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Dissolving Nature/Nurture: Development as Coupled Interaction

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Abstract

For many, the Nature/Nurture approach to development is a quaint figment of the past. We have moved on, one might think; everyone thinks that both categories are important for development, not merely one. The reality, however, is not so simple. In this dissertation, I argue that contemporary biology has not succeeded in getting out from under the shadow of Nature/Nurture, despite everyone being some sort of interactionist about development. The central aim of my project is to offer a form of developmental interactionism worth having, which succeeds in shedding the pernicious aspects of Nature/Nurture. I begin by giving an overview of several candidate notions of interaction. One particularly promising notion is coupled interaction, where multiple variables co-determine each others' products, and cannot be well understood in isolation. I follow this up by examining the Central Dogma of genetics, and argue that the evidence supports the Extended Genome Thesis – the notion that the genome partially extends beyond the body in a sense that is analogous to coupled interaction. I then extend the analysis to the inherited/acquired distinction and argue that the distinction is meaningless. Traits are not inherited wholesale; they must be constructed anew in ontogeny. This means there is not a principled difference between whether the trait is inherited or acquired. My project culminates with an argument that the Nature/Nurture distinction itself is incoherent, based on the available evidence. The world simply isn't separable into distinct categories in the way Nature/Nurture implies; the reality tramples these conceptual boundaries. Neither category can do the work traditionally ascribed to it without its counterpart, and so those results should be understood as emerging from a coupled system. This system transcends the traditional internal/external divide, and so cannot be decomposed. Thus, Nature/Nurture is incoherent because there is no real difference between the components of one category and the components of another. Instead, the parts are coupled to form the ontogenetic (developmental) niche. To round out the project, I canvas some areas of science that I think are worth keeping a close eye on, given their capacity to enhance or harm the way understand development.

Keywords

Philosophy of biology, Nature/Nurture, interaction, genetics, development, ontogenetic niche, coupled interaction, Extended Evolutionary Synthesis

Summary for Lay Audience

As organisms develop, one can wonder if their development is guided by some pre-formed plan (i.e. through their genes, evolutionary history, etc), if development is more contingent on circumstance, or perhaps a mixture of both. This is the crux of the Nature/Nurture distinction. Contemporary biology often considers itself to have moved beyond the overly-simplistic Nature/Nurture distinction, but tacitly includes problematic aspects of it. My thesis aims to diagnose this problem and propose a solution based on characterizing development in terms of interaction in a way that gets around the shadows of Nature/Nurture. To formulate the solution, I begin by looking at several different kinds of interaction across various parts of science, examining their strengths and weaknesses. There are a few kinds of interaction that can do useful work for my position, but I focus on coupled interaction, which is where the values of multiple variables are inextricably bound together (and cannot be solved separately). I then pivot to examine genetics, and the notion of inherited/acquired traits in the subsequent chapters. When looking at genetics, I defend the Extended Genome Thesis – the notion that the genome transcends the internal/external divide of the body. I argue that the genome and environment are mutually reactive to each other, meaning genes and environment can be understood as an example of coupled interaction, where the meaning of each depends on its counterpart, and cannot be understood in isolation. Similarly, I argue that there is no real difference between a trait being inherited or acquired; in both cases the trait must be constructed for the organism during development, and this will always rely on both the genome and the environment. My project's central argument culminates in Chapter Five, where I argue that the Nature/Nurture framework is not merely false -given the available evidence – it is incoherent. Things traditionally-described as 'Nature' cannot be kept developmentally separate from the things described as 'Nurture', and both sets rely on each other to do the work traditionally ascribed to them. Thus, there is no real difference between the categories, and the Nature/Nurture distinction collapses.

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Table of Contents

Chapter 1 – Introduction, Background & Motivations

The Nature/Nurture distinction has been pronounced dead several times, by various different scholars (Oyama 2000a. 2000b; Keller 2010). Despite this obituary, several authors have remarked that the distinction's shadow seems to still linger over contemporary thinking in negative ways. One of the interesting aspects of this persistence is that effectively everyone agrees that Nature/Nurture is a poor framework for thinking about development. If everyone agrees it is a poor framework, then it is an interesting question how it has persisted despite concerted efforts to be rid of it. As I will argue (at length) throughout this project, I think one of the reasons for its persistence is that some of the concepts of contemporary Neo-Darwinism have smuggled aspects of the Nature/Nurture distinction into modern discussions. Metaphorically speaking, with one hand we try to kill the distinction, while with the other we usher it to safety as a prized possession. My hope - however forlorn - is that a more thorough analysis of development, with a focused attempt to dissolve the Nature/Nurture distinction, may allow us to see a way forward without the trappings of this pernicious shadow.

This is obviously a substantial undertaking, and not a matter of a couple paragraphs of clever argument. The larger problem(s) also need to be made tractable, by breaking them down and examining each component in turn. To this end, this introduction to my larger project is comprised of three main sections: background, motivations, and summary. (It is also worth noting that each section in this dissertation is numbered by chapter, then section, then any subsection. For example, the three sections listed above will be numbered 1.1, 1.2, and 1.3 respectively, since they are sections 1, 2, and 3 of Chapter 1. Further numbering denotes additional subsections. For example, heading ‘2.1.2’ indicates that the reader is in Chapter 2, Section 1, subsection 2, etc. Sections of subsequent chapters will be numbered in the same way – ie 3.1, 4.3, etc. I will include some examples below in the structure summary to help make matters clear).

As noted above, the structure of this chapter proceeds in three sections. In the first section (i.e. 1.1), I will offer some background information on the relevant subjects that the project seeks to engage with. This includes some history on the Nature/Nurture framework (1.1.1), preformationism (1.1.2) and interactionism (1.1.3), and finishes with a quick primer on genetics and the Central Dogma of molecular biology (1.1.4). Beginning this way will, ideally, make the jump to more technical discussions less difficult for readers that don't have a background in genetics. Section Two (i.e. 1.2) offers some motivations to believe that the Nature/Nurture framework is socially and epistemically pernicious (1.2.1 & 1.2.2). At this point, I also offer a quick sketch of the overall project and how I see matters coming together. Finally, Section Three concludes the chapter with summaries of each chapter and how they fit into the larger project.

1.1 - Background

With the main vision and motivations for the project out of the way, I wanted to turn to some background information to ensure the reader is well-equipped to engage. To maintain flow, I've attempted a specific order of the terminology that this section covers. I start by briefly describing the Nature/Nurture framework, which then flows into a discussion of preformationism since the Naturalist tends to have preformationist leanings informing their position. The dominant position in developmental science, however, is a kind of interactionism, which seeks to reconcile preformationism with its counterparts. Thus, the primer will flow from preformationism to interactionism, and describe the contemporary position. I will round out this section with a short piece on the so-called central dogma of genetics. This piece comes at the end, because I aim to show that it draws on preformationist ideas, and in doing so upholds the Nature/Nurture distinction.

1.1.1 - Nature/Nurture

I take it that most are likely familiar with some form of the Nature/Nurture distinction. However, given so much of my larger project centres around this framework and its implications, it is worth giving a brief overview of the history associated with this framework.

To begin, there are several different 'incarnations' of the Nature/Nurture debate. The themes exhibited by the distinction are not new, and as such have shown up several times throughout the history of philosophical (and scientific) thought. For instance, one such precursor discussion comes up in the Early Modern period, in debates between rationalists and empiricists (Griffiths 2020). (I will note, however, that there are always exceptions to broad categories such as 'rationalism', so in what follows I will attempt to balance the broader story with more specific examples). Briefly put, a central point of contention between these two schools of thought centred around how we gain knowledge. Rationalism holds (to varying degrees) that at least some things are knowable by intuition alone, instead of requiring experience (Markie 2017).

For example, consider Descartes' famous claims on the matter. Intuition was fundamental to Descartes' claim that he cannot doubt that he is a thinking thing (the classic 'cogito ergo sum'). And importantly, for Descartes, this kind of intuitive-and-deduction based knowledge is more certain than anything our empirical experiences can provide (Markie 2017), since we cannot be certain that our empirical experiences are not the work of an all-powerful, malevolent demon. As Markie (2017) summarizes Descartes' position: “Descartes tells us, “all knowledge is certain and evident cognition” (1628, Rule II, p. 1) and when we “review all the actions of the intellect by means of which we are able to arrive at a knowledge of things with no fear of being mistaken,” we “recognize only two: intuition and deduction” (1628, Rule III, p. 3).” Thus, while Rationalists may disagree about various things, Descartes' reliance on intuition and deduction offers a classic, concrete, paradigm to work with to explain the parallels between rationalism/empiricism and more contemporary Nature/Nurture.

To couch the topic in slightly different terms, one might inquire as to the source of this certain knowledge. We might say that some knowledge is innate, or nativistic (ie present from birth). There can be different reasons for knowledge to be innate, but common themes invoked God or that at least some knowledge is required to be innate due to our rational nature (Markie 2017). In the *Meditations*, Descartes opts for the former option. Shapiro (2011) sketches the way Descartes connects these dots in the following way:

> *"[Descartes'] reflection on this drive for knowledge reveals it to entail the recognition that "I am a thinking thing." Equally, further reflection on what it is to be a thinking thing yields the criterion of certainty and knowledge. I take this to be the upshot of the Fourth Meditation. That meditation yields a method for avoiding error in our judgments of truth and falsity. That method, however, is premised on features of our nature as thinking things: that we have an idea of God and from that fact are able to assure ourselves that God is not a deceiver; and that we are able to perceive clearly and distinctly and are determined to affirm what we so perceive. Together these features of our nature entail that what we clearly and distinctly perceive is true." (Shapiro 2011, 16-17)*

Shapiro's highlighting of these facets of 'our nature as thinking things' helps to make the connection to how a Cartesian-style Rationalism serves as one part of an earlier iteration of a Nature/Nurture-style disagreement. By discussing thinking and our connection with God in this way, the connection is something like how our nature is part of our essence, and our nature is to be thinking things. Very clearly, this sort of thinking occupies the 'Nature' side of the divide, and Empiricism thus occupies the same contrasting position as 'Nurture'.

For empiricists (broadly speaking), there is no source of knowledge outside of experience (Markie 2017). On this way of thinking, knowledge is something we acquire, rather than something we are born with. Some empiricists distinguish between different kinds of experiences, and thus different sources of knowledge. For instance, Locke distinguishes between reflection and sensation *(Essay*, Bk 2; Uzgalis 2020). Where sensation lets us acquire knowledge about the world around us, reflection offers us knowledge of the goings-on in our own minds. More specifically, Uzgalis (2020) notes that "Locke holds that the mind is a *tabula rasa* or blank sheet until experience in the form of sensation and reflection provide the basic materials—simple ideas—out of which most of our more complex knowledge is constructed." Substantive knowledge of the

world around us thus relies on experience (sensation), and more complex ideas can be formed through reflection. Children may be born with minds ready to acquire information, but they are dependent on experience for their operation (Duschinsky 2012).

