individuals (Jablonka 2001, 110).

Symbolic inheritance systems provide for the transmission of learned culture through symbols. Symbolic inheritance systems transfer behavior or habits of thought. Symbolic inheritance differs from behavioral inheritance because it carries encoded information. Unlike genetic inheritance, a symbol refers to the object it symbolizes "by convention, or according to a reference-rule that enables it to refer to other signs in the system" (Jablonka 2001, 111). Symbols do not stand in for reference objects simply because they resemble them. DNA refers to, or stands in for, an amino acid sequence because it causally produces that sequence. The word 'cat' refers to a small furry animal because we have decided it does so. Specific behaviors transferred through symbol systems include language, mathematics, dancing, music, and visual and motor arts. Language, music, and mathematics clearly have formalized rules for the use of symbols. These are grammar, musical notation, and mathematical axioms. Visual arts, such as painting, may have less explicit reference rules, but they are symbols because they are part of a symbol system (Jablonka 2001, 112). The process of transferring symbolic inheritance is the process of teaching and learning the reference rules for a symbolic system.

4. Phenotypes are constructed through the process of development rather than transmitted through the process of inheritance. Construction rather than transmission is the crux of the theory as Oyama describes it. After examining types of inheritance in the last section, we realize that traits are not passed from parents to offspring directly. Statements such as "You have your mother's nose" or "You have your father's eyes" are normally meant metaphorically, not literally. Instead, parents give their offspring the resources for the construction of their traits. Oyama explains that instead of thinking of the DNA or the environment themselves as containing information, we should think of their interaction as containing information (Oyama 2000a, 16). Meaning only comes about through the use of information in the appropriate context. In order for a text to have meaning it must be read by a person who understands the language in which it is written. For DNA to have meaning it must be placed in an appropriate cell. Isolated DNA in a test tube contains no meaning. Environmental meanings are also constructed. The chemical makeup of a salmon's home river contains no meaning unless the salmon is there to recognize it. Developmental meaning only arises through interaction of developmental resources.

DST inhabits a middle ground between preformationism and classical epigenesis. Preformationism holds that a fully formed embryo is contained in the gametes, generally the sperm. Development is then only a process of growth from embryo to adult. It is not a process of structural change. Classical epigenesis holds that order arises from chaos by the action of a vitalistic force. DST recognizes that structure exists prior to development. Cells, and all matter, have structure. However, the structure of a fertilized cell is not the structure of an adult organism. Nor does the fertilized cell contain the structure of the

adult organism in the form of genetic instructions and blueprints. This is merely another form of preformationism. Rather, the structure of the adult comes about in the process of development. Information, as structure, also comes about in development. It does not preexist development. The outcome of the developmental process is contingent on the available resources and their interaction. Thus developmental information is contingent upon the developmental resources. Since developmental information does not preexist development it cannot be transmitted from parents to offspring. "Instead it is ontogenesis, the inherently orderly but contingent coming into being, that expresses what is essential about the emergence of pattern and form" (Oyama 2000, 3).

5. Control of development is distributed rather than centrally controlled by one factor. The fifth tenet follows from the lack of instructions in the genes and the joint determination of phenotype by genes and environment. Genes alone do not control development. Phenotypic variation can come about through genetic or environmental variation. Genes are assumed to contain the form of an organism because genetic variation causes phenotypic variation. This is not merely a case of correlation of two events. We can trace a causal chain from the change in the DNA sequence to a change in the phenotype. The process from abnormal gene to phenotype in PKU is so well known that we can prevent the disease with proper nutrition. Yet PKU is considered to be a genetic disease. Sometimes a change in environment causes a change in phenotype. Cuttings of *Achillea millefolium* with the same genotype grow to different heights depending on the elevation (Griffiths et al. 1993). This too is a causal process. As Oyama points out, we often think inconsistently about the effect of genes and the effects of environment. When genotypic variation causes a phenotypic variation that is

insensitive to environmental conditions, we conclude that development is under genetic control. However when an environmental variation causes a phenotypic change that is insensitive to genotype, we do not conclude that development is under environmental control (Oyama 2000a, 16-17). Developmental and environmental factors are causally symmetric.

An analogy for understanding decentralized control in biology was put forward by Gunther Stent. Stent describes the development of an ecosystem. At first there is only barren land. Gradually the hardier plants creep in. As they grow they create the conditions for other plants to grow. Perhaps lichens gradually break down rock into soil eventually allowing for grasses to live. After enough plants are growing herbivores can migrate to the area. Once herbivores are in place carnivores will be able to move in. Birds immigrate bringing a new influx of seeds. Each organism exerts local control and follows its own life pattern. There is no global blueprint or instruction manual that the construction of an ecosystem follows. Each step in the process changes the environment slightly and permits the next step in the process to proceed. That is, each step is contingent on the preceding step. The contingent pieces combine to produce an orderly process. However the outcome of the process depends on the input and similar inputs will tend to produce similar outcomes. Commonly recurring processes need not be interpreted as necessary or planned. (Griffiths and Gray 1998).

6. Evolution is construction. Natural selection is not an external force that puts pressure on living organisms. Instead natural selection is the name we give to the outcome of the interaction process between organisms and their environment. Therefore

the process of evolution constructs species out of organism-niche interactions rather than selecting already formed organisms.

Darwin conceived of variation being internally generated in an organism. The environment then selected among the varieties for the organism best suited to the environment (Lewontin 2001, 59). DST holds that the organism and the environment both affect each other. Environments are not static and unchanging nor do they present organisms with an unchanging fitness problem to solve. Human beings change their environments in dramatic ways, but all other creatures do this as well. 15 million years ago the Australian climate changed from rainforest to an arid, fire-prone landscape. The change gave eucalypts a survival advantage over rainforest vegetation. Eucalypts have small leaves, thick bark and are drought resistant. They frequently shed both leaves and bark which contain volatile oils, providing ideal conditions for fire. After a fire the seed capsules release their seeds into an ideal growth environment. The fire kills off competing plant varieties and releases nutrients in the soil. Eucalypts modify their environment to be more fire-prone because it gives them a survival advantage (Gray 1992, 197). Thus there is no difference between internal factors producing variation and external factors selecting that variation. 'Internal' factors change the organism, which then changes its environment which then 'externally' changes the organism. Internal/external divisions are unnecessary and obscure the co-construction of organism and environment.

CHAPTER 4

A DST GENE CONCEPT

The genetic determinist view of the history of genetics described above is not the only story that may be told. Throughout the history of genetics, the gene concept has changed with the insights from new experiments. As knowledge and understanding increase, old assumptions must be given up. The new gene concept is not only a response to genecentric thought patterns and assumptions. It was produced, as the other gene concepts, by scientists' efforts that stretched older concepts until they broke. The latest change is toward a less genecentric conception of development, and even a less DNA-centric conception of genes. However, the new gene concept has theoretical as well as experimental ancestors. Even as early geneticists disparaged the cytoplasm, embryologists emphasized the role of the entire cell in development.

The same history that produced deterministic gene concepts also gave rise to the process molecular gene concept. This is a concept put forward by Eva Neumann-Held in an attempt to extend DST to the molecular level (Neumann-Held 2001, 75). The process molecular gene concept holds that the gene is the process that leads from a DNA sequence to a linear polypeptide. A polypeptide is a small piece of protein. This conception counteracts genetic determinism in two ways. First, by focusing on the entire process the definition does not focus on the DNA. It recognizes that the information contained in genes is not only sequence information from DNA. Second, the endpoint of

the gene process is a linear polypeptide. It is not a complex characteristic such as height, hair color, a tendency to depression, or athletic ability. Built into the concept is the idea that genes make proteins. Characteristic traits are made by the further interaction of the proteins and numerous other factors (Neumann-Held 2001, 76).

The transition point between mendelian and molecular gene concepts also contains the seeds of the new gene concept. The chromosome theory of inheritance was the first theory that made the genes physical entities and located them correctly. Yet the chromosomes recombine during meiosis in ways the particulate genes are not supposed to do. Theoretical commitments to particulate genes lead to the belief that recombination only occurs between genes not within genes. For a time genes were defined as units of recombination. Unfortunately for the stability of the gene, recombination happens inside of genes as well as between genes. Portin says "in actuality there is no criterion for distinguishing between intragenic and intergenic recombination unless one can define the gene boundaries by other means" (Portin 1993). However, until evidence for intragenic recombination accumulated in the 1940s and 1950s, recombination as a criterion for the gene prevailed. We now know the gene is not a particulate, atomistic unit.

As understanding of DNA at the molecular level increased, the molecular gene concept became too simplistic. The stability of DNA no longer seemed attributable to its structure alone. Keller explains assumptions about the stability of the gene and its self-replicating ability are incorrect in light of our current knowledge. The copying, or replication, of DNA will not proceed without a number of enzymes and nucleotides. The DNA must be unzipped, or split down the middle, by an enzyme called DNA helicase. The unwound DNA is stabilized by proteins called single stranded binding proteins. Two

enzymes are needed to achieve the creation of a new DNA strand. One enzyme, called primase, specializes in starting the DNA strand. Another enzyme called DNA polymerase specializes in finishing the DNA strand. There must also be a sufficient number of nucleotides with which to build the new strand. (Griffiths et al. 1993).