It is quite easy to see how empiricism (and Locke's approach in particular) serves as the Nurture-oriented foil to rationalism's Nature-analogue. Indeed, even the metaphor of the blank slate has carried over to modern times. But where the latter approach emphasizes knowledge and content divorced from experience, the former holds that experience is indispensable in forming knowledge. And so, while there are many other important aspects of the disagreement between these two schools of thought, part of the disagreement is about whether humans have these innate qualities, or whether we are blank slates that come to acquire our traits and knowledge. More pointedly: do we have a Nature that gives us certain knowledge or traits by default, or are traits and knowledge acquired entirely through Nurture?

Given that the themes of these conversations seem to repeat, it seems that there has not been merely one Nature/Nurture-style disagreement, but several. There are also other aspects to this history worth examining. For instance, there is also - understandably - substantial overlap between the Nature/Nurture distinction and discussions of human nature and its implications. Frustratingly, the term 'human nature' seems to have many meanings, and there is substantial disagreement about the term's explanatory power, its conceptual extension, and whether the term has any actual meaning at all (Roughley 2021). The term can also be used in various inflationary and deflationary ways. For instance, one might regard the human species as an essential historical product of evolution, and so there may be something essential that picks out the species against similar creatures. In a similar vein, Aristotle's claim that humans are 'rational animals', or allied claims that we are 'political animals', seek to characterize members of a particular group by virtue of a set of properties. As such, many aspects of human nature discussions can often draw on essentialism – properties or relations that constitute the structure of the grouping itself. There is a fairly easy connection one can see between discussions of essential qualities and innate properties; if X is a prerequisite to come into existence as a member of Y, then we may expect X to be innate. My aim here is not to give an entire

history of discussions of human nature – that would take several books – but I wanted to highlight the above issues. First, it's important to be aware that claims around human nature can have many meanings and so the term can be extremely vague as to its extension. Second, given the way the Nature/Nurture distinction operates, I think that the most common ways that discussions of human nature will connect with the distinction are through notions of innateness or essential properties. In some cases the properties in question might be described as innate: they might be traits that show up without 'nurture'; or they might be traits that seem to persist *despite* nurture.

One last piece of more recent history before we move on. The 20th Century had a few important points where some form of the Nature/Nurture distinction experienced a surge in popularity. One such period came both before and after WWII, pertaining to instincts (Griffiths 2020). This extended dialogue is often called the Lorenz – Lehrman debates (ie see Lehrman 1953; Lorenz 1937; Lorenz & Tinbergen 1938/1957). It was thought that instinctive behaviour was an evolved characteristic in the same way an organism's bone structure is, such that homologous comparisons could be made. Lehrman argued against this, noting that in many cases endogenous and exogenous (ie both internal and external) influences can interact when producing behaviour, and there are many documented environmental effects on behaviour. The point, said Lehrman (1970), is that Lorenz's attempt to draw a sharp distinction between instinct and acquired behaviour simply didn't stand up. Lorenz later responded that traits themselves are not innate, but the information which underlies the adaptedness of the trait is (Lorenz 1965). A trait can thus be considered innate if its development is guided by inherited information rather than environmental information. Thus, a distinction between inheritance and environmental acquisition is of great importance to Lorenz and his school of thought.

A second important event was the Minnesota Twin Studies. A comparative study involving hundreds of pairs of twins who were raised apart, the researchers (Bouchard et al 1990) hoped to discover the origins of individual differences in various traits such as individual abilities, social attitudes, etc. The thought was that differences in monozygotic twins should be caused by environmental effects (since monozygotic twins come from a single zygote and thus share all their DNA, meaning there are no differences in DNA

between them). As such, the hope was that by exploiting the genetic similarity between twins, we could learn about how traits developed. Given the general findings of the study that monozygotic twins raised separately had the same chance of being similar as if they were raised together (Bouchard et al 1990; Bajhat 2017), the study was very important for those arguing that genetics played a strong role in shaping developmental outcomes (as opposed to environmental influence). (It's worth mentioning at this point that I am skeptical of the evidential weight of twin studies, and in Chapter Five I explicitly critique their methodology and use. However, since the Minnesota study was large and influential, it is still worth noting this study as historical background). But if we think that development is mostly governed by the genes, then we might think that (other things being equal) a developmental pathway is largely preordained if you 'have the genes for it'. This way of thinking is connected to preformationism, and is the next bit of terminology I will focus on.

1.1.2 - Preformationism

Preformationism (and its competing alternative: epigenesis) is a school that has had a few different incarnations, and each incarnation has had varying terminology and focuses. The core dispute around which preformationism and epigenesis are arranged involves the following question: do entities have form and organization from their earliest point of existence, or does their form and organization merely arises over time? For preformationists, form and organization are part of the organism since its very beginning (i.e. see Maienschein 2017). One of the reasons someone might favour this sort of approach is that it lets you 'bake in' some phenomenon to be explained. For instance, if we think that form only arises over time and beings otherwise start 'unformed', then what is it that causes this form to arise? How does something interact with a formless entity? If some sort of organization is part of an organism from its earliest point, then we can start to get around tricky questions like 'how does an 'organizing spirit or life force get in to the formless entity and affect it?'

Over time, preformationism became less about the preexistence of form, and more about some kind of predetermination or predestination (Maienschein 2017). In the contemporary context, genetic determinism offers a version of preformationism, by

appealing to evolutionary influence of genetic content, whereby the phenotype is formed through genetic inheritance. Even outside of genetic determinism, contemporary preformationism is much more a doctrine about information and how that information specifies or organizes something. For instance, it might seek to explain the reliable outcomes of form for individuals. The adult phenotype (i.e. the set of developmental outcomes for the adult organism associated with its genotype) is a phenomenon to be explained; why is it like X instead of like Y (i.e. why do most humans have roughly the same body shape and proportions? If the parent has phenotype X, why does the offspring reliably have a similar phenotype?) The preformationist attempts to solve this problem by specifying where the information for the final product is contained, and the channels by which this information is accessed. The idea is that this information about the adult phenotype precedes the existence of the actual adult phenotype, just as a blueprint precedes the construction of a building. Thus, for the preformationist, development is just the unravelling of a pre-existing 'plan' for how the completed phenotype will be. The discovery of DNA gave a mechanism for this sort of story; after all, genes are the result of evolutionary selection (they have been selected *for*). Since genes can persist across generations and precede the organism itself, that continuity is taken to explain continuity of form for the organism. Through sexual reproduction, alleles are passed on to the offspring. If one assumes that the blueprint for the phenotype is in the genes, then sexual reproduction can explain both how the offspring comes to have this phenotype-specifying information and why developmental outcomes seem so reliable. (Their genes already possess the finished plan already, and development consists in the executing/ unravelling of that plan, so following this pre-formed plan explains the reliability of form and inheritance). This gives rise to metaphors of genetic programming and hard-wiring to capture the way in which the final phenotype is aimed at. In the 1800s, Haeckel offers a classic example of a preformationist view. His 1867 work claims that ontogeny (ie development) is the rapid recapitulation of phylogeny (ie a species' evolutionary history). On this view, an individual organism's development follows and is caused by the evolutionary history of that individual's species. As Maienschein (2017) puts it, summarizing Haeckel's view: “Form comes from form of the ancestors, and unfolds following pre-scripted stages.” Speaking in this way clearly evokes a sense of

development aiming at a target, where that target is set by the evolutionary history of the species, and development just is the unfolding of a preexisting plan towards the predetermined target.

The contrasting view of preformationism is epigenesis: the view that organisms do not start formed. Rather, form only emerges slowly over the course of development (Maienschein 2017). Aristotle had such a view. In looking at baby chicks, he noted that early eggs do not house completed chicks in them. Rather, the chick gradually came to have a heart and other organs; very slowly did form emerge from the not-formed (Aristotle 1979). While the parents might be the generators of new life, each life must begin anew and unformed. Older accounts of epigenesis tended to be paired with vitalism, the view that living organisms are fundamentally different from nonliving things because they possess some kind of fundamental life force. At the time (through the 18th century), accepting epigenesis also involved accepting vitalism, since the life force was seen as driving the emergence of form from the unformed (Maienschein 2017). Various stripes of epigenesis have clashed with preformationist views for centuries. However, the acceptance of genetics and DNA inheritance was seen as an evidential tilt in favour of preformationism, especially as vitalism became more and more unpopular, and so epigenesis as a result.

1.1.3 - Interactionism

If one does not endorse preformationism or epigenesis, interactionism offers something of a compromise. To be an interactionist about development, one thinks that developmental outcomes/ individual form are explained by both internal and external causal factors (see Barker 2015; attributed to Godfrey-Smith). Thus, there is a relatively low barrier to entry, but the potential for substantial disagreement over what parts of individual form are attributable to internal or external causes, or to what degree they are so-attributable, etc. As such, there is a great deal of theoretical room for different kinds of interactionism that all potentially disagree on what sorts of things are interacting and to what degree that matters.

In the wake of the Nature/Nurture debates, most people seemed to settle on the idea that development is not explained wholly by one category or another, but rather both serve a role (Tabery 2014). This shift is important for a couple reasons. First, as Oyama (1985/2000a; 2000b) discusses at length, the shift was accompanied by a change in the language used, but the change in language merely papered over conceptual difficulties in the Nature/Nurture framework. As such, the conceptual difficulties were not fixed; they were merely imported with a new paint job that concealed the old cracks in the foundation. But to the degree that the old cracks are concealed, this means that it can be an uphill battle to convince folks that the conceptual issues have remained. Oyama remarked in 1985 that a relatively common reaction to her work on the need for interactionism and to finally move beyond Nature/Nurture was confusion. To paraphrase: "we're all interactionists; why do you keep beating the dead horse of Nature/Nurture? We all agree that it was problematic and have moved beyond it." Oyama's response was that she would love nothing more than to stop beating a dead horse, but every time she turns around, the horse kicks her, or does rude things to the state of the literature, and so she feels that the fight has not yet been resolved. For a number of reasons that I will touch on through the course of this project, I think Oyama was – and still is – quite right in her diagnosis of this problem. But her interlocutors were right to point out that everyone is an interactionist nowadays; you almost never see someone espousing pure genetic or environmental determinism. And so this means the crux of the issue involves disagreement between different forms of interactionism. As Oyama would put it, the point is to find interactionism worth having; a form that does not simply paper over the foundational cracks in the old framework, but instead engages with those conceptual issues and works to resolve them.

While it may be true that everyone nowadays is an interactionist, the received view is a particularly weak form of interactionism. I adopt Barker's (2015) terminology in describing this general view: conservative interactionism. While I describe this view more in subsequent chapters (especially Chapter Three), it is worth saying a few things presently as well. In short, conservative interactionism is interactionist in the sense that it admits both internal and external factors explain individual form. However, its idea of interactionism is additive: internal causes (typically genes) and external causes (typically

environmental) separately contribute to development, and development just consists in summing genetic and environmental effects. This sort of idea is easily visible when folks say that 'both' Nature and Nurture are responsible for development. Typically, that statement means Nature and Nurture each pull in their respective directions, with the developmental outcome being somewhere in the middle depending on the respective strength of the pulling on either side. Both Nature and Nurture are seen as sources of individual form.

This view can also be known by other names. Besides conservative interactionism, sometimes the term ‘variation-partitioning’ is used (Tabery 2014). According to Tabery, the aim of this approach is to try and explain the Nature/Nurture relationship in terms of statistics (Nepi 2018), with a focus on determining how much variation can be attributed to opposing factors. On this approach, traits in a population can be attributed either to genetics or the environment depending on how their variation is statistically partitioned, with the explicit assumption that the interaction is additive.

This type of talk crops up commonly in evolutionary psychology, and according to some scholars (see Jablonka & Lamb 2014) also closely matches the way the general public tends to think about genetic causation. According to Barker (2015), folks who espouse some kind of conservative interactionism will make the following sort of claims. First, both internal an external causes are related to development. The difference is that the internal causes – the DNA blueprint, or a similar metaphor – reflect the evolved nature of the creature and so have a preferred developmental outcome that they aim at. External causes merely serve to enable or modify this outcome in some way. An example of this is Dawkins' (1976) metaphor of development and inheritance as a recipe: the recipe determines what kind of dish is being made (internal causation), but leaving the dish too long in the oven (or not having the right ingredients) can substantially alter the outcome of the recipe. This leaves room for environmental effects to alter the developmental outcome. However, since the internal causes 'prefer' some outcomes to others and thus must resist perturbation to some extent, more and more extreme environmental effects are needed to push the outcome further from its 'intended path'. This suggests that the type of interaction implied here is additive: internal causes will try

to keep the outcome close to a preferred state, and as a result, external forces must be quite powerful to diverge from this. This offers a clear picture of some phenomena as 'more natural' than others depending on how close to the supposedly preferred outcome they are.

This type of conservative interactionism is certainly taken to be the standard, received view in much of developmental science by the postgenomics literature. As Stotz (2008) remarks: “Today’s received view of development attempts to reconcile both [the preformationist and the emergent] visions: a (multicellular) organism begins as one cell packed with ‘innate’information of how to build the phenotype, from which the final form emerges in interaction with the ‘acquired’ influences from the environment.” (Clarification added). Clearly, on this type of picture both Nature and Nurture contribute to ontogeny as separate categories. Given the insistence that the environment only serves to modify the recipe rather than be a part of the teleology, this perspective also seems to rely on the idea that internal causes constitute the nature of the thing and the environmental causes map onto its nurture. Thus, the nature is internal, causal, stable and genetically inheritable, whereas the nurture is external, modifying, plastic and variable (Stotz 2008; Kitcher 2001). We can see a sketch of the above stance in action through the Central Dogma of molecular biology, which is the next bit of terminology I will cover.

1.1.4 – Central Dogma/ Genetics Primer

In this section, I want to cover both the basics of genetics as well as the Central Dogma, to make sure the reader can better understand the nuances of the discussion. I first give an overview of important terms in very simplified form, then show their role in the Central Dogma, and finish with a brief discussion of how the Dogma upholds the Nature/Nurture framework.

To start, most people are familiar with the double helix form of DNA, which is in the nucleus of our cells. The fact that it's a double helix means there are two complementary strands that are coiled together. During cell division, the DNA needs to be replicated. For replication to happen, this double helix is 'unzipped' and the strands are

separated. Each half of the double helix thus serves as a template for the copying and construction of its counterpart.

Chromosomes are structures in the nucleus of the cell that are comprised of long sequences of DNA. DNA is composed of nucleotides in a long chain, and sequences of DNA/ nucleotides that code for proteins are called genes. RNA are strands of nucleotides like DNA, but there are a few different kinds, and they perform different roles within the cell. Messenger RNA (mRNA) is so-called because it serves as the messenger from the DNA to the ribosome (a part of the cell outside the nucleus), and the ribosomes begin constructing proteins based on the sequence of nucleotides brought by the RNA. This whole sequence is called transcription and translation: DNA is unzipped and transcribed to RNA (transcription), which then takes the nucleotide sequences to the ribosomes to begin the construction of proteins and amino acids (translation). Since genes are composed of sequences of DNA (and thus nucleotides) that code for proteins, translation is the process through which genes are associated with the proteins they are said to 'code for'. Moreover, these terms – DNA, RNA, and proteins – are important for understanding the Central Dogma, which I will describe presently.

The Central Dogma, like so many of the topics I have covered above, concerns the flow of information within biological systems. Specifically, the Central Dogma holds that genetic information flows in only one – 'outward' – direction: from DNA, to RNA, to proteins, to phenotype, where a phenotype is the collection of an organism's traits (Mattick 2004; Bussard 2005). An extension of this dogma was the belief that genes were universally (or at least primarily) protein-coding (Mattick 2004). The upshot of this picture is that genetic information pipeline involves genes coding for proteins via mRNA and translation, which means genes are generally synonymous with the proteins that they code for, and the information for the phenotype comes in the form of these proteins.

The ways in which this picture supports the Nature/Nurture distinction is a central topic of Chapter Three. However, it is worth giving the reader an early (if abbreviated) picture of where this whole line of argumentation is leading. There are three points to make here regarding how this picture of genetics imports the Nature/Nurture distinction:

the 'innateness' of DNA; the entirely outward flow of information from gene to phenotype, and the way development is understood on this framework.

First, while inheritance is covered more fully in Chapter Four, everyone has a general understanding of how sexual reproduction means we inherit a subset of the genes of our parents. The gametes (sex cells) of our parents meet, and fertilization involves the unification of the two sets of DNA. Thus, in a very straightforward sense, DNA is present in the zygote from its earliest existence. To be a zygote is (among other things), to have DNA. If we take DNA sequences (ie genes) to code for proteins, then it is a small step to say that the genetic products of the genome are determined as soon as the genetic materials of the parents are combined. In this sense, this stance favours an 'innateness' of DNA: it is present from the earliest point of an organism's existence, and (by definition) to have a gene is for it to code for proteins. Additionally, half of our genetic material (and whatever proteins it will code for) precede our existence entirely, since this material comes from our parents, grandparents and so on. Thus, it is easy to see how a focus on genes and their 'genetic information' can have a preformationist slant, since many of the genes we inherit pre-exist us.

Second, as per the Central Dogma, there is an outward, unidirectional flow of information from DNA to phenotype. While this need not imply that all of the information for the phenotype is genetic, it does imply a certain level of independence or interactive isolation of genetic information. This is to say that while there is the possibility of various types of genetic interactions (ie gene x gene; gene x environment), according to the Central Dogma these interactions are not taken to change the informational contents of the genes themselves. To say otherwise would violate the unidirectional flow of genetic information. This means that if we think genes contain information, and this information can precede our existence, then that information is stable since information only flows outward from it. This is exactly the sort of role that Nature fulfils; some stable entity with innate information about how the organism will turn out (ie its phenotype). It doesn't block the possibility of environmental effects (ie Nurture), but it maintains that environmental effects are informationally distinct from genetics, since there can only be outward flow of genetic information. Even the

admission of gene x gene, or gene x environment interactions doesn't change this picture. Remember that, on conservative interactionism, genes and environment each contribute separately as a source of individual form. But that is a developmental topic, which leads to my third point.

Third, there is very much an implied picture of development on this approach. If the information for the phenotype is already 'in the genes' and flows outward, then development can be understood as the unravelling and deployment of that information to form the phenotype. The genetic component of development is thus separate from (and can be potentially interacted with and modified by) environmental information. This conception of development is why there are metaphors of 'genetic blueprints', or of hard-wired information, since development just is the execution of pre-existing genetic instructions and products. There might be environmental factors that modify this plan in some way (ie if there is insufficient food, so the body can no longer execute the intended plan), but that is perfectly consistent with the conservative interactionist picture. If development primarily consists in following genetic information (modified by local environment), then this picture of development has retained the functional description of Nature/Nurture. Genes and the central dogma play the role of Nature, while Nurture is accounted for through environmental interactions, learning, and other such effects. The developed organism just is taken to be the combination of these two factors, which each contribute their own information for specifying the final phenotype. The picture is interactionist insofar as the phenotype is explained by reference to both internal (genetic information/Nature) and external (environmental/Nurture) causal forces.

One final note before we move on to motivating my own project. In the above sections, I used the term 'phenotype' several times, and mention its connection with an organism's genotype. There is a long history of these two terms being used together, linking the way genes, genome and traits are discussed together. I have kept using these terms in the historical sections above because I think that is most faithful to the respective positions I have described. However, I dislike the genotype/phenotype terminology, as I think it helps trap us in a kind of preformationist language. In the sections to come (and

throughout the rest of the project), I eschew these terms and instead refer to 'developmental outcomes' (or occasionally just 'outcomes'). In making this choice, my aim is to avoid some of that complicated history and speak more directly about the developmental outcomes themselves, without insinuating that the conversation must loop – without fail - back to the genes that underlie these traits.

1.2 – Motivation & Aims

With some background information out of the way, we can now turn to what I would like to accomplish in this project. My project has both negative and positive portions, as well as descriptive and prescriptive aspects. The central aim of the negative project is twofold. First, it aims to describe ways in which contemporary developmental biology has smuggled in conceptual ties to the Nature/Nurture framework. By exposing these conceptual ties, I also want to argue that there is a mismatch between the types of evidence available to us under a Nature/Nurture framework and the types of evidence that we actually have. This mismatch is part of my second, larger, argument that the Nature/Nurture framework is not merely outdated, but is conceptually incoherent. Thus, if we are importing parts of an incoherent framework into contemporary thought, there is strong motivation to understand how it is incoherent and what tools we might have available to try and fix any potential issues.

I don't want this project to come across as strictly negative. That is to say, I am not merely arguing that the Nature/Nurture framework is wrong, but I am trying to advance a different way of understanding what development is. The core issue, as I see it, rides on what it means to be an interactionist about development. I highlighted this in the background section above on interactionism: while everyone is an interactionist nowadays, not all forms are created equal, and the point is to advocate for a kind of interactionism that doesn't commit us to the same mistakes we are trying to escape from. For present purposes, however, my positive project argues that we should replace an additive understanding of development with one that holds it to be irreducibly interactive. This is not mere terminological gloss. The picture I am putting forward has strong conceptual ties to Oyama (ie see 2000a/b), the postgenomic literature (ie Stotz 2006; 2008), and the extended evolutionary synthesis (ie Jablonka & Lamb 2020; Laland et al

2015). It is a picture wherein development is highly contingent, and any understanding of human nature is just identical with whatever phenotype results from the ontogenetic (developmental) niche. This is a picture where we have access to multiple genetic and non-genetic inheritance systems that overlap and interact in various ways. It is a picture where there is effectively no distinction between Nature and Nurture, because they are subsumed by a larger developmental system whose components can intertwine, co-determine each others' products and interact in reliable or novel ways. It is also a picture, I think, that better accounts for the available evidence than any sort of framework with conceptual ties to Nature/Nurture. The hope is that it is the sort of picture that will allow us to move forward, having finally shed this pernicious shadow of Nature/Nurture, and embrace conceptual clarity.