Even this complex system of replication enzymes and their protein helpers is not enough to ensure an accurate copy of the DNA. After the DNA is copied more enzymes must proofread the DNA for mistakes and repair them. There are proofreading and repair systems that specialize in choosing the correct nucleotide to insert in the growing DNA strand, systems that test to see if the latest nucleotide is correct, and systems that examine strands for mistakes after they are completed, in effect checking the work of the first two systems. Finally there is a system that fixes environmental damage, especially from UV light. The frequency of mistakes with proofreaders is about one mistake in ten billion base pairs, but it is estimated that without proofreaders it would be one in one hundred (Keller 2000, 27). DNA can be accurately replicated with the help of many cellular mechanisms. But clearly it is not the inherently stable molecule that it was assumed to be. What stability it has comes out of the whole process of replication and repair. DNA maintenance is an active process and not a property of the DNA molecule alone.

DNA is taken to be the paradigm example of a replicator. This makes it especially important to understand DNA replication. The structure of the DNA plays a role in creating a copy of itself and in ensuring that copy is of the correct form. The material imposes constraints on what can be built out of it. The DNA is one of the tools that is used to create a copy of itself, but it is not the only tool. A dress pattern for

sewing clothes at home can achieve the same effect. If the pattern is pinned to cloth and the cloth carefully cut to size, the pieces of a dress are produced. But if the pattern is pinned to more pattern paper and the paper is cut to size then another identical pattern is produced. DNA and dress patterns share two qualities. First they both have an ability to create a product: RNA or dress pieces. Second they have the ability to be templates for their own reproduction. In both DNA replication and dressmaking, the first pattern was necessary to make more patterns. But equally necessary was someone to do the cutting of the new pattern. Each part of the system has its role and the roles cannot be reversed.

Even paradigm replicators need mechanisms separate from themselves in order to replicate themselves. With this admission the replicator/interactor line blurs. If only DNA is a replicator then cellular machinery must be part of the interactor. Since DNA replication does not take place without cellular machinery replicators are not separate from interactors. Alternatively, nest site imprinting, song transmission, micro-organism symbionts and centrioles are all replicators (Sterelny et al. 1996). A more accurate description of DNA is to name it self-templating. This makes DNA into a molecule that is passively copied, not one that copies itself. If DNA is to remain an active self-replicator then other entities besides DNA are self-replicating as well.

While the relation between genetics and embryology proceeded in terms of gene action and genetic control of development, it might not have. Even in the early days of genetics there was recognition that genes could not provide the only controls to development. In 1934 T.H. Morgan wrote “The implication in most genetic interpretation is that all genes are acting all the time in the same way. This would leave unexplained why some cells of the embryo develop in one way, some in another, if the

genes are the only agents in the results. An alternative view would be to assume that different batteries of genes come into action as development proceeds" (quoted in Keller 2000, 56). "All the genes acting all the time in the same way" is a facet of gene action. It could not explain the differential activation of genes. "Some of the cells develop one way, some in another" means each of the cells in an organism, except for the gametes, contains an identical set of chromosomes, DNA, and genes. And yet multicellular organisms contain an array of quite different tissues. If the genes are assumed to contain instructions for building a body these instructions must be read differently in each tissue. Some genes must be expressed, or turned on, and other genes must not be expressed or turned off. The question was how this was accomplished. "Different batteries of genes" coming into action necessitated gene regulation. Gene regulation was not given an explanation until Jacob and Monod's work of 1959 to 1961.

The new metaphor for understanding gene regulation was the 'genetic program'. Keller mentions the ambiguity in the term 'genetic regulation' or 'genetic program'. The language leaves it unclear whether the genes are running the program or being acted upon by it (Keller 2000, 87). Oyama points out that when the workings of structural and regulatory genes are described, instead of merely labeled as a genetic program, control appears distributed between genes and environment. She discusses Monod saying "When he engages in straightforward description, the complexity and interdependence of causes are clear. When he interprets these processes in more general terms, however, an interesting thing occurs. He says, for example, that the genome 'entirely defines' protein function" (Oyama 2000a, 31). If we pay attention to the actual workings of gene regulation the genes are not the sole controllers of the 'genetic program'.

A group of structural genes and their regulatory genes is called an operon. The *lac* operon is so named because it makes the enzyme that allows bacterial cells to digest the sugar lactose. The *lac* operon is turned on or off as it is needed by the bacteria. In the case of the *lac* operon this is accomplished by blocking the transcription of DNA into RNA. This transcription is done by an enzyme called RNA polymerase which works its way down the DNA strand transcribing as it goes. In order to prevent transcription a protein called a repressor (because of its ability to repress the lac genes) sits on the DNA strand immediately before the *lac* genes. When the RNA polymerase arrives at the repressor it is physically blocked from moving further along the DNA and transcribing it. In order to turn the *lac* gene on, the repressor is removed from the DNA and the polymerase is allowed to transcribe it. In this example the structural gene is the one that makes the enzyme that digests the lactose. The regulatory gene is the one that makes the repressor protein. So far it looks as though the genes are controlling the expression of the other genes. However there is one more component to the *lac* operon. This is the way the repressor protein is removed to switch the lac genes on. While the repressor can stick to DNA, it can also stick to other molecules, one of which is lactose. When bacteria are fed lactose, the lactose sticks to the repressor molecule, pulling it off the DNA and allowing the polymerase to do its job. Therefore the *lac* operon is, in part, controlled by the presence of lactose in the environment. Sole regulatory control does not rest with the genes themselves.

The journey from DNA to protein is quite complex and involves many opportunities for regulation. First of all, the DNA is stored in the cell in a very compact form. Before it can be transcribed it must be uncoiled to allow the polymerases access to

it. Second, the polymerase requires many helper molecules called transcription factors to do its job. Once an RNA transcript has been produced there is more processing. The front end of the mRNA is capped with a methylated guanine. The back end of the mRNA has a tail of adenines added. Two of the important processes are splicing and editing. When a given segment of DNA is transcribed into RNA the whole sequence is not retained. Various bits are chopped out. These bits are called introns. The bits left in and reattached to each other are called exons. There are various ways this can be done which are called alternative splicing. Sometimes introns are left in the RNA or exons are taken out. Alternative splicing can also produce frameshifts by connecting exons of different lengths. A frameshift changes the starting point for creating a protein thereby creating a different protein. After splicing comes editing. There are two types of editing. In one process nucleotides are deleted or added to the transcript. The added nucleotides were not in the DNA sequence to begin with. In the other process one nucleotide is exchanged for another nucleotide. C is changed to U and A is changed into G. (Neumann-Held 1999, 122). At the end of this process the RNA transcript can have a sequence that does not exist in the DNA from which it came.

Neumann-Held gives more examples of the contextual nature of DNA function. DNA coding regions are often preceded by a promoter region. Promoter regions assist DNA transcription by providing a location for the appropriate enzymes to bind to the DNA. Promoters are often identified by characteristic sequences. Yet, as in the case of splicing sites, sequence is not sufficient for identification. Neumann-Held explains the DNA sequence GTCCATATAAGA may be read as GTCCA-TATAA-GA which is a promoter, or as a string of codons GTC-CAT-ATA-AGA which translate to valin-

histidin-isoleucin-arginin. Which of these alternatives occurs depends on the reader. In her words "the DNA has neither structure, nor, function, nor program, nor information" (Neumann-Held 1999, 119). There are a variety of observations that uphold this conclusion. Promoters are generally identified by their sequence which either contains a lot of TA as in the example above, or a lot of GC. However all TA or GC rich regions in the DNA do not function as promoters. Some of these regions function as promoters at some periods but not others. These are called overlapping genes. This means that promoters must be determined experimentally through function. Function of a sequence, and therefore meaning, is constructed through context, not merely through sequence. Other regulatory regions near the promoter, *cis*-acting elements, are similarly context dependent for their function. *Cis*-acting elements are regions that, like the promoter, help transcription by providing binding sites for proteins. They each effect the function of other *cis*-acting elements in non-additive ways. An older piece of evidence is position effects, where the activity of a gene is changed by changing its location in the genome.

Taken together this evidence suggests that DNA sequence is necessary but not sufficient for DNA meaning. This evidence lead to Neumann-Held's proposal of the process molecular gene concept. The process molecular gene concept is especially appropriate to DST because it applies DST principles on a molecular level. DST claims developmental information comes about only in the interaction of genes and environment. The process molecular gene concept claims that genetic information comes about in the interaction of genes and other cellular machinery. Non-genetic factors do not merely affect the expression of genes. They partly define what a gene is. Context is intimately and inseparably connected to genetic meaning. Neumann-Held says "The

developmental context includes the physiological and metabolic state of the organism as well as geographic conditions, social space, and cultural conditions" (Neumann-Held 1999, 118-119). A gene is a particular reading of a DNA sequence in a particular context. Meaning is in process.

Clearly the concept of what a gene is has changed as we gain greater understanding of actual cellular processes. At the very least we should be careful how we use the word 'gene'. Keller, Portin, and Neumann-Held suggest that this word has become so ambiguous it may be losing its usefulness for us. They suggest the problem be solved by updated gene concepts or more precise terminology. More precise terminology may be of most use to scientists or scientifically literate lay people. When scientists use the term 'gene' they understand which exact molecular item or process they intend through context. They know whether they mean RNA or DNA. They know which pieces of the DNA they refer to, whether it be coding regions or regulatory regions. They know to which stage of the RNA processing they intend to refer. Different words can clarify exactly which part of a process we wish to talk about.

Ultimately, I believe new gene concepts will better combat determinism than care in choosing words can. It is the overall idea of how genes work and what they do that needs adjusting. The evidence that Neumann-Held cites is common knowledge among scientists. The interpretation of the knowledge and what gene concepts it can support varies. Sarkar puts forward an argument similar to Neumann-Held's that information cannot be gleaned from DNA sequence alone. In response Maynard Smith says "Sarkar's argument that the code does not allow one to predict amino acid sequences (because of complications such as introns, variations from the universal code, etc.) is

seriously misleading; biologists do it all the time" (Maynard Smith 2000). Maynard Smith understands the evidence presented by Sarkar, yet uses a different gene concept than what Neumann-Held would suggest. Clearly agreeing upon the evidence and sharing a precise vocabulary is not always sufficient to change beliefs about the way genes work. We must be willing to accept a new interpretation.

A general conception of what genes are and how genes work is important outside of scientific circles as well. The public and the media need a way to think about genes that is not deterministic. Carlson suggests that for non-scientists the "functional sense is more helpful than the gene in its complex biochemical or molecular sense" (Neumann-Held 1999, 126). However, as Neumann-Held points out, the 'functional sense' is the sense in which DNA controls the production of RNA and through RNA, protein. It is the sense in which DNA contains information, or a program for our lives. Most of us probably do not have knowledge about extensive RNA processing or the role of the cytoplasm in development and the 'gene for' locution obscures the influence of non-genetic factors and implies that the genes are in charge.

Many of the traits we worry about having the 'genes for' are diseases. Neumann-Held (1999, 132) suggests that since some of these inherited disease have a late onset they may be under that same sort of developmental control that the genes are. If we can understand the developmental triggers perhaps we could stop inherited diseases without changing the genes that cause them directly. She recognizes that most traits are influenced by genes in some way and says most traits can be altered by altering the genes. However, because genes are 'for' a trait only in a specific context, altering genes may not always be the best option for control of disease. The primary example of this is

phenylketonuria. This disease produces mental retardation. It is caused by a recessive gene and can be prevented through nutrition. Even though it has a genetic cause, the easiest solution for healthy children is an environmental adjustment. Less clear cut cases involve genes for behavior. The so-called gene for aggression is a mutation in monoamino-oxydase A. This mutation is not the only source of aggression nor does it always cause aggression (Neumann-Held 1999, 133). We must be careful what we speak of 'genes for' lest we imply genes have more causal power than they do. This can lead to inappropriate medical treatments or discrimination on the basis of genotype.

What seems most fundamental about all of the gene concepts, even the evolutionary gene concept. is that the gene has a function or produces an effect. That effect may range in different theories from a phenotype, to a protein such as an enzyme, to a polypeptide. In each case genes are judged by what they do. In Dawkins' evolutionary gene concept, as the unit of selection, the gene is judged by whether or not it builds an adaptive phenotype. In the mendelian concept the gene is identified by the phenotypic effects it has. In the molecular concept a gene is identified by its sequence and the protein it codes for. I have tried to present evidence that shows if we wish to retain the gene as the unit of function the concept of the gene must be expanded to include more than just DNA. It must include methods of regulation such as cellular proteins or environmental triggers. It must include the control of the DNA to RNA transcription process. If we do not wish to expand the gene concept we must recognize that DNA alone does not function as a 'gene' does. DNA is one part of a system that manages development and heredity. It is an essential and irreplaceable part, but only one

among many nonetheless. Control and management of the system depends on the proper interaction between the parts, but not on any one part itself.

The triumphs of the molecular gene concept were the discovery of the structure of DNA and the cracking of the DNA code. With these tools scientists appeared to have the ability to read DNA function directly from DNA sequence. Later discoveries proved this to be a false hope. Function cannot be inferred from sequence without knowledge of the context. Variations in RNA splicing and editing, promoters, and coding regions all illustrate this point. DNA sequence was also supposed to contain genetic information that provided for the development of an organism. It seems clear that DNA alone cannot construct an organism. The exact roles that genes and other developmental resources play has not yet been completely elucidated. Yet the insufficiency of DNA sequence described above implies that genes do not carry all the information necessary to development.

CHAPTER 5

CONCEPTS OF INFORMATION

There are two distinct versions of information in use in biology: intentional information and causal information. Proponents of both of these versions refer to the theory of information or communication put forward by Claude Shannon in 1948. This theory originally applied to telephone signals not biology. In particular, Shannon was trying to solve the problem of accurately sending a message through a noisy channel. He wanted to find a way of encoding the message that would allow the fastest and least error-prone transmission (Pierce 1961, 146-147). There are a few controversial aspects to the mathematical theory of information. The first controversial aspect of the theory is whether or not it provides a definition of information or whether it only provides a way to measure information previously defined. The second aspect is whether or not the entropy measured in Shannon's theory is the same as the entropy used in the second law of thermodynamics. I will not address this second controversy further since it does not seem relevant here. The third problem with the theory is that it does not address the meaning of the message transmitted. Colloquially, information is connected with knowledge, meaning, or understanding. Confusion is generated between the technical and colloquial uses of information.

Shannon's theory supposes a source of information and a receiver of that information. The information that passes between the sender and the receiver is the signal. The path the information takes from sender to receiver is the channel. From a

theoretical standpoint, the channel is just a causal relation between the signal sender and the signal receiver such that the receiver depends on the sender (Dretske 1979, 38). Dependence means a change in the sender causes a change in the receiver. This causal relationship that makes up the channel is dependent on all sorts of factors called channel conditions. Channel conditions differ in particular situations, but they are what makes communication between sender and receiver possible. The channel conditions between two people talking on the telephone include the telephones themselves, the telephone wires and poles, and the company that operates the phones. The signal is the message passed between the people. In an experiment, information from the source, or signal, is the variable that one is trying to study. When we stipulate a source and a channel, we also stipulate noise in the signal. Noise is information that interferes with the reception of the signal. It is information at the receiver that does not come from the source. Dretske says "noise is the information available at *r* that is not received from *s*" (Dretske 1961, 19). Noise often comes from the channel rather than the source. It is the static on the telephone line or the variable in the experiment that cannot be completely controlled for.

Information has passed from a source to a receiver when uncertainty at the receiver about the state of the source has been reduced. This prompts claims that Shannon's theory defines information as reduction in uncertainty or negative entropy. The mathematics of the theory provides a method of measuring uncertainty prompting Paul Young to claim the theory can only measure, not define information. Young explains, "Aside from any other consideration, the Shannon-Weiner formula is a measuring device, and so, to equate H with information in its general sense is to confuse

a measuring device with what it measures. A formula that measures the amount of apples in a barrel obviously is not the same as the apples. This has been one of the details most commonly overlooked by those who co-opted this term from the mathematics of communication theory" (Young 1987, 8). The technical term 'information' has been co-opted by biology and perhaps in unfortunate ways.

Entropy can be thought of in three different ways. Most simply, it is the number of bits needed to encode a message. The mathematicians have a formula for figuring this out. Second, from the point of view of the sender of a message, the entropy is the amount of choice she has in forming the message. This may be constrained both by the sender's intentions and the language the sender uses. Third, from the point of view of the message recipient, the entropy is the uncertainty the receiver has as to the message's meaning before the message is decoded or read.

Oyama calls her book on DST *The Ontogeny of Information*, emphasizing that a particular concept of information is central to her argument for DST. There are three facets to the DST view of information. First, Oyama explicitly states that her definition of information is "a difference that makes a difference" (Oyama 2000a, 3). This definition includes genes, but does not single them out as the only source of developmental information. A difference in DNA sequence leads to hemophilia instead of a healthy phenotype in humans. A difference in egg incubation temperature leads to different sexes in crocodiles. Both the DNA sequence and the nest temperature are instances of developmental information. Second, the DST definition of information emphatically denies that there are instructions, blueprints, or recipes for development contained in the DNA. Asserting any of these is preformationistic. It suggests that the

ultimate form of the organism exists before this form comes into being through the process of development. Finally, information develops as the organism develops. Because the information about an organism does not predate the organism, information about an organism must begin with the organism. As an organism grows in complexity from embryo to adult the information it embodies increases.

Simplifying Shannon's theory by leaving out the mathematics shows that information is a relationship between the sender and the receiver. Because of the dependence between source and receiver necessary to transmit information, another way of interpreting or defining information is covariance between source and receiver. This is the causal view of information because the information at the source causes a change at the receiver. It neatly maps onto Oyama's information concept of "a difference that makes a difference". When source and receiver covary, information has passed between them. In turn, covariance suggests the presence of a channel through which the information has passed.