My motivations for why we ought to reject the Nature/Nurture framework are twofold. First, it is socially pernicious. Second, as alluded to above, I think the distinction itself between Nature and Nurture is conceptually incoherent, and thus not a real distinction. I take these two claims to be somewhat related. After all, if I am correct and there is no real distinction to be made between Nature and Nurture, then it becomes very easy to manipulate what gets classified as either category. Insofar as the scheme is changeable and bound to ideological/political positions, then it might also resist attempts at scientific clarification. So there is a connected worry that epistemic and political concerns are tied together in this fashion. Certainly there is the potential for ideas about an innate human nature to be misused politically. Frankly, I would argue that many such arguments have already been made, and I will touch on some of these below.

A framework's perniciousness also serves as motivation for rejecting it, though of course other criteria may be required as part of a more thorough argument. For present purposes, I think there are two main threads that highlight the perniciousness of the Nature/Nurture framework, and serve as motivation both for its rejection and for a closer analysis of how we manage to keep importing its contents into contemporary concepts. These two threads are: A) the notion that 'biology implies destiny or teleology'; B) the related idea that biological traits imply specific social roles or hierarchies. The former thread mostly aims at highlighting outcomes, while the latter is more oriented towards

interpreting the developmental outcome. (Thus, one can disagree both about the reliability of some developmental outcome given a specified biological factor – the former thread – and/or the value judgement that such outcomes just naturally imply hierarchies of social value). Much of the critique of these two themes centres around how they ignore the remarkable plasticity that humans seem to exhibit, and thus fail to successfully imply that social or political conclusions follow from any 'fixed' nature of the biological underpinnings. These critiques nicely highlight the points of disagreement about how to characterize the evidence that is available to us. But before matters get too far ahead, I want to look at the above threads in more detail.

1.2.1 - Examples of Social Perniciousness

The first type of example of perniciousness I'm going to discuss involves the invocation that biology implies destiny. This is also sometimes referred to as 'biofatalism' (ie see Barker 2015). As with any claim, the exact form and strength of it can vary, and such variance affects the degree to which it shows up in various literature. Perhaps the most egregious is so-called 'race science', which often seeks to explain group differences - of whatever variety – in terms of genetic underpinnings and the heritability of traits. Frequently this involves a fascination with group differences in IQ, and the degree to which any such differences remain stable across generations. Murray and Herrnstein's *The Bell Curve* (1994) offers a classic, if now dated, example of this type of approach (for a more recent applications of these same principles, consider Murray's 2021 book *Facing Reality: Two Truths About Race in America)*. In *The Bell Curve*, while the authors suggest that IQ is influenced by both genetic and environmental components, one of the central worries of the book pertains to the stratification people into high-IQ and lower-IQ classes through the heritability of IQ. This leads the authors to some rather ugly conclusions about the predicted difficulties in managing a nation comprised increasingly of low-IQ groups, and that social welfare programs only encourage poor women to have more children. And given the focus on heritability of IQ, the worry is that if lower-IQ groups have more children then this accelerates stratification. Even within the authors' framework, this supposed phenomenon would be less of a worry if they thought IQ was highly malleable. However, they claim that IQ scores are largely stable over a person's

life (though not perfectly so; some variation is expected). As such, the claim about the relative stability of IQ, coupled with the (usually) high heritability of cognitive abilities, jointly underlie their conclusion that contemporary egalitarian politics are somehow in conflict with the biology of differences. In this case, the argument is that biology is destiny, given the stability of IQ scores and the role of heritability (despite what politics we might endorse).

While the first thread (biology as destiny) is readily visible, the second thread is also present. *The Bell Curve* explicitly paints a picture of a political future where some large segment of the population (the lower IQ group) may need to effectively be made wards of the state. In order for this conclusion to be drawn, the authors need to explicitly assume that the variables are comparable against a linear scale, but also that lower scores imply a certain social role by virtue of being at the bottom of the hierarchy. We want smarter people in charge, and so the presence of a given trait (high IQ score) implies a better social role than a lower score. And given the additional assumption that biology is destiny, these social roles are unlikely to change dramatically for an individual (and probably even less for groups as a whole, given the emphasis on heritability).

It goes somewhat without saying at this point that the methods, assumptions and conclusions of *The Bell Curve* have been soundly critiqued regarding their lack of scientific rigour (ie see Graves 2001; Gould 1996). There is even a chapter in Herron & Freeman's (2014) textbook *Evolutionary Analysis* devoted to debunking the 'Bell Curve Fallacy'. So I am invoking this book because I think there is a nugget of wisdom worth discussing, but it is explicitly a nugget of wisdom about how the representation of biology in the various strains of pop-science can have negative social impacts. These impacts, in turn, can motivate a higher level of scrutiny on the frameworks that enable these negative social impacts. As this topic pertains to the developmental issues I am concerned with, the clear emphasis on genetic underpinnings of some manifest trait is taken (whether by the authors or racist layfolk) to mean two things. First, it is taken to mean that there is a sufficiently deep genetic difference between races, and second that any manifest differences are to be primarily explained by internal, genetic, factors (often because of the heritability of the trait in question). This emphasizes the notion that there

are genes 'for' specific traits – IQ, aggressiveness, etc – which in turn implies that since genes 'govern' these traits, it is the possession of the gene (or lack of it) that determines the developmental outcome. If this framework were taken seriously, then attempts to intervene in society and make people's lives better may be doomed to fail. After all, if one must possess the requisite gene(s) to have trait X, and group Y does not have possess them, then external interventions may be unlikely to accomplish anything since the requisite source of internal causation is missing. This theme has historical usage in a fairly oppressive context, as it is often used as a fatalistic, naturalized argument for why some marginalized group 'will never do better'.

A less dark variation on the theme of 'biology implies destiny' instead implies that biology and development has a certain kind of teleology. Teleology, famously, is one of Aristotle's four kinds of causation. It involves explaining things in terms of the purpose or goal that they aim at, instead of more proximal causes. For example, one might ask what caused a business to open, and one way of answering would be to say that the business opened in order to make a profit (as opposed to saying the owner turned on the lights and unlocked the door). As a second example, forks have prongs in order to help us spear food for consumption (as opposed to specifying how the shape of a fork is punched by machinery). There are a few ways one can invoke teleology about biology, and lots of literature about the role of teleology in biology (Lennox 1993; Allen & Neal 2020; Mayr 1988). A recurring theme involves tying teleology to explanation, and asking whether teleology is indispensable for explanation (Allen & Neal 2020). But often tied to this focus on explanations is the additional notion of teleology-as-design, where organisms are they way they are because they were designed to be that way (whether by God or, more commonly, Natural Selection). For example, one might say that the purpose of the heart is to pump blood, and that purpose came about through natural selection.

1.2.2 – Teleology & Contemporary examples

Against this backdrop, I have a slightly more narrow stripe of teleology-as-design in mind that pertains to development. This kind of teleology is the sort that shows up in discussions of targeted outcomes, or aims, such that some outcomes are preferred and in some ways sought after by the system. In other words, development is treated as a goal-

directed process, as the system comes to be structured in the way it was designed to be structured. This is important because a notion of targeted outcomes implies a rightness or wrongness about some state of affairs, since outcomes can be categorized based on whether they were preferred or not (or the degree to which the outcome missed the target, etc). To have a target to aim at implies a preference for certain kinds of outcomes, but to have a system aiming at a target suggests that the system itself works to bring about the targeted outcomes against other possibilities. As such, it becomes easier to see how this notion of teleology-as-target functions as a weaker version of the 'biology implies destiny' theme. After all, if a system inherently 'prefers' some outcome or set of outcomes and blocks alternatives, it is a small step to say that possessing System X 'just destines' (or 'predisposes') you to have Outcome Y, since that is the target the system aims at naturally. Thus, barring some sort of intervention or perturbation of the system that the targeting 'controls' cannot account for, it is nearly a given that individuals with System X (whatever it might be) will probably also have Outcome Y. Moreover, if individuals have System X but do not have Outcome Y, one might think something has gone wrong in some sense. If a system is said to be aiming at some target, then not having that target obtain is to miss the target and have something go wrong. However, the capability to miss in this way is a large part of why I take this to be a distinct (and less strong) version of the 'biology implies destiny' notion. While the two can overlap with their stance on how the future individual form/outcome is determined by internal causes, the aiming metaphor implies that the relationship between System X and Outcome Y might not be absolute in the way that 'destiny' implies.

For a more contemporary example of how both biology-as-destiny and biology-as-natural-hierarchies are employed, one can consider the kinds of rhetoric around the way transgender folks are treated, and how trans identities are pathologized under traditional gender frameworks. There is a massive literature on this topic, and I cannot hope to cover it all here as that would be the work of several books. Instead, I am only trying to dip a toe into this topic, since I think it links up with the themes of my project in interesting ways, and frankly I think it is a topic of great contemporary interest that explicitly weaves in questions of biology, social identity, gender performance, and yet more threads. (Very obviously, content warning for the following couple paragraphs).

While we have made some progress in normalizing and accepting trans folks, there is a blatant history of pathologizing trans identities, especially when it comes to accessing medical services that require a previous diagnosis of gender dysphoria or identity disorder (Prunas 2019; Schultz 2018). In a society that thinks of gender as a rigid binary which stems from the sex one is assigned at birth, the existence of trans and non-binary folks potentially gets understood as mistakes of development. In other words, that way of thinking about the relationship between sex and gender mirrors the notion that biology implies destiny, where being assigned a given sex at birth just implies a corresponding gender performance. On this traditional framework, to break this cycle (from sex to corresponding gender) is to have one's development go awry in some fashion, since there is thought to be a preferred outcome that is aimed at. This idea of something having gone wrong then gives teeth to attempts at treating such outcomes as pathological. Additionally, older narratives of 'being born in the wrong body' also feed into this sense of development gone awry (even though these narratives may be useful or important for some trans folks in other ways).

Similarly, there is a great deal of contemporary discussion about the inclusion of trans folk in sports, and what 'fairness in sport' means in light of this topic. Most frequently, news articles and public discussion will centre around trans-women participating in women's sports, especially if they are successfully winning these events. Again, there is a lot going on in such discursive events, and I am – by necessities of space and time for the current project – only giving a very truncated sketch of such goings-on. However, I think one thread that can be found in these discussions is the implied suggestion of sex-based developmental pathways. That is to say, to be male is to developmentally acquire (at some point) a set of properties that are stereotypically associated in some way with male-ness. This can include a greater predisposition for gaining muscle, for strength, etc; pick your favourite traditionally male-associated trait. But such an attitude does not help trans folk find acceptance. Instead, it suggests that a trans woman participating in women's sports is still likely to acquire (if they have not already) characteristics associated with their birth-assigned sex, which cis women ostensibly do not have access to. Again, this way of thinking about development is to treat it as the mere unrolling of a predestined set of characteristics; as if a blueprint were

established as soon as sex was assigned. While there are certainly elements of biology-as-destiny in this rhetoric, I think it is somewhere in between that stronger version and the weaker one, biology-as-target. (Which version is maximally in play at a given time will likely depend on the speaker). My suggestion that the weaker version might be part of the discussion is contingent on the degree to which we think 'maleness', etc is monolithic. For example, there are obviously human males who are not particularly strong, and females who are very strong and would beat a very large portion of males at strength-based tests (or tests of speed, etc). As such, the degree to which these properties are treated as statistical ranges (as opposed to specific values of a property) reflects the way that one can invoke either the stronger or weaker version of development as biology-as-destiny.

In the same 'biology as destiny' vein, there are other contemporary examples. For instance, consider contemporary (August 2019; 2021) studies about whether or not there is a so-called 'gay gene'. These studies have been featured in *Nature* magazine (ie Lambert August 29th, 2019; Reardon August 23rd, 2021) indicating that this is not a mere fringe conversation. The general consensus seems to be there there is not a 'gay gene', but it is still worth noting how these results are discussed. The first article (Lambert 2019) cites Ganna et al (2019), who claim that there is no 'gay gene' per se. However, the study did ostensibly point to five spots on the genome linked with same-sex behaviour, though none of these was reliably predictive of individual sexuality. However, genetics and environment are again pitted as alternative (competing) explanations of behaviour: “Ganna and his colleagues also used the analysis to estimate that up to 25% of sexual behaviour can be explained by genetics, with the rest influenced by environmental and cultural factors — a figure similar to the findings of smaller studies.” (Lambert 2019)

Similarly, Reardon (2021) cites a study by Zietsch et al that looks at modelling the genetic inheritance of DNA that may predispose individuals to same-sex encounters. By studying over half a million genomes, Zietsch et al (2021) looked at previous studies that noted small DNA similarities between individuals who admitted to same-sex encounters, and modelled these over 60 generations. “They found that the array of genetic variations associated with same-sex behaviour would have eventually disappeared, unless it somehow helped people to survive or reproduce.” (Reardon, 2021).

The study also noted genetic overlap between regions associated with same-sex behaviour and risk-taking behaviours. This lead some commentators to interpret the study as being less about sexuality, and more about other factors. Dean Hamer (retired geneticist who conducted very similar studies) was cited as commenting: “Instead, [Hamer] thinks the researchers have found genetic markers associated with openness to new experiences, which could explain the overlap between people who have had a homosexual partner and heterosexual people who have had many partners.” (Lambert, Aug 29th, 2021 – clarification added)

My point is less about the individual results of these studies, and more about the overall picture that I want to highlight. I began this section by citing *The Bell Curve*, which is hardly contemporary work, even if some of the thought patterns have persisted. But I don't want to imply that I am merely engaging with the ghosts of the past, and so I have cited Murray's recent (2021) book, a contemporary discussion on biology of trans athletes, and the way we frame studies around the search for/ repudiation of the notion of a 'gay gene'. These are all contemporary discussions, and so, to the degree that these conversations import aspects of the Nature/Nurture distinction, this means we are still grappling with aspects of that distinction even as we regard it as an issue that we have surpassed.

The above examples are meant to give a quick taste of some ways that I think the remnants of the Nature/Nurture framework can be socially pernicious. If a framework leads to negative social outcomes, then those outcomes might serve as some small motivation for modifying or abandoning the framework. Of course, other values might also be in play, and there can be interesting discussions about potential tradeoffs between epistemic and political concerns. So if there were useful epistemic reasons to keep the Nature/Nurture framework around, then the cost/benefit analysis might be more complicated. However, throughout the rest of this project, I am arguing that the frameworks associated with Nature/Nurture, such as contemporary conservative interactionism, are not epistemically illuminating and do not easily accommodate the available evidence. Instead, a different and more radical kind of interactionism is needed to understand development.

Speaking of understanding development, there is a point worth mentioning here before we get too deep into the project itself. In this volume, I frequently make use of the term 'development'. This is a term that can mean many things to many different people. For developmental biologists, the focus may be on how particular tissues fold, how cell differentiation occurs, or how patterns form (i.e. multiple valves in the heart; limb symmetry, etc.) (Love 2020). Other fields, such as behaviour genetics, may instead look to associate some gene of interest (or genetic activity of that gene) with some particular trait or property (i.e. aggressiveness) (Longino 2013). My approach to the term is fairly holistic, which means that I will likely use the term in ways that are much more broad than many researchers in genetics, cell and developmental biology might recognize. The background through which I am approaching this project is developmental systems theory, which itself has quite a holistic approach to what counts as part of development (Longino 2013). For me, development includes the processes and causes (both broad background conditions and more proximal mechanisms) which, over time, shape and maintain the continual expression of some trait or property of an organism. And indeed, the stability or change in some trait or property of the organism is part of its developmental trajectory. To lift the curtain on a central thesis of this project, I do not think that genes and environmental factors are developmentally separable, and that developmental trajectories fundamentally rely on the interaction of both. I also think that some 'developmental outputs' (i.e. behaviour) can be part of a feedback mechanism that may then change or reinforce the organism's ongoing developmental processes over time. As Longino (2013, 83) puts it (citing Cairns 1991):

> *"ontogeny (the development of the individual organism) is guided by three types of factor: (1) near universal maturational and contextual factors, i.e. genetic and environmental features common to all members of a population or species; (2) idiosyncratic but maturational and contextual-relational factors, i.e., genetic and environmental factors peculiar to the individual; and (3) constraints generated by the developing individual in the course of development. Any developmental trajectory reflects a fusion of these three factors."*

As such, I may refer to development in ways that pick out any of these three factors, or to reference the coming-about-over-time-ness of some trait. This is a very broad use of the term, and indeed the holism is part of the point, given the aims of this project. But the reader should keep the above discussion in mind when reading through my arguments, and remember that development is intended to be understood in an extremely broad way.

1.3 – Chapter Summaries

In this introduction, I have thus far introduced my larger project, offered some motivations for it, given a brief glossary of important terms, and quickly sketched how I see some of those terms and threads fitting together. My overarching goals are to critique the way contemporary conservative interactionism upholds the Nature/Nurture distinction, argue that the distinction itself is incoherent, and replace conservative interactionism with a more radical form of interactionism that better matches what the evidence warrants. In so doing, I want to dissolve the 'two-origin' story of Nature/Nurture and replace it with a different theory about how the developmental system spans these traditional divisions. These goals require several argumentative steps to be in place before everything can be tied together, so in what follows I will offer a brief summary of the chapters that follow, and remark on how I see the parts fitting into the larger whole. This should offer the reader a helpful overview of what is to come.

1.3.1 – Chapter 2: Candidate Notions of Interaction

Chapter Two aims at a modest kind of linguistic anthropology. That is to say, if proverbial battlegrounds are between different kinds of interactionism, then it is useful to canvass different ways in which the term 'interaction' can be used. This chapter discusses interaction as causation, exchange, statistics, and coupled equations. These senses of interaction can be found across the sciences, from physics to psychology, biology, chemistry, and so on. While I think most of these forms of interaction will have some role to play in the story of development, I pay special attention to interaction in the statistical and coupled equation sense. The reason is that I think they are especially useful in articulating my more radical understanding of interactionism. For instance, statistical

interaction gives us a well-understood sense of how two variables can depend on one another, whereas the coupled equation sense gives a well-understood depiction of how some system can be 'irreducible'. Throughout the dissertation I rely heavily on this notion to illustrate what I mean when I discuss development as the irreducible interaction of a larger developmental system (ie the 'phenotype-in-transition', as Oyama (2000a) puts it).

1.3.2 – Chapter 3: Genetics and the Extended Genome

Chapter Three expands on the interactions discussed in Chapter Two, with a specific focus on interactions in genetics. The chapter is composed of two main sections, in an 'as imagined vs how actually' contrast. The first section goes over the received view (the 'as imagined'), again describing conservative interactionism and its fundamental connection with contemporary gene-centric views and neo-Darwinian thought. I also allude to the historical battlegrounds between neo-Darwinism and Lamarckism, to help show how the lines between matters traditionally understood as Nature and Nurture got strengthened and included in biological frameworks. The second section offers what I think is a more realistic (ie 'vs how actually') picture of interactionism in genetics. I first defend the Extended Genome Thesis using several examples, with the aim of showing that the genetic 'context' extends beyond the body (hence the term 'Extended Genome'). Because this genetic context extends beyond the body, and given more recent work with the genome has emphasized the metaphor of a reactive genome (rather than proactive genes), I argue that the gene-environment “dichotomy” actually functions as a larger coupled system that transcends this divide. Because it is coupled in this way, it can be understood as interaction in the sense of nonlinear coupled equations – mutually dependent variables, and the equation is not reducible to the independent sum of its parts. A central take-away from this is that if my arguments are correct, then the gene/environment dichotomy that makes up part of the traditional Nature/Nurture framework is not much of a dichotomy at all. The parts are not opposing, but rather are mutually-dependent aspects of a larger system, which can co-determine each others' products.

1.3.3 – Chapter 4: Acquisition, Inheritance, and Ontogenetic (Re)Construction

Chapter Four mimics the argument style of Chapter Three, but shifts focus to the acquired/inherited dichotomy. It argues that, despite the Nature/Nurture framework (and neo-Darwinism) relying on a firm distinction between acquisition and inheritance, there simply isn't evidence of such a neat separation of the categories. The first section of the chapter presents evidence that the distinction is blurry at best, and shifts the focus to inheritance systems, as per Jablonka & Lamb's (2005; 2014) use of the term. I discuss the plurality of inheritance system (ie more than genetic inheritance) that we have access to within the developmental niche, and argue that all phenotypic traits are contingently (re)constructed in ontogeny through the developmental niche. To that end, there's no real difference between acquisition and inheritance: both kinds of traits emerge contingently in ontogeny, both can be driven by access to the various extended inheritance systems, etc. The second section of the chapter argues that acquisition and inheritance themselves interact as part of the coupled system: acquired behaviours can drive new inheritances (or themselves become inherited under the right circumstances), and vice-versa. To this end, I explore a couple examples of how this feedback loop can unfold, such as genetic assimilation (where an acquired trait becomes inherited). Again, the emphasis is that there simply isn't evidence for a hard distinction between acquisition and inheritance, but both the Nature/Nurture framework and the neo-Darwinist approach rely heavily on the notion that there is such a hard-and-fast distinction. Instead, on my picture, the phenomena traditionally captured by the Nature/Nurture categories cross over such divisions, co-determine and interact in a variety of ways. But they can function as dependent parts of a larger developmental niche which transcends that traditional divisions of subjects into Nature and Nurture as bounded and opposing categories.