DST focuses on the covariational aspect of information. The DST insight into information theory is that sender and channel can be reversed. In other words, who or what is the sender of information is at least partially observer dependent. In the case of the telephone, it seems fairly obvious that the speakers are the senders and receivers of information and that the telephone and the wires are the information channel. However, the difference between channel and signal is not always so clear, and can be reversed. A favorite example of DST is the television. Usually the TV is the information channel, the TV program the screen displays is the information signal, and the viewer is the information receiver. When the viewer is a repairperson working on a broken set the

relationship changes. The repairperson is the receiver of information, but the TV test card the screen displays is the channel and the TV set is the source of the information signal (Griffiths 2001). As Sterelny and Griffiths explain "The sender/channel distinction is a fact about our interests, not a fact about the physical world" (Sterelny and Griffiths 1999). Biological information is as observer dependent as the information from the TV. A geneticist may wish to discover the effects of a certain mutation on corn plants. She will attempt to grow each plant in the exact same environment, with identical amounts of sunlight, water, and soil nutrients. Then any variation that appears in the plants should be due to their genes. An animal behaviorist may wish to study the effects of environment on the development of maternal care in rats. She will try to use genetically identical rats to isolate the effect of the environment. In the first example the DNA was the source of the signal and the environment was the channel. In the second example it is exactly reversed. The signal source becomes whatever aspect of the experiment is of interest to the scientist.

Because of the interchangeability of sender and channel, DST advocates the causal parity of genes and environment. The idea of parity encompasses some of the six tenets described above such as joint determination, extended inheritance, development as construction, and distributed control. Parity denies the privileging of genes in any way including singling them out as suppliers of form, replicators, or units of selection. Both genes and environment provide information to the developing organism. Genes and environment, or genes and everything else, do not constitute two different categories of inheritance or developmental information. Rather the two intertwine and influence one another. Hubbard gives a few examples of how characteristics we generally think of as

biological are influenced by the cultural. Height, weight, and strength are biological traits. Each of these is affected by nutrition, and at least weight and strength are influenced by physical activity. The quality of nutrition a person receives and the amount of physical activity she or he engages in is affected by their position in culture. A more striking example is the biological trait of menstruation. We consider an average twenty-eight day cycle lasting from age twelve or thirteen to age fifty normal, healthy, or natural. But this is not universal among women. Hubbard mentions the !Kung people who live in the Kalahari desert. Because of their life style, specifically diet and exercise, !Kung women do not begin to menstruate on average until they are eighteen. They ovulate infrequently (by our standards) and reach menopause in their thirties or forties. If they switch to a sedentary western lifestyle, !Kung menstrual patterns change to patterns considered normal by western science. (Hubbard 1990, 125-126). Hubbard says that because the !Kung menstrual patterns change "the difference between their experience and ours is not genetic"(Hubbard 1990, 126). The onset of menstruation is proximally caused by hormones stored in fat. Menstruation starts when a threshold amount of hormones are built up in fat reserves. Body fat is partly connected to diet, a cultural factor. Menstruation, a stereotypically biological trait is inseparable from cultural influence. Although this trait, like all traits, is affected by genes, it is not fixed or unchangeable.

The most formidable assumption that the parity thesis denies is the idea that genes supply the form to the growing organism and the environment supplies the material. When the gene is seen as the conveyor of both form and stability it becomes the conveyor of the essential being, or nature, of an organism. While the idea that organisms, and

people in particular, have a true self that can be discovered under outer layers probably did not originate with genetics, genetics in particular and biology in general became the seat of this self. Oyama sees the roots of form in Cartesian assumptions about inert matter motivated from outside by a mind or the Aristotelian division between efficient, formal, final, and material causes. Mapped onto development, genes take the place of Cartesian mind while the body is mere matter. Similarly in the Aristotelian system, genes are efficient, formal, and final causes of development while the cell and nutrients provide the material cause (Oyama 2000a, 14).

The genes as form/environment as material view has many guises. Naming the genes as instructions, blueprints, or recipes are all versions of this view. DST considers all these formulations to be preformationistic. Traditional preformationism held that there was a fully formed organism inside each sperm. The mother merely provided a safe location and proper nutrition for the growth of that organism. Modern preformationism changes the mechanism slightly, but not the general outlines of the story. The final form of the organism is present before its development in the form of instructions. The process of development is merely one of giving flesh to the form or of following a recipe. No new information is added to what is already specified as the organism. Modern preformationism assumes organisms have a nature encoded in their genes that determines what they are to become. DST points out that neither traditional nor modern preformationism explains how development comes about. Traditional preformationism 'solves' the problem by assuming that development is merely growth. Modern preformationism 'solves' the problem by attributing it to the power of genes.

In contrast to preformationistic views, DST holds that information increases throughout development. Oyama says "information is conceived to be a special kind of cause among all the factors that may be necessary for a phenomenon, the cause that imparts order and form to matter" (Oyama 2000a, 3). With information as structure in mind, two issues become clear. First, information is not an outside force that is impressed upon matter. All matter has form already inherent in it. Second, the structure of an organism changes and becomes more complex throughout its development. Therefore the information that organism embodies must also increase. A DNA molecule has structure, as does the cytoplasm of a cell and the protein molecule they create. The DNA molecule specifies the linear sequence of amino acids in the protein molecule. The linear sequence and cytoplasmic environment together specify the folding of the linear chain into a three dimensional structure. Lewontin notes that when scientists first attempted to produce human insulin in bacteria, the insulin was physiologically inactive. The protein had the correct amino acid sequence, yet the wrong three dimensional shape. When the culture conditions were changed the protein was folded correctly (Lewontin 2000, 116). The structure of a finished protein comes about not just because of the structure contained in the DNA and the cytoplasm. It comes about through the interaction of the two. The interaction creates new structure, new information, that did not exist previous to the process.

By refusing to differentiate between causes of form and raw materials, DST redefines the usual nature/nurture dichotomy. Typically, nature is equated with the form in the genes and nurture with the influence of the environment. In contrast, DST recognizes at least four different systems of inheritance that go into the production of an

organism. Each system plays a unique role. There is no reasonable basis for placing genetic inheritance in the nature category and epigenetic, behavioral, and symbolic inheritance in the nurture category. Focusing on different systems may be useful in answering different questions. Arbitrary divisions are not useful. In DST terms nurture is the developmental process and nature is the outcome of that process. An organism constructs its nature out of the developmental resources that it is provided.

Causal parity is not to imply that all developmental resources are the same or equally important. Claiming that genes and nutrition play the same role in development is the strawman version of DST (Griffiths and Knight 1998). DST seeks to realize the roles of each resource, not to claim sameness between each resource. In the process, they have realized that the role of genes is not unique in the way we previously thought. Genes are important because nothing else fills their role in creating proteins. They cannot be singled out on the basis of being the only form of inheritance between parent and offspring. Nor are they copied more directly than all other resources. Genes do not contain a different type of information from other resources. Genes cannot be separated from other resources because they are inherited and others are acquired. Genes, cytoplasm, cell membranes or cell walls are all necessary and none of them sufficient on their own. Genes are not more necessary than other factors.

There have been attempts to find nonsubjective ways of distinguishing between sender and channel. Dretske describes checking the voltage of a current with a voltmeter. Ordinarily we consider the electrical system to be the source of the information about the voltage, the magnitude of the current to be the signal, and the voltmeter to be the channel. Receipt of the signal depends on the proper functioning of the voltmeter. The voltmeter

can be considered a channel and not a source of information because of the construction and characteristics of voltmeters. It is stable and lacks relevant alternative functional states. For instance, it does not vary its resistance. As long as the voltmeter is in working order, it can transmit only two pieces of information; the voltage and the fact that the voltmeter is in working order. It is not a source of variation so it is not a source of new information. It is acting only as a channel and not as a source. Explaining the difference between the working conditions of the voltmeter and the voltage, Dretske says "the conditions about which one has received information become *fixed*, and thereby qualify as part of the channel, for as long a period as there exists no genuine possibility of their changing into some alternative state. That is, they function as part of the channel for as long as their *persistence* in the fixed condition generates no new information" (Dretske 1979, 118). According to Dretske, sources have alternative states which channels lack. Those alternative states are stable, and they consistently produce a change in the receiver. There is a stable dependence relation between those alternative states in the source and alternative states of the receiver that does not exist between noise and receiver or channel and receiver. However, Dretske acknowledges that there are systems where channels and sources are symmetric. Some systems have more than one source of information. Imagine a system with a battery that wears down as the system runs changing the system's functioning. In this case, the battery is too variable to count as a channel. This is a system with relevant alternative states. The battery, and the system it powers, are sources of information. The signal from this system is a composite signal because it has more than one source.

The issue is whether or not Dretske's differentiation method applies to genes and environments as well as it does to voltmeters and currents. His distinction differentiates channels from sources on the basis of variation and constant background. Experiments are designed to differentiate one variable from all the rest of the causes at work in a system. A geneticist can create a mutation in a specific gene to examine its effects. While this experimental approach allows us to think of genes as signal and environment as channel, we should not confuse handy experimental design with developmental reality. Clearly in the case of crocodile sex determination the environment has relevant alternative states. Once a crocodile's sex is fixed, environmental temperature fluctuations do not alter it. Temperature may cease to be a source of information in that respect. Still, the sex determination was informed by both environment and genes. The environment may provide other information at other developmental stages. Composite signals do not have to be permanent to provide information. Dretske's example of a composite signal may apply to development. Either genes or environment can be the source of the signal or the channel.

Another attempt to differentiate signal and channel come from James MacLaurin. He gives an example of reading a poem off a sheet of paper and attempting to determine whether the poem or the reader's glasses are the signal. The reader's ability to read the poem depends on the correct prescription of the glasses. In this sense reception of the poem covaries with both the piece of paper on which the poem is written and the use of glasses. Since communications theory identifies information with covariation, there seems to be no way to tell whether the paper or the glasses is the source of the message. MacLaurin proposes that the confusion comes about because communications theory

does not examine the content of messages. If we examine the content we can identify the source. He says "If the information that a channel transmits to a receiver is always and only noise, then a variation in the channel cannot alter the content of the message, only the amount of the message that is received" (MacLaurin 1998). Changing the prescription of the glasses will affect how much of the poem is decipherable. It will not change what the poem says. The only way to change what the poem says, or the content of the message, is to write a different poem on the paper. This tells us that the poem is the signal and the channel is the glasses.

There are two problems with MacLaurin's proposal, one of which he states himself. He says "The stipulation of some information source as a signal will always depend on what we consider to be the message" (MacLaurin 1998). The source of information is what we designate it to be. When reading a poem the poem is the message and the glasses are the channel. When reading an eye chart in the doctor's office the letters are the channel and the glasses are the signal. Distinction by stipulation does not suffice to make the DNA (or the environment) the source for developmental information. This must be discovered empirically. We might observe a species of flower that grows to six inches tall when given fertilizer, but only grows to be four inches tall when grown without. We then hypothesize the role of the DNA and environment. If we assume that the DNA is a complete set of instructions that need certain base conditions in order to be fulfilled, then the environment is relegated to channel conditions. Perhaps the DNA says to the flower "Grow to be six inches high". Then if the flower receives all the nutrients it needs it will grow to be six inches high. If it is malnourished it will be smaller. Alternatively, if we assume the DNA carries only part of the requirements for building an

organism then the environment is a source of information. Here perhaps the DNA says to the flower "Grow to be four inches, or five inches or six inches high". The environment and genes co-inform the ultimate height of the flower. Without experimentation we cannot decide which interpretation is more correct. MacLaurin states that the channel/source distinction depends on stipulation. This is an inappropriate answer for science.

The second problem with MacLaurin's proposal is that we must know the content of the message in order to differentiate signal from channel. MacLaurin knows that Shannon's original theory did not concern message content or meaning, so his proposal changes the theory in order to achieve what the original theory did not attempt. The distinction works for his poetry example or more generally when the message is from a human being. We can compare the intended signal with the received signal and determine if new information was provided or not.

As applied to biology, the second problem means that we would already have to know what the content of DNA information is. But, we do not know the message the DNA contains. Knowing the sequence is the not the same as knowing the message in the DNA. Knowing the function of the DNA is a closer approximation to knowing the message. Unfortunately, we cannot separate DNA function from DNA context. Assuming there is such a thing, we can never know the real, true, or intended message in DNA because it is always interpreted in a context. If there is no intentional or context independent message in DNA then MacLaurin's distinction does not work. For the nearsighted reader, putting on her glasses creates new information from the poem. She can read lines that were previously illegible. It is only from the poet's point of view that

the glasses do not generate new information. As nearsighted readers of DNA, we find new information in many of our experiments. There is no poet we can question to see if we have found the original meaning.

Genecentric views of development assume that genes provide the information signal and the environment is the channel. When channel and signal distinctions are shown to be observer dependent, genecentric views must find another method of privileging the genes. These views of development often rely on intentional views of information. Intentional information is information that is 'about' a thing in a symbolic rather than a causal way. Thoughts, speech, or writing contain intentional information because they are about a topic. Intentional objects probably cannot think, but they are shaped by the thoughts of their creator. Intentional objects are intended, meant, or supposed to perform a certain function. Millikan says, "In the broadest possible sense of 'intentionality,' any device with a proper function might be said to display 'intentionality'" (Millikan 1984, 95). Sterelny and Griffiths give the example of a map of Sydney as an example of intentional information. The map has a proper function, or a function it was meant to perform, which is representing the city of Sydney. Because there is one task a map is supposed to do, it may do it more or less well. The map might misrepresent the city by showing streets that do not exist. A misprint on the map does not give alternative information about Sydney. It gives wrong information about Sydney. This makes intentional information different from causal information. Intentional information may be wrong information. Causal information cannot be wrong because there is nothing it is supposed to do.

Intentionality of human artifacts may come from the mind of their designer. In biology, where there is no mindful designer, intentionality comes from natural selection. We assume the proper function is the function that a structure has evolved to perform or that a structure is maintained because it performs that function. Intentionality through natural selection is called teleosemantic information (Griffiths 2001). Maynard Smith says "In biology, the statement that A carries information about B implies that A has the form it does because it carries that information. A DNA molecule has a particular sequence because it specifies a particular protein ... The element of intentionality comes from natural selection" (Maynard Smith 2000).[1] A gene of a particular sequence makes a protein of a particular sequence. The protein proves advantageous to the organism of which it is a part. Because of this, the organism survives, reproduces, and its genes are passed on to the next generation. This view assumes that genes, rather than the proteins, are the units of selection. Thus it was the gene, not the organism or the protein that was selected for. In order to make this possible the gene, and not any other factor, must be the primary cause of the protein.

The intentional view of information is the view behind the 'gene for' manner of speaking. Just as a map is 'for' describing a city, a gene is 'for' a specific trait. Griffiths believes the way we speak of abnormal phenotypes demonstrates this usage. We do not speak of a gene that codes for malformed limbs in the presence of thalidomide. Yet there are genes that produce this effect. If we were speaking in purely causal terms we would speak of the 'genes for malformed limbs' (Griffiths 2001). However, we tend to view this situation as interference with the proper functioning of these genes. Where we

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1 [1]Dr. Preston explains that Maynard Smith's formulation of Millikan is not entirely

recognize malfunction we also recognize a proper function. Intentional information and proper function are a potential source of genetic discrimination. Genes retain their proper functions and their informational content whether they function properly or not. There is the danger of understanding people on the basis of the genes they carry rather than the people they are. Thus a person with genes for diseases, lesser intelligence, or aggression may be judged on the basis of these genes rather than their actual qualities. Intentional information views let their holders believe that people are more truly what their genes dictate instead of the phenotypes they display.

We can imagine a person who displays a normal amount of aggression in ways his or her society condones. When this person's genome is examined, he or she is found to carry a sequence that has been designated 'the gene for aggression'. The intentional and causal views of information give alternative explanations of these facts. On the intentional view, a person might contain 'the gene for aggression' even if he or she is not aggressive. If the gene does not produce aggressive behavior, it is either malfunctioning or has been counteracted by some environmental change. The gene retains its proper function even when it does not perform it in every case. The person who carries this sequence has been designed by natural selection to be aggressive. His or her true self is aggressive despite cultural overlays. This person is a phenocopy of a normal person. On the causal view this particular sequence is not producing aggressive behavior. If a sequence is not producing a given effect, it is not a 'gene for' that effect. Genetic and environmental causes have produced a normal person (as their society defines normal). The causal view does not separate what a person is 'genetically intended' to be from

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accurate, but his point is correct.

what he or she is. There is no true genetic self that is misinterpreted by the environment. The real person is the sum total of his of her phenotypic traits and his or her genotype. On a causal view an organism cannot be a phenocopy.

Shannon's theory of information is connected to intentional biological information through coding. Information theory assumes a message encoded by one human being for another human being to decode and understand. It gives formulas for discovering the most efficient encoding methods. DNA has been described as a code. The power of the word is such that we believe DNA is a code to which Shannon's theory might apply. As proof, Maynard Smith mentions an author who used Shannon's formula to calculate how many bits of information each base contains. Griffiths describes the argument by which we arrive at this conclusion, "(1) there is a genetic code (2) In molecular biology there is talk of signals, switches, master control genes, and so forth (3) Therefore, the information flowing in (2) is information encoded in the sense of (1)" (Griffiths 2001). He says arguments of this type are "clearly meant to suggest that since the 'genetic code' is 'real science' and not mere metaphor, it is only a matter of time before other information talk in biology becomes real science too. At best , this is a very weak inductive argument, at worst it is equivocation on the word 'information'" (Griffiths 2001). The term 'genetic information' gains legitimacy through the interaction between information theory and the production of protein through the genetic code.

The coding terminology supports the proper function view of the genes. First, the code is the system by which the proper function is divined. We believe the proper function of a gene is to produce a specific protein. It was the cracking of the genetic code that allowed us to predict what proteins a particular DNA sequence would produce.

When we know the sequence of a gene we believe we know what that gene does. This is the sort of mentality that lets us believe the Human Genome Project will allow us to understand humanity. Second, codes are representations of specific messages. The message does not change in meaning by being encoded and decoded. This would render the code useless. The coding terminology implies that there is one definite message that the genes are meant to send. When they are used by human beings, codes convey intentional information. Using the same word for a biological process suggests that it too carries intentional information.