1.3.4 – Chapter 5: Dissolving Nature/Nurture; Objections/Replies

Having spent the previous two chapters undermining the distinction between two sets of dichotomies (gene/environment and acquired/inherited), Chapter Five aims to tie these various threads together. There are five main sections and topics that this chapter addresses. Section One offers a brief summary of the contents of earlier chapters, to

remind the reader of the various threads in play. In Section Two, I elaborate on the metaphors I have been using – irreducible, embedded, etc – and sketch how I take our development to be a matter of co-creation between ourselves and our niches (especially ourselves *in* our niches). The point is that development is a process that combines a network of contributing factors - some are genetic, others are epigenetic, social, ecological, etc. But the point is that even these categories reflect convenient ways of carving up the world. The developmental niche reflects the way these resources are presented to us, and development consists in their interactions and ways they come to be used to the developing organism. Section Three focuses entirely on making the claim that the Nature/Nurture framework is incoherent, and draws on the previous two chapters to argue that the traditional dichotomies of gene/environment and acquired/inherited are better reconceptualized as parts of a larger developmental system that transcends these traditional divides. My argument for incoherence relies on this blurred boundary between the old dichotomies. Specifically, I claim that the process and entities traditionally categorized as either Nature or Nurture often defy these categorizations, and interact in ways that transcend the Nature/Nurture division. This gives us a tool to claim that this division doesn't match the available evidence and should be rejected. But there is more than that. Since the phenomena which are traditionally grouped as either Nature or Nurture can defy these groupings and transcend them, this weakens the ability for the traditional framework to defend the distinction between categories in a meaningful sense. And if there's no real distinction between the things traditionally-grouped as Nature and those grouped as Nurture, claims about the contents of one category can also refer to the contents of the other (opposing) category. A framework which cannot reference its contents without also referencing their opposing counterparts is, I argue, incoherent.

The final two sections of Chapter Five (ie sections Four & Five) examine some consequences of the earlier arguments. For instance, Section Four offers a critique of twin studies, on the basis that the methodology explicitly relies on conservative interactionism, with all of the problems that implies. Twin studies are the focus here because I suspect those who disagree with my picture of development would love to highlight twin studies as examples of how wrong I am, and so it is a topic that I need to address. Section Five finishes by offering a few supplementary arguments regarding the

relationship between Nature and Nurture, and a few first steps in reconceptualizing the relationship between them.

1.3.5 – Chapter 6: Now What? Next Steps & Implications

Finally, in Chapter Six, I am primarily interested in sketching ways forward that take seriously what I have discussed in previous chapters. This work is, by nature of being a sketch, less thorough than what I have presented in other chapters. There are, however, a couple common threads that tie the motley topics together. The first part involves highlighting work that I think has been especially useful, and discussing why these experimental inroads might be promising in a post-Nature/Nurture world. The second part centers around the developmental niche. Namely, if we agree that the developmental niche is a key entity in the way I describe throughout this thesis, then a natural research project is to investigate how different developmental resources come to be presented within the niche. The final topic that I offer some thoughts on is what to make of development that doesn't import the idea of an innate target. An extension of this idea, I argue, is the notion that biology doesn't make “mistakes” in the sense that phenotypes don't miss some predetermined target. I think this notion has immense political and social importance. The important thing, then, is to find other ways of determining what types of developmental settings we want to encourage and discourage, and on what grounds. I offer some preliminary thoughts on the matter, but adequately fleshing out this question would be the work of an entirely separate dissertation.

Chapter 2: Candidate Notions of Interaction

Among other things, the introduction considered a few socially pernicious aspects of the Nature/Nurture framework. Such examples of negative social effects for a given framework may give us some reason to want to reject a framework that gives rise to negative consequences in this way. However, we have good reason to want the rejection of a framework to be accompanied by either: plausible alternatives and/or epistemic reasons as well. My aim in this project is to do both, though my emphasis is decidedly on the epistemic side of the rejection.

This chapter thus gets the ball rolling on the epistemic side of my argument. As mentioned in the introduction, I take the fight around development to be between rival forms of interactionism. If that's the case, then a good first step is to get a bit clearer on what an interaction is. As such, my intention in this chapter is both descriptive and modest. It is descriptive insofar as my aim is to conduct a bit of linguistic anthropology (so to speak) by showcasing a few different kinds of interactions, and briefly discussing what makes them tick in terms of the areas of science where they see use. My aim is modest in that, for most of these areas of science, I am not an expert. It is far too difficult for me to be an expert in social psychology, mathematics, and physics (both quantum and classical). As such, I want to be upfront that I do not intend to be an expert in all of this material. Despite that, however, I think there is still an interesting and manageable project: the term 'interaction' is used in several different areas of science, and it is worth canvassing how disparate all these uses are.

I also want to further examine their usage, and offer a way to analyze some of the differences between kinds of interaction. For instance, I think it is illuminating to look at what remains constant or fixed, compared to what changes as a result of the interaction. Are new entities coming into being? Or does the number of interactants remain constant while their properties (such as mass, velocity, etc) change? Are the interactants empirically separable? Are they conceptually separable? Different kinds of interactions

offer different answers to these questions, and because of that I think these types of questions are useful tools in demarcating kinds of interactions. After all, if there are multiple ways in which the term 'interaction' can be used, then it's worth getting clear on the different types of argumentative lifting that can be done by deploying specific meanings of the term.

The chapter is structured in the following way. Each of the four candidate notions of interaction is addressed in a separate section: 1) causal interaction, 2) interaction as exchange; 3) statistical interaction; and 4) interaction in the sense of coupled equations. For each section, I begin by introducing the candidate notion of interaction. Where possible, I try to provide examples to help demonstrate how the term is used. I will then analyze the interaction in terms of the following criteria: 1) are the components of the interaction empirically separable? 2) are the components conceptually separable? 3) Across the interaction, do any noteworthy elements remain constant, or do they change? Examining the candidates in this manner helps illuminate their differences, and helps illuminate the type of argumentative lifting that each candidate notion can do. While I think that multiple kinds of interaction have a role in describing development, I am primarily interested in what statistical and coupled interactions allow. As I will argue later, these two kinds of interaction are of particular interest because they give a way to formulate well-understood senses of dependence and irreducibility, respectively. Elsewhere in this dissertation (though primarily in Chapter Four), I lean heavily on the coupled equation sense to characterize the developmental system as a single, coupled, irreducible system, where the values of its parts are co-determining and mutually dependent. Characterizing development as irreducible interaction in this way helps get us to a kind of interactionism that is worth having, and gets around some of the problems I have noted with conservative interactionism.

Before diving into the candidate notions, I want to make a few definitional remarks on my analytical criteria of empirical and conceptual separability. For a thing to be empirically separable at all, it must be possible to empirically intervene or test it without also testing its confounds. For example, Lenz et al (2019) conducted a study on conceptual and procedural knowledge of fractions in math (ie 'know that' vs 'know how').

Depending on whether they were testing conceptual or procedural knowledge, the experimenters altered the task that participants were required to perform. For instance, conceptual tests required the subject to vocalize the mathematical meaning or importance of the subjects own actions when working with fractions. By contrast, procedural knowledge merely requires rote algorithmic procedures. Because each of these facets could be probed independently, Lenz et al determined that (under at least some conditions), procedural and conceptual knowledge of fractions are empirically separable, since one can test one category without also testing the other. Under many other test conditions, the two categories are not empirically separable simply because the task is too coarse grained to pick out only the empirical consequences of one variable over the other. And many tasks involved with calculating fractions surely involve both conceptual knowledge about the fractions as well as procedural knowledge about how to find the solution. One can thus make differing claims about whether two constructs are empirically separable in general, compared to whether they are separable in some given task.

As a final note, there is also the matter of conceptual separation. I am treating this matter in the following fashion. For a thing to be conceptually separable, one must be able to identify it separately from another thing. For instance, looking at a red square, the 'redness' and 'squareness' are conceptually separable; I do not need to invoke the square's shape in order to identify the colour of it. However, even in thought I am not able to separate 'squareness' from a polygon that has four internal right angles and whose sides are all equal length. To be the latter is just to be the former. It is perfectly possible to have conceptual separation between two (or more) things that are empirically confounded in messy ways. The relationship between health outcomes and socioeconomic status is one such example. It is perfectly possible to think about health outcomes as a distinct category, but the literature indicates that these two categories are entangled in complex ways. (Also as a bit of foreshadowing, the topic of health outcomes associated with socioeconomic status is addressed more at length in Chapter Four).

2.1 – Four Candidate Notions of Interaction

2.1.1 – Causal Interaction

The first kind of interaction we will consider is generally the most straightforward, and likely the most familiar to many. Namely: interaction as causation. In its simplest form, this has the structure of 'A causes B', though of course there may also be many-to-one relationships as well as one-to-one. A slightly diminished reading of this may also include the idea of some entity 'affecting' another, where this is still understood causally, but the entity in question may not be primarily responsible for the final outcome (ie A affects B), or A might play a causal role in B's outcome without fully determining every aspect of the final outcome.

A classic example of the causal view is the physics of billiards. If I am lining up a shot in billiards – say a simple example of knocking the white ball into a coloured ball - I am trying to interact with the white ball in the right way such that it goes on to interact with the coloured ball in a way such that the coloured ball will reliably be knocked into a pocket. 'Lining up a shot' is merely a shorthand for trying to aim, which is in turn an attempt to maximize the likelihood that the resulting interactions (as a chain of causal events) produce the outcome that I wish. A more full description of the interactions in question could be summarized by the equations for the conservation of linear momentum. Assuming there is no friction between the balls and that the collision is perfectly elastic (to simplify the equation), we can describe the outcome as follows: the shooter imparts kinetic energy via the cue to the white ball A, which begins moving on account of the conserved energy. The direction of its movement and the velocity depend entirely on the direction in which I am imparting the energy, and the amount of energy respectively (ie how hard I hit the ball). The white ball A continues moving along this linear path until it strikes some coloured ball B. When A comes into contact with B, it will transfer a portion of the kinetic energy it is carrying, thereby allowing B to also move in a linear direction. The direction of travel in turn depends on the angle of contact between A and B. This type of interaction – the transfer of kinetic energy – may chain further if the rolling ball comes into contact with other balls on the table, as it would in the opening shot of the game where the player is trying to break the grouped up balls.

Billiards is a paradigmatic, if simple, case of interaction as causation where the nature of the interaction involves the transfer of force – kinetic energy in this case – from one discreet object to another. In this example, the interactants are the billiard balls (at least 2), and the player who imparts the kinetic energy by taking the shot. Each ball is empirically separable from each other, as is the player from the balls. That is to say, we have reason to think that these entities are discreet and non-overlapping (we can pick them up and move them around without changing the properties of the other interactants), and there are experimental conditions where one can manipulate one billiard ball while the other remains unaffected, and likewise for the player. This pattern – where all the entities are discreet and easily seen as empirically separable – is easily reproduced when we think about classic examples of causal chains.

In addition to being empirically separable, the 2+ billiard balls and the player are also conceptually separable. This seems straightforwardly true, in that I am able to identify a plurality of billiard balls within the example (ie recognizing that they are discreet and not identical), and also a (probably) non-ball shaped human.