Another source of a large part of the confusion over biological information is whether or not that word is best understood in a colloquial or technical way. Technical terms in biology are sometimes terms we use in nonscientific speech as well. This invites conflation of technical and colloquial meanings. Maynard Smith says that "Transcription, translation, code, redundancy, synonymous, messenger, editing, proofreading, library" (Maynard Smith 2000) are all technical terms. These terms also refer to human processes. If the biological processes are sufficiently like the human processes these words also describe the conflation may not be a large problem. Maynard Smith appears to hold this view. He says of his technical terms "the similarities between their meanings when referring to human communication and genetics are surprisingly close" (Maynard Smith 2000). There are enzyme editors instead of human editors that fulfill the proofreading and editing functions. There are collections of DNA sequences instead of books we call libraries. DNA sequences are coded by natural selection instead of human coders. Using an analogy to familiar processes helps our understanding of unfamiliar processes.

Everyone does not agree that the connection between biological processes and human processes is close enough to dispel worries about conflation. Keller claims that any biological reference to Shannon's theory does not use 'information' in the sense Shannon intended. Watson and Crick's reference, although not the first, was dramatic, coming as it did with their discovery of the structure of DNA. It exemplifies what Keller thinks of as the metaphorical use of information in biology. By metaphorical, Keller appears to mean that the meaning of 'information' has shifted from its technical meaning. They said "In a long molecule, many different permutations are possible, and it therefore seems likely that the precise sequence of the bases is the code which carries the genetic information" (1953 quoted in Keller 1995, 17-18). Shannon had published his paper on communications theory just a few years before and it was easy to believe that it could apply to genetic information as well as other types. If DNA was a code Shannon's theory appeared a perfect fit to measure its complexity and thereby its information. However, it was rejected early on by some biologists because it implied deleterious mutations carried just as much information as adaptive mutations. Shannon's theorem can only measure the complexity of a code. It cannot measure how that code functions in an organism. As long as a harmful mutation was produced by a complex DNA sequence, it would be considered to contain a lot of information. Biologists disliked the idea that a harmful mutant contained as much biological information as a beneficial mutation and for this reason rejected the application of information theory. DNA was supposed to be the book of life, not a recipe for nasty mutations. For this reason, Keller believes that these type of statements about biological information became metaphorical rather than literal after this point (Keller 1995, 19).

Keller also states that biologists use information in its colloquial sense and gain legitimacy from referring to the mathematical theory (Keller 1995, 93). Certainly each of the workers mentioned above adapts 'information' to their own use. DST adherents mainly use the covariational aspect of the theory and ignore the coding aspect. This technique allows them to ground their parity thesis. In order to attack the parity thesis, Maynard Smith uses the coding aspect of the theory to highlight the intentional role of genes in development. Genes become a different sort of cause from any other developmental cause because they are intentional. In order to find a principled difference between signal and channel MacLaurin assumes the signal has a content and examines its meaning. Neither content nor meaning had any place in Shannon's original theory. 'Information' has different meanings for different workers. Johnston expresses the problem of ambiguity saying "When a computer scientist or communications engineer speaks of information being encoded in a program, or being input to a program during its execution, the meanings of those terms are unambiguous and they can always be translated into a precise (even if cumbersome) specification of exactly what is happening. When the same terms are used by a developmental theorist (especially when talking about behavior) no such precise specification exists" (Johnston 1987).

In this respect, the ambiguous use of information may function much like the use of 'gene action' described by Keller. 'Gene action' allowed progress to be made in transmission genetics while ignoring development. Yet at the same time it promoted a genecentric view of development by ignoring questions that could not be answered. Information talk may prove useful in the same way that 'gene action' talk did. It may also prove harmful. Developmental systems theorists generally believe that genetic

determinist views of development spring in part from viewing genes and environment as different types of causes of development, or as containing different types of information. The colloquial use of 'genetic information' suggests intentionality and that there is a blueprint for the entire organism present at fertilization. It suggests genes are directing our growth and development. The causal view suggests parity between genes and other resources. This means understanding information in biology, technical or non-technical, causal or intentional, will influence how we construct our identities, what we believe ourselves capable of and the types of society we consider best for ourselves.

CHAPTER 6

DYNAMICAL SYSTEMS AND COMPUTERS

We have a habit of understanding genes and genetics in terms of our latest technology. The newest of these is understanding genes in terms of computer programs. The term 'program' applied to gene function was introduced by Jacob and Monod in their paper on gene regulation (Keller 2000, 80). The existence of regulator genes and structural genes began to explain different cell fates in development. It explained how gene function could be more complex than the 'gene action' understanding. A 'program' implied that structural genes acted in a coordinated plan contained in the regulatory genes. The plan itself was written in the genetic code by natural selection. The genetic program is the source of control in development and specifies the form of the adult organism. I do not wish to suggest that genes are functioning as a particular type of computer. As soon as a new technology comes along, genes will be compared to it and the new analogy will be considered more fitting than the last.

Rather, I wish to point to a particular theory of cognition called dynamical systems, and use it to emphasize a point about complex organized systems. That is, once a certain level of complexity is reached, decentralized control works much better than centralized control. Organized, coherent behavior is possible without a mastermind or master molecule. We may wish to hold on to the idea that the genes are in control of development because there must be a leader in control to produce such organization. Dynamical systems shows this is a false assumption.

Cognitive science and artificial intelligence have attempted to understand how cognition works in biological brains and model that cognition into computers. There are two models for how cognition works. The first is the computational or classical AI model. This model assumes that the mind is a sort of computer that directs the body. In cognition, the mind-computer goes through a series of sequential steps. First, sense organs deliver inputs to the brain. These inputs are changed into symbolic representations that the mind manipulates to produce a course of action or a decision. Inside the mind is a representation of the outer world. To design a course of action the mind mentally moves through the map before coming to a decision. When the decision is complete, the mind outputs more symbolic representations of the course of action or decision. Finally, these symbols are translated into motor commands to the muscles which the body then carries out. Because the mind uses symbols for representing the outside world and the body, this model assumes that the mind can be studied without studying the body or the outside world. Structurally, the computational model assumes that there is one control center that gives orders to the rest of the system. "Internally, the cognitive system has a modular, hierarchical construction; at the highest level there are modules corresponding to vision, language, planning, etc., and each of these modules breaks down into simpler modules for more elementary tasks" (van Gelder and Port 1995, 12)

This model of cognition is reminiscent of our model of genes and gene action. The genes are the 'mind' of the organism directing its development. In them, and only in them, resides the information that builds the organism. They use a symbol system, the genetic code, which must be translated into protein before it can direct the development

of the organism. The action of the genes can be studied in isolation from the rest of the cell or organism. Keller quotes Bonner saying the "master programme constituted in turn a set of subprogrammes or subroutines. Each subroutine specifies a specific task to be preformed. For a plant, his list includes cell life, embryonic development, stem development, root development, reproductive development, and others, Within each of these subroutines is a list of cellular instructions or commands" (quoted in Keller 2000, 85-86). In general, the gene action model supposes the central control of the processes used to create the organism and its behavior lies in the genes.

However the computational model of cognition has had at least two problems which have led to the development of a new model of situated or embodied cognition. Computational models are neither fast nor flexible. Computers or robots programmed in a computational model are unable to respond to a changing environment with ease. Nor can they function quickly enough to respond to real time situations. A housefly programmed in such a way would be swatted with a flyswatter before it could decide whether to fly away and where to fly to. Nor might it recognize the danger in books, newspapers, and shoes in time to dodge. Clark says "The classical AI planning system can sit back and take its time, eventually yielding a symbolically couched description of a plausible course of action. The embodied planning agent must take action fast-before the action of another agent claims its life" (Clark 1999, 7). The computational model assumes that providing a system with enough explicit knowledge will make it intelligent. This has not yet proved to be the case. Dynamical systems assumes that making a system able to respond to its environment in real time will make it intelligent.

In order to make a system fast and flexible it must have a few specific differences from a computational system. The dynamical model assumes distributed control rather than central control. A striking example of organization without central control at work in computers is given by Pattie Maes. She created a scheduling system that assigns tasks to different computers in a network. The computational method of solving the problem would create one system that monitored the work of all the other computers in the network. It would have explicit information on all the types of computers in the network and the tasks they must perform. It would have to calculate the most efficient schedule from among all the possible schedules. The problem with the computational method is that it is not terribly robust. If anything goes wrong with the scheduling system the whole network will become disorganized. The dynamical systems approach is to let each machine control its own tasks. When a machine has more work than it can handle it bids out the work to other computers in the network. The overworked computer gives the excess work to whichever other computer can accomplish it fastest. The result is an organized, efficient, robust system with no centralized control. It has the advantage over the traditional computational system that no one computer is essential to the running of the whole system. (Clark 1999, 43)

The interaction of factors in producing action leads to the next aspect of dynamical systems. One of the factors that must be integrated into the system is the environment. We saw in the previous example how work might be efficiently distributed without a special program designed for scheduling. When behaviors become more complex than scheduling, decentralized control may seem less plausible. However, instead of turning to central command the decentralized agent can turn to the

environment for help in organizing behavior. Using the environment as a part of the system is both a requirement for a successful decentralized system and a strength of this method. There are two aspects to the use of the environment. First, dynamical systems do not generally use internal representations of the environment. That is, they do not carry a detailed map of their space in their memory. When walking across the living room of one's house at night when it is dark, people do not generally memorize how many steps they can take before running into the furniture or exactly where to turn to go into the kitchen. Instead we reach out for the couch or trail a hand along the wall. Memorizing the exact position of every detail would be a waste of brain space. We let our hands tell us where the coffee table is instead of our memory. This is an example of using the environment as a guide instead of an internal representation or mental map. Robot programmers save memory space in much the same way. Giving a robot a precise representation makes it less flexible. If the environment changes when the representation does not the robot may have difficulty adjusting. For instance a robot may be programmed to pick up bolts from an assembly line that is two feet in front of it. If the bolts are misplaced on the assembly line and are only a foot and a half away, the robot will not be able to pick them up. If the robot has an internal map of where the assembly line and bolts are meant to be, it cannot adjust when they are out of place.

Second, dynamical systems uses the environment as one of the controls on the behavior. The robot could have a sensor that is tripped every time a bolt passes it on the assembly line and only when a bolt passes. This would trigger the robot to reach out for the bolt. This is a more flexible approach than programming the robot to reach out for a bolt every two minutes. If the assembly line sped up or slowed down the robot would

miss bolts or would reach out too often. Allowing the robot to sense the bolts allows the environment to play a role in generating the appropriate behavior. Clark says "In situations where a more classical, inner-model-driven solution would break down as a result of the model's incapacity to reflect some novel environmental change, 'equal partners' solutions are often able to cope because the environment itself helps orchestrate the behavior" (Clark 1999, 43).

The MIT robot lab designed and built a robot, Herbert, to pick up soda cans around the lab that used a dynamical systems approach. Herbert would need to move about in a changing environment and identify soda cans out of all the other objects in the laboratory. A traditional method for solving the problem would be to build a robot that could identify soda cans and recognize its environment through visual data. The visual data provide the robot a mental model or representation of the room and the soda cans. Using the data it would compute a path to the soda can. Clark points out that this method fails. Not only does the environment in the laboratory change too quickly because of its human occupants, but we cannot yet give any robot visual recognition as accurate as our own. Instead Herbert's behavior was designed to be controlled by the environment. The robot had programs for locomotion and obstacle avoidance. It recognized soda cans through a combination of simple visual recognition and touch. Recognition of the proper shape triggered grasping and collection (Clark 1999, 14). There are three important differences between Herbert and a more traditionally designed robot. First, unlike the traditional robot, Herbert does not have detailed visual sensors. Second, it does not compute a trajectory through a model of the laboratory. Instead it rolls until an obstacle blocks its path, then activates its obstacle avoidance programming. In this respect it is

like a wind-up toy that walks until it hits a wall. Herbert is different because it has sensors to detect the wall, or furniture, before it hits it. But it does not plan a route and then execute it. It acts and responds to the environmental constraints to its action. Third, it has no centralized control. Each programmed routine, like locomotion, obstacle avoidance, can detection, and can collection, is triggered by the appropriate item in the environment. When the environment triggers one response, that response could inhibit inappropriate responses. Coherent behavior comes about through the connections between the subroutines and the environment, not control by one system. Like the scheduling system described above, all the subroutines work together to produce the correct behavior.

Biological brains must be able to function in real time or they will not survive for much of it. It is important to synchronize behavior with environmental stimuli. Artificial systems, and perhaps biological ones too, do this by allowing the environment to produce the behavior when necessary. The industrial robot described above only reached for the assembly line when it sensed a bolt. Moreover, action in real time means that many different parts must interact together all at once. In real time, decisions and actions are often simultaneous and not the sequentially ordered process that computational systems are built to handle. In describing a tennis game, van Gelder and Port say "As you move into place, your perspective on the approaching ball is changing, and hence so is activity on your retina and in your visual system. It is your evolving sense of how to play the point that is affecting your movement. The path of the approaching ball affects which strategy would be best and hence how you move. *Everything is simultaneously affecting everything else*" (van Gelder and Port 1995, 23). Playing tennis successfully requires

that the mind perform many different and interacting cognitive tasks at the same time. Performing one task may also affect another task.

Finally, dynamical systems do not always use a language-like symbol system to explicitly describe and program behavior. Instead the problem is solved by embodiment. Embodiment means that a system is realized physically instead of being represented symbolically in a computer model. Clark uses van Gelder's example of a Watt governor that regulates the speed of steam engines for industries that need a steady source of power. Watt governors have two arms with metal balls at the end that turn on a central rod. This rod attaches to the flywheel on the steam engine and turn as the flywheel turns. As the flywheel turns faster and faster the arms rise up with the centripetal force of the turning flywheel. This is like the spinning swings at amusement parks. As the arms rise they close the throttle on the steam engine to slow it down. As the engine slows and the arms fall, they open the throttle so the engine speeds up again. In this way the governor keeps the steam engine at a constant speed. A computational governor would need commands like "Measure the speed of the flywheel" or "calculate the desired alteration in steam pressure" (Clark 1999, 99). The Watt generator however does not need coded instructions or a measuring capacity. It does this through its actions. In the dynamical systems sense, cognition is not happening in a separate mind. It happens through the use of the body. In fact modern governors designed for greater precision sometimes work less well. More precise governors have difficulty finding a constant speed. They are finicky to calibrate and tend to overcorrect the steam engine. The older models had more friction between the moving parts and this tended to dampen the overcorrection. Dynamical systems understands the body (of the organism or the robot) as playing a role

in generating the correct behavior. The physical construction of the organism matters. It can take the place of explicitly coded directions to a computational system.

The final relevant aspect of the dynamical systems perspective on cognition is its understanding of plans-as-programs. Herbert, the robot described above, navigated through a laboratory and accomplished its assigned task without having an explicit plan to accomplish this task. Biological brains appear to function in similar ways. The plan-as-program model takes the plan as a set of instructions that, if followed, will lead to the desired goal. Humans do not generally make this sort of plan. Instead, our plans interact with the environment. Plans may need adjustment if the instructions output different results than expected. Still, this method does not encompass the extent of the interaction. Even if the plans need a bit of readjustment, there is still a set of internalized instructions to lead to solution. Solving jigsaw puzzles exemplifies this difference. People do not examine the pieces, solve the puzzle in their heads, and then put the pieces into place. Instead people pick up the pieces, manipulate them, and then try out different combinations. Physically manipulating the puzzle pieces generates more information which generates different strategies for solution. Interaction with the puzzle pieces changes the mental processes we need to use to solve the puzzle (Clark 1999, 63-64). This is not a case of adjusting an already complete plan to correct unexpected results. The plan is generated as the puzzle is solved and does not exist in its entirety until the puzzle is finished. Our problem solving thinking does not happen solely in the mind. It happens in our actions and interactions with our environment. Traditional programmers did not always recognize this in attempting to create artificial intelligences. They tried to place intelligence and problem solving in the program and neglected the robot and its

physical surroundings. Clark says "classical rule-and-symbol-based AI may have made a fundamental error, mistaking the cognitive profile of the agent plus the environment for the cognitive profile of the naked brain" (Clark 1999, 61).

Perhaps the gene action model has made a similar mistake confusing the genes with the genes plus the body of organism plus the environment. It is easy to see blueprints and programs where none exist. The tendency to assume plans where they do not exist is exacerbated by the often confusing nature of dynamical systems. They are difficult to design because they come to solutions in unexpected ways. The solutions that human engineers and designers think of do not necessarily mimic the way the human brain functions. Similarly, the patterns we perceive in development do not imply the presence of a plan or blueprint for development.

A dynamical systems approach applies to far more than artificial intelligence and computer programming. Dynamical systems can be used to explain development in biological organisms. In this context, dynamical systems shares many of the same beliefs as developmental systems theory. Both of these frameworks conceive of the problem of explaining development in the same way. The regularities of the developmental process are not explained by pointing to a set of instructions or rules in the genes. Invoking genetic instructions only places the problem of preformationism in a different location without solving it. Nor is the problem solved by placing the source of information in the environment instead of the genes. Genetic and environmental determinism share the erroneous assumption that developmental outcomes are determined before the developmental processes which create them. Successful explanations of development describe the processes by which new forms are generated out of existing forms and how

individual novelty coexists with species regularity (Thelen and Smith 1994). Dynamical systems is an appropriate framework for explaining development because dynamics is about describing processes that change over time.

A developmental blueprint view focuses on the species regularities rather than individual variation. An individual organism undergoes enormous changes in the passage from single cell to adult. Yet a species as a whole shows great stability in the developmental process. The process takes a simple organism and changes it into a more complex organism. It involves many discrete stages that pass into one another in an unvarying order. Once through a stage, an organism never returns to a period of less maturity. The developmental stages an organism passes through are a result of the blueprint contained in a central controller. This central controller directs the organism to mature in accordance with the blueprint it contains. Thelen and Smith explain that it is our recognition of the orderliness of the process that suggests a blueprint or plan. In describing the progression of the stages we believe we have explained them. "The very act of describing and classifying a continuous stream of changes into universal types made it easy to invoke a universal classifier, an agent (the brain) who knows the stagelike outcomes ahead of time and guides the organism to those outcomes" (Thelen and Smith 1994). Developmental blueprint views hold that development has a single cause and that the outcome of the developmental process is represented somewhere in the system before the process occurs.