On to the next question: what remains constant, and what changes? The velocity of the billiard balls will change throughout the extended interaction (ie taking the shot, collision, and final ball placement), with the balls eventually coming to rest somewhere on the table. (Though I suppose if I am a truly terrible player, a ball could conceivably come to rest on the floor). The placement of each ball relative to the other balls on the table will also change, and indeed that is part of the complexity of the game. The posture of the player taking the shot (ie their physical arrangement of limbs) will change as they are taking the shot, and very likely will change afterwards as they either celebrate a successful shot or bemoan a miss. Presumably they will also stand up and circle around to where they expect their next shot to be. However, the balls do not cease being balls as a result of the interaction (assuming there were no major structural flaws in the balls, or that the amount of kinetic energy imparted was not wildly beyond most human capabilities). Indeed, the ability of the game to continue beyond a single turn is largely

contingent on the fact that the form of the balls doesn't really change after the interaction. If that weren't the case, I suspect games would be much shorter.

While billiard balls offer a perfectly straightforward example of causal interaction, not all cases are so easy to understand. Quantum entanglement is one such example that is decidedly more difficult for the average person to wrap their heads around, though for some properties such as position and momentum correlation for quantum particles are not unlike the classic case of billiard balls (Bub 2020).

Quantum entanglement was first put forward as a term by Schrödinger in 1935, expanding on an earlier argument by Einstein, Podolsky & Rosen (EPR). The argument is based on an experiment wherein two particles are prepared in 'pure' quantum state, and then the particles are moved apart. After they are moved apart, there are correlations between their various properties that match (ie momentum or position). Measuring either the momentum or position of one particle will enable the certain prediction of the respective property of the other particle. There is a catch, however. Only one measurement can be done (between momentum or position) since they cannot be done simultaneously. And doing one kind of measurement seems to disable the other correlation of the particles on other properties. As Bub (2020) puts it:

> *"The subsequent measurement of momentum, say, after establishing a position correlation, will no longer yield any correlation in the momenta of the two particles. It is as if the position measurement disturbs the correlation between the momentum values, and conversely. Apart from this peculiarity that either correlation can be observed, but not both for the same pair of quantum particles, the position and momentum correlations for the quantum particles are exactly like the classical correlations between two billiard balls after a collision."*

Schrödinger (1935) uses an analogy to describe the strange outcome:

> *"Yet since I can predict* either *x_1 or p_1 without interfering with the system No. 1 and since system No. 1, like a scholar in an examination, cannot possibly know which of the two questions I am going to ask first: it so seems that our scholar is prepared to give the right answer to the* first *question he is asked,* anyhow. *Therefore he must know both answers; which is an amazing knowledge; quite irrespective of the fact that after having given his first answer our scholar is invariably so disconcerted or tired out, that all the following answers are 'wrong.'"*

For Schrödinger, the point is that the two systems seem to enter into a common state on account of their (temporary) physical interaction, and we can know this because of the correlated values for a given property. But after the systems separate, they cannot be described in the same way. Measuring one system seems to affect the second, because the systems are entangled on account of their earlier interaction. This leads, for Schrödinger, to the strange conclusion that the best knowledge of the whole might be distinct from best knowledge of the parts. The systems might be entirely separate, and we might very well understand the interaction that takes place, but the nature of the interaction seems to entangle the two systems such that we do not understand the whole by virtue of measuring the parts (see Schrödinger 1935; Bub 2020).

I do not want to go too off-topic in bringing up quantum entanglement, but it offers a rather interesting twist to the standard causal story. At the very least, it certainly shakes up the analysis I presented above for the billiards case. Here, in the post-interaction state, the quantum state has changed, and the systems no longer seem empirically separable to me. By intervening in one system, the other changes too – much like how posing a question to Schrödinger's academic thus makes them forget the answers to the rest of the questions that they otherwise would have gotten correct. Given that we can identify there are multiple systems that are entangled, however, there still seems a conceptual separation to me.

My point is not that quantum entanglement totally forces us to rethink standard causal interaction. I am not qualified to make that argument, nor is it entirely necessary

for my larger project. My point in raising quantum entanglement is to gesture at some potential complications for what is often thought is a very straightforward kind of interaction. I think that, most of the time, causal interactions are assumed to be like the billiards case*:* discreet, separate entities interacting in such a way that some force or effect is applied from one entity to another, but otherwise the form and general properties of the interactants remain stable. And that might well work most of the time. But it's worth remembering that there are other, more complicated ways that things might causally interact.

2.1.2 – Interaction as Exchange

The second kind of interaction that I want to discuss is the sort where it consists in some kind of exchange. This usage is likely less common than simple causal interaction, but nevertheless it is used in more than one area of science. The examples I have chosen come from quantum mechanics & chemistry (both use the same model), and psychology, and will be addressed in the order so listed.

2.1.2.1 – Exchange Interaction in Quantum Mechanics

As with the earlier foray into quantum mechanics, I will endeavour to keep this overview as simple as possible, since it is not the easiest topic to simply drop into without a lot of background. To begin, in quantum mechanics and chemistry the Exchange Interaction is part of electromagnetism, and is primarily part of ferromagnetism (Griffiths 2005). Put most simply, this kind of interaction is an effect that takes place between identical particles, which are particles that are indistinguishable from each other even in principle (Griffiths 2005). (I'll describe the effect in greater detail briefly).

In quantum theory, particles don't have definite positions in between times they are measured. As such, rather than tracking definite positions, a probabilistic approach is used. Wave functions - complex probability amplitudes – govern the probability of finding a particle at each location. However, over time the wave functions for particles

tend to spread out and overlap. Once this has occurred it becomes impossible to determine in subsequent measurements which particle position is connected with the one that was previously measured (Griffiths 2005). Since this cannot be determined, the particles are indistinguishable, and treated as identical.

According to Griffiths (2005), when the wave functions of indistinguishable particles overlap, the exchange interaction results in the change of the expectation value of the distance between identical particles, depending on what kind of particles they are. For our purposes, there are two kinds of particles worth noting: fermions and bosons. Fermions are particles that have half-integer spin, such as electrons, quarks, neutrinos, neutrons and protons. Bosons, by contrast, have integer spin, such as photons. When subject to the effect of an exchange interaction – the exchange of both spin and spatial coordinates as wave functions overlap– the expected value of the distance between identical bosons is decreased, and increased for fermions. In layman's terms, one can see how this interaction is related to magnetism, given it seems bosons come closer together as if they were magnetically attracted, whereas fermions act as if repelled.

That's all well and good, but how does this kind of interaction fit into the sort of analysis I've been giving? To start, the interaction in this case is electrical in nature, where the exchange of charge and location produces the effects of repulsion or attraction. The particles involved are clearly conceptually separable, as we recognize that there are indeed multiple particles involved such that this effect arises. They are (in principle, at least) empirically separable as well. Theoretically the particles are separate, but empirically confounded to an extent by being identical and indistinguishable. The types of particles involved remain constant throughout the interaction, but their location and other properties may change. The specificity of this kind of interaction may mean that it does not generalize to other domains well, or that generalizing it requires abstracting away from specific properties. That may limit its use more broadly. However, there is a second form of exchange interaction used in science: social exchange theory. That will be the next topic to analyze, before offering some concluding remarks on the general category of interaction as exchange.

2.1.2.2 – Social Exchange Theory

The second kind of interaction as exchange that I want to discuss can be found in psychology, namely Social Exchange Theory (SET henceforth). As the name implies, it is primarily focused on characterizing social interactions. To do this job, it primarily focuses on social and/or economic goods that might be exchanged as a function of the social interaction. This can take several forms across different sub-topics, and so I will briefly touch on a few of these different applications.

SET is often used as a game-theoretic type approach, looking at conditions under which people are more likely to engage in some sort of relationship based on perceived rewards or costs. For instance, Homans (1961) – one of the founders of an approach to SET – put forward three propositions to characterize the game-theoretic aspect: (1) 'Success': if one finds they are rewarded for some action, they tend to repeat it; (2) 'Stimulus': the more often a stimulus has resulted in a reward in the past, the more likely a person will respond to it; (3) 'Deprivation-satiation': the more often in the recent past a person has received a particular reward, the less valuable further units of that reward become to them.

This Homans-esque approach still sees use in some contemporary literature. For example, when looking at Parkinson's patient referrals to non-terminal palliative care, Prizer et al (2017) characterized the matter thus:

> *"Social Exchange Theory (SET) [as] a value expectancy theory, involving a balance between individual expectations regarding the outcomes of performing an action as well as their subjective values or rewards about those outcomes. SET proposes that individuals are motivated to interact with another person in a specific activity if they expect a positive outcome from the interchange. If the benefits of the outcome are more rewarding than the cost of the activity, the relationship will be valued and the individual will be more likely to engage in the collaborative behaviour again in the future. With regard*

> *to the current project, physicians may be expected to refer patients to palliative care if they believe the specific rewards from the referral will outweigh any costs." (Prizer et al, 2017, 862).*

Given the economic-style approach, it is unsurprising that SET has also been applied within business and management contexts. As Chen & Choi (2005, 2) note, "social exchange theory has also been applied in the management and organization literature. For example, within organization and management theory, social exchange theory has been mainly applied to examine exchanges between superiors and subordinates (Marcus and House, 1973) or within work groups (Molm et al., 1999)."

Anthropological and sociological approaches to SET have instead focused on the general systems of exchange. Summarizing this shift in focus, Chen and Choi (2005, 3) note that:

> *"Within sociology and anthropology, one of the key contributions of social exchange theory has been to explain the origin of different types of exchange found in different societies. Unlike social contracts theory, which assumes rational choice in the acceptance of authority and in the origin of hypernorms, social exchange theory makes no such assumptions. Drawing from its sociological and anthropological studies in the field, social exchange theorists explain the origin of exchange structures in terms of social factors such as power and status. Unlike social contracts theorists who assume universal norms that lie at the base of social contracts, social exchange theorists focus instead on explaining the variations in exchange structures between societies."*

This focus on exchange as a feature or function of social status also provides some interesting insights. Citing Mauss (1967), Chen & Choi note that:

> *"Drawing on the example of gift giving and receiving in Maori society, Mauss demonstrated that in 'primitive' societies, exchange of gifts does not involve a simple exchange of goods, products and wealth between individuals but rather exchange between collectivities that were*

> *mutually obliged to exchange between themselves. Mauss argued that these obligations – (i) the obligation to give; (ii) the obligation to receive; and (iii) the obligation to return – formed the basis for the structure of society. Moreover, not only goods are exchanged, but more importantly ceremonies, rites, women, children, gestures of respect, etc. Many gifts thus serve a social function such as creating or confirming status within the society."*

Something I find quite interesting about the inclusion of status within the calculus of social exchange is that it opens up the possibility of transformative exchange, whereby a new social status or position can emerge from the interaction. By this I mean that the individual, by virtue of participating in the exchange in the right way, may emerge a new kind of social entity. For example, one could describe coming of age ceremonies, marriages, or position acceptance ceremonies as socially transformative in this way. In each of the above examples, the new social or legal status (generally) emerges from a context that is contingent on both what sort of interactants are present, as well as how they interact.

For instance, in many societies there are restrictions about which types of people have the legal power to perform marriages. There are often also requirements for a marriage witness, etc, in order for the marriage to be official in a legal framework. But by virtue of the individuals coming together in the right way for a given ceremony, someone will be granted a new social status. Of course, this can also alter the individual's relations, responsibilities and privileges, depending on the previous and current social positions of the individual(s). For example, coming of age ceremonies tend to bestow a position of adulthood on the individual, which may unlock new options, powers, and responsibilities that they did not have access to previously. These may range from now being expected to provide for the group, pay taxes, take a lead in social rituals, etc.