Thelen and Smith show that the development of walking in human infants is best understood from a dynamical system framework rather than as the manifestation of a developmental blueprint. They focus on two stages in the development of walking. First,

infants produce steplike motions when they are held upright until two months of age when they cease stepping. Second, infants resume stepping at eight to ten months when supported on their feet. Surrounding the first stage they observe that human infants will kick their legs after two months of age when supine. This kicking movement is nearly identical to the upright stepping movements that are lost at two months of age. Infants will also kick throughout this period when sitting or prone. Three month old infants who have ceased stepping will resume stepping when placed in water and stepping infants will cease when weights are placed on their legs (Thelen and Smith 1994).

Thelen and Smith also examined the resumption of stepping at eight to ten months. Infants who have not yet resumed stepping on their own will step if placed on a treadmill. The infants were able to adjust their stepping if the speed of the treadmill was altered. A treadmill with a separate belt for each leg was used to test the infants' coordination at differing speeds for each leg. Infants were even able to adjust the speed of each leg independently. While walking on the treadmill infants stepped in a more mature fashion than their age normally suggested. They alternate their legs more consistently and step heel first rather then landing flatfoot or toe first (Thelen and Smith 1994).

Thelen and Smith use their results to demonstrate the dynamical systems explanation of development. Control of development is not hierarchically organized with a central controller issuing commands to organismic subunits. Instead development is the process of many parts learning to work together as a coherent whole. It is a process of opportunistically seeking out solutions rather than a directed process. Thelen and Smith's experiments suggest two of the components involved in stepping behavior.

Changes in muscular strength in combination with leg mass constrain when stepping occurs. Infants normally gain weight quickly after birth. This weight is in the form of subcutaneous fat instead of muscle. More dramatic weight gains are correlated with quicker loss of stepping. Non-stepping infants resume stepping when their leg mass is reduced in comparison to their strength by placing them in water. Stepping infants cease stepping when their leg mass is increased in comparison to their strength by placing weights on their legs. The continuation of kicking movements when supine or sitting implies that other components of the stepping system remain in place despite changes in strength and weight. These experiments focus on only two of the many factors that must be involved in stepping. However, the conclusion to be drawn is that development is a balancing act between a variety of components. Developmental change is a product of the shifting balance, not the manifestation of a blueprint. The age at which stepping ceases or resumes is a contingent rather than inevitable fact.

One of the components of the dynamical system is the environment or the context in which the behavior occurs. In the previous paragraph we saw how different environments elicited different responses. Treadmill stepping demonstrates how the environment can orchestrate a particular behavior. At seven months of age infants can produce a walking pattern on the treadmill when they are incapable of producing it on their own. The adaptability of the step pattern to treadmill speed suggests that pattern generation does not happen within the infant alone. The stretching of the legs backward by the treadmill imparts the necessary energy so that the leg swings forward again like a pendulum. In more mature walkers, the backward stretching of the leg triggers the central nervous system to direct the appropriate muscle movements to swing the leg

forward again. Step patterns are produced through the interaction of the body obeying physical laws, communication between legs and nervous system, and the treadmill. Infants clearly have the necessary muscular-nervous connections in place so that the legs can respond to environmental manipulation. However, it is the environment that provides the organizational inputs at this stage. It is not the reading off of instructions that control behavior, but physical and environmental constraints.

The construction of development from multiple components without centralized control fits the experimental data. It also explains the adaptability of behavior to different environmental conditions to promote survival. "An adult person asked to locomote at a comfortable speed on a flat, carpeted surface will produce a highly stable and invariant pattern of movement... However, if the surface is an icy sidewalk, or the person puts on high heeled shoes, or develops a blister on his or her foot, the characteristic pattern of locomotion may well change. This ability to readjust the pattern in response to intentional, organic, or environmental constraints arises from the previously mentioned ability of subsystems to dynamically assemble" (Thelen and Smith 1994). A program for development would need multiple if then statements to respond to differing conditions. It might be unable to respond to a situation for which it had no programmed blueprint. A development process constructed out of many components can change and adapt whenever one of the components changes. The ability of the system as a whole to change is built into it. Thus an unexpected change in environment need not damage the system. It merely helps the system assemble itself into a new form. An individual that can easily tailor its responses to the external world is more likely to

survive than an individual that cannot respond appropriately to its environment. Dynamic construction is a survival advantage.

Development can be described as a dynamical system. This means the organization of development can be understood by drawing lessons from another dynamical system, artificial intelligence. First, organized behavior is possible, and sometimes even works better, without centralized control. Control of development is distributed throughout the genes, the organism, and the environment as control of a robot is distributed through encoded program, physical body and environment. Second, the system does not always require explicit representations of its environment. Using the environment directly instead of representing it internally can be more efficient. Third, the system often does not encode the specific behavior itself, but subprograms that lead to this behavior. Fourth, is the combination of these lessons. Behavior is often organized by the environment. It takes the place of a central planner. Using the environment means fewer programs that must be encoded specifically. It guides action. Fifth, the physical instantiation of the robot also constrains its action. Again, this allows for less programming space to be used on explicit instructions. The final two lessons are most important for applying to our understanding of genes and organisms. These solutions are not designed in the ways we find most intuitive. We tend to impose a traditional componential organization on them that is not there because we find that organization easier to understand. These systems involve not just the workings inside the brain or the programming code as mind and cognition. Mind and cognition involve brain, body, and environment. When we examine development we need to be aware of its design

techniques, not our own. We need to realize that organized development and behavior can occur without a master control molecule.

CHAPTER 7

CONCLUSION

Genetic determinism arises from the particular historical process of understanding the role of genes in development. Assumptions of stable, particulate factors that created traits and were passed unchanged through the generations formed the basic identity of the gene. Genes as creators of traits persisted through mendelian to molecular gene concepts, although the mechanism became more elaborate. The evolutionary gene concept particularly uses the stability of genes to single them out as units of selection.

In genecentric development, the creative power of the genes became the answer to the preformationist/epigentic dilemma. Genes as instructions explained how order was naturally impressed upon matter. It appeared to avoid the non-answer of preexisting order in preformationism. Intentional information in genes provided the correct outcome of the developmental process and a source of the true nature of the organism. They set the life plan for the growing organism.

Developmental systems theory challenges the assumptions of the genecentric view of development. Intentional information in genes does not solve the preformationist/epigenetic dilemma. It merely places preformationism in a different location. A true resolution to this contradiction is the development of order, or information, itself in the process of development. The development of an organism is like the production of a snowflake. Snowflakes are both symmetrical and unique. They are symmetrical not because of some overall plan, but because their growth is determined

by the microclimates they pass through as they fall. The microclimates act upon the properties of water crystals and the previous structure of the snowflake. Thus the development of each snowflake is a partially orderly process with chance components (Begley cited in Oyama 2000a, 234-235). The development of living creatures involves order and chance in similar ways.

The DST understanding of developmental information as process generated and primarily causal provides a framework for reinterpreting the nature/nurture dichotomy. The division between nature and nurture comes about because of an assumption of intentional information in the genes. In this context, the nature of an organism is equated with its genetic plan or program. Nurture is the changes made in the program due to the materials available for its production. Nature is what is most fundamental and unchanging about the organism, while nurture provides superficial and flexible alterations. DST heals the nature/nurture division by a new understanding of the role of genes. As genes have no meaning outside of their functional context, they do not produce an essential nature. Non-genetic influences are not peripheral or superficial to development because they give meaning to the genes by providing a functional context. All developmental resources, genetic and non-genetic are necessary to successful development. They cannot be divided into resources for form and resources for matter. Nurture is the process of development and nature is its outcome.

DST fits into a larger theory of dynamics. Dynamics is a mathematical theory devoted to explaining change in any type of system that evolves over time. Developmental systems and dynamical systems share similar explanations for making sense of changing processes. Primary among these is that complex systems do not need

centralized control. They are able to self assemble out of their component parts. Self-assembly and decentralized control can be difficult to understand. We tend to assume that orderly processes need programs and central controllers. Second, both DST and dynamical systems hold that causes are normally multiple. All of the component parts of a system contribute to causing its characteristics. While different components may play different roles, none of the components is primarily responsible for the system as a whole. Using computer programming and artificial intelligence as an example of the dynamical system approach helps us to correct our bias towards single causes and centralized control. We build robots that appear to us as though they have central control. Yet we know that the robots are examples of orderly behavior through decentralized control. Applying this lesson to biological development means that we need not assume that it is under the control of the genes or is primarily caused by the genes. A developmental or dynamical systems approach means searching for multiple causes and the connections between them.

DST is an effective framework for answering genetic determinism. It denies to genes the privileged role that a genetic determinist or genecentrist view of development holds. Genes are simply one more developmental resource that parents provide to their offspring. They are necessary but not sufficient for development to take place. DST will allow us to take a more holistic view of organisms, especially humans, and their development. In a DST view judging a person on the basis of their genes, rather than the person as a whole in their specific environment, does not make sense. DST cannot get rid of discrimination or prejudice entirely, but it claims that using genetics as a basis for prejudice is incorrect.

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