Now clearly, the kinds of exchange interactions I have sampled from quantum mechanics and psychology are quite different. In quantum mechanics, the interaction is between identical particles and how they act (probabilistically speaking) as if repelled or

attracted to one another under certain conditions. The interaction is thus the relation between the particles and how it changes. In psychology (at least under social exchange theory), social interactions are exchanges of social goods, which prompt evaluation of the social goods being exchanged, and a calculation of whether the relationship is worth keeping in light of the value one receives from it. An interaction in this case is the context in which social goods are given and received, and something is a social interaction if multiple entities are in some way exchanging goods that have a value. There is, however, a slight abstract analogy between the more emergent forms of social interaction and the quantum mechanics example. In both of those cases, the interaction is characterized by the relationship between interactants within the broader context. The properties of the interactants (spin, social position, etc) influence the form that the interaction takes and the range of outcomes. Thus, interaction as exchange can be broadly understood as a relationship between multiple entities, where the nature of the relationship and the potential outcomes are constrained by the properties of the interactants themselves.

How, then, does social exchange theory fit into the analysis framework I have been using? In general, since the emphasis is on the relationship between multiple entities, the entities must be conceptually distinct for this characterization to make sense. There will also be at least some contexts where the interactants are also empirically separable (ie one can change the price of items in a vending machine when it is not being presently used, and making this change need not have an implied change for the individuals who routinely use the vending machine). However, in many contexts, a change in price may prompt the individuals to re-evaluate their vending shopping, so they are not empirically separable in every context. Similarly. analysis of what changes and what stays the same will be dependent on context. The price of some good could change, behaviour could change, social status, legal status – all of these properties could theoretically change or stay the same depending on what kind of exchange is taking place. However, given that an exchange is taking place, something needs to change insofar as it is being exchanged. The set of interactants and the social context in which they are interacting will constrain which properties are stable and which change.

2.1.3 – Statistical Interaction

Two kinds of interaction down, two to go. The third kind of interaction that I wanted to discuss has to do with statistics. However, even within statistics there are a few different kinds of interaction, and I think only a subset of those are interesting in a way that merits talking about them as a different kind of interaction. As such, I begin with a few comments on the first kind of statistical interaction, which I take to be mostly an extension/ more generalized version of interaction as causation. Because I have mostly covered this kind of interaction already, I am uninterested in it here, and my comments are offered mostly for the sake of completeness. I then turn to describe the kind of statistical interaction that I think is worth focusing on. Specifically, the kind of interaction I am interested in is when multiple independent variables interact, such that they mediate each others outcomes. This, I will argue, gives us a well-understood notion of dependence, which will be important for the later stages of this project.

How, then, is the term 'interaction' used in statistics? There are at least two distinct ways the term is used. I will begin by describing a usage that I am not interested in, and offer a brief explanation as to why. The first and most obvious usage crops up when multiple variables are being compared in some way, where there is at least one independent and one dependent variable. The most common tools used for this are ANOVA (Analysis Of Variance), ANCOVA (Analysis of Covariance), and multivariate analysis techniques (whether regression, MANOVA, or MANCOVA). In general, these types of techniques aim to build a model using the data you provide, and then make predictions based on it. Part of the data (ideally a large part) will be accounted for by the model, and some will not be (ie the 'error'). For instance, you might take data about ice cream sales in a given town, and attempt to predict what future sales will be given the weather forecast (since you can look up the old forecasts for the days in your data set). If there is enough data, you might be able to build a statistical model that makes some good predictions. But presumably your model will also get it wrong at least some of the time, depending on the nature of the relationship between the variables and possible interference of third-party factors. In general, I think these types of models are best described as aiming at tracking some kind of causal interaction. In the above example,

presumably there is some sort of causal relationship between weather, temperature, and ice cream sales. The math is just generalizing some version of the causal interaction. Since I have already discussed causal interaction in an earlier section, I am not interested in going over it again, and this is why I do not think this sense of statistical interaction is offering something new to the discussion.

If not generalized causation, what other kinds of interaction are available? Well, in addition to the ways in which independent and dependent variables can interact, there is also the possibility of independent variables interacting with each other as a two-way interaction effect. In the latter sort of cases, the effect of one variable depends on the status of some other variable (Dodge 2003; Cox 1984). There are many examples of this. For instance, there is a relationship between cancer risk from smoking, and also cancer risk from inhaling asbestos (Lee, 2001). Both raise the risk of cancer independently, but there is some research that indicates the risk is multiplied for smokers who also are exposed to asbestos. The effects are not merely additive; the cancer-causing power of the smoking variable also depends on the 'value' of the asbestos exposure variable. The two variables thus interact to produce an outcome greater than the mere sum of its parts, which means that there is a dependence interaction between the two. A second example of dependence interaction can be seen in the study of individuals learning French in different locations (Godden & Baddeley 1975). There were two learning conditions: one was learning while scuba diving, the other was standard classroom learning. After a period of learning the language, students were then tested, and the tests took place both underwater and in class. Researchers expected the main effect to be that learning while scuba diving was simply inferior, as it is a strange learning environment. But that's not what was found. Instead, they found that test performance depended on whether the individual was tested in the same location they learned. So scuba-learners who were scuba-tested scored highly, whereas scuba-learners who were classroom-tested scored poorly. The two independent variables interact such that the effect of one depends on the value of another, despite both being independent variables. In such cases, asking for the value of one variable will not get you very far unless you also know the value of the second variable. In this sense, the effect of some variable (learning location) on the main effect being studied (learning outcomes) depends on the value of some other independent

variable. The effect is thus an emergent pattern between two independent variables based on how their values mutually influence each other.

Thus, rather than be a mere generalization of a causal process, the kind of statistical interaction that I am interested in is the degree to which variables influence one another. That is to say, how the value of one variable influences or affects the value of at least one other (and in doing so, exhibits a dependence relationship). However, beyond highlighting the relationship between independent variables, this kind of interaction need not commit in advance to how the variable relate to each other. By this I simply mean that, barring other experimental results or findings in other fields, we will only have a mathematical estimate of how the change in value of one variable will affect another, and in a given model we may try to analyze this and figure out how much variance of the data is accounted for by the relationship between these two variables. There is also the possibility of mediation effects or mediation models, where a mediation effect is when there is some sort of effect chain, such that Variable 1 affects Variable 2, which affects Variable 3. In this chain, Variable 2 is the mediator between Variables 1 (the predictor) and 3 (the outcome/response/ dependent variable). A mediation effect is thus any effect that lies between two variables of interest and whose value is affected by the predictor variable, and can change the value of the response.

With all that said, let us further analyze this dependence interaction with the same lens that we have applied previously. In the sorts of cases I am interested in, there are multiple IVs, and the effect of one IV depends on the presence and/or value of the other IV, such that knowing the value of one IV is insufficient to determine its effect (at least in the context of the interaction). While the interactants may be empirically separable in at least some contexts (ie one can study cancer risk associated with smoking without such efforts affecting asbestos), they are much less likely to be empirically separable within whatever contexts they are interacting. While this may seem a simple point, I think it is worth emphasizing. It will depend on the way in which the effects of the variables are dependent on each other, but given this dependence relation there are probably more sensitivities to empirical interventions on one variable or another. While it might be generally the case that each variable can be wiggled independently of each other, there

seems to be something special about the context in which they are interacting since the variables effects then have this dependence relation that they otherwise would not have. Depending on the nature of that relation in that context, it might not be possible to wiggle one variable without also affecting its counterpart. It also strikes me that conceptual separability is a much more interesting topic in this discussion. In general, the interactants will be conceptually separable – we recognize that cancer risk, smoking and asbestos are all distinct things. In the context of the interaction, however, I am not sure matters are quite so clear. Given that the values of the IVs can mutually influence each other, the 'value' of each IV is neither empirically nor conceptually separable from its counterpart. The presence (and/or value) of the second variable in part determines the value of the first variable; they are dependent on each other, and so are much more difficult to separate. This type of interaction is one I want to emphasize for the framework I am building. It shows how interaction can result in a downstream emergent property, contingent on the values of its interactants. This gives us a sense of 'pure dependence', since we have multiple variables whose effects are jointly determined with other variables. The benefit of this is that this version of emergence-interaction is well understood and statistically well defined. This gives a useful tool for helping measure and define more complex and robust interactions, which is one of the end goals of this project. In the context of my larger project, I want to lean on the ways that interactions of this sort can have such intimate linkage of parts, such that multiple parts of a larger system can be dependent on each other. Before turning to the larger project, there is one final kind of interaction that I think is important to discuss: interaction as coupled equations. So that is the next port of call.

2.1.4 – Coupled Interaction

And so we come to the final section of this first chapter: the fourth of four kinds of interaction. For practical purposes, I'll start by giving a brief description of what nonlinear coupled equations are, before turning to analyze how this kind of interaction is of critical importance for my overall project.

Nonlinear coupled equations are equations (or sets of equations) whose variables do not have values that are separable from the other variables in the equation. Thus, these equations need to be solved at the same time, rather than follow standard simplifying procedures such as solving for one equation at a time. If the variables cannot be isolated (as is the case often enough), this implies that both (or however many are coupled) equations contain information that is necessary for the solution to be found. And since the variables cannot be isolated to find a solution, this means that in some important way, the system comprised by these equations cannot be decomposed or reduced without also fundamentally changing them.

Given my emphasis on the importance of mutual dependence in the section on statistical interactions, it is fairly easy to see where I want to take this discussion. If the coupled systems cannot be decoupled (and thus they mutually influence each other's variables), then this gives us a useful sense in which some system is irreducible. This sense of a system being irreducible can then be extrapolated to suitably analogous alternative domains. In subsequent chapters I attempt to do exactly that, and characterize the gene/environment dichotomy as a larger, coupled, developmental system. How, then, can nonlinear coupled equations be applied in the way that I intend?

I am not the first to try and use coupled equations in this way. For instance, Chemero offers a clear explication of all this in his book *Radical Embodied Cognitive Science* (2009), and so I will lean on that to help explicate matters. For Chemero, coupled systems are an important part of radical embodied cognitive science and dynamical systems theory. For instance, Chemero notes:

> *"In radical embodied cognitive science, the explanation of cognition is dynamical, and (wide) computationalism is explicitly rejected. Agents and environments are modeled as nonlinearly coupled dynamical systems. Because the agent and environment are nonlinearly coupled, they form a unified, nondecomposable system, which is to say that they form a system whose behavior cannot be modeled, even approximately, as a set of separate parts." (Chemero 2009, 31)*

In addition, Chemero highlights what I think is the most interesting application of coupled interaction, given how the parts are so mutually bound up with one another:

> *"It is only for convenience (and from habit) that we think of the organism and environment as separate; in fact, they are best thought of as forming just one nondecomposable system, U. Rather than describing the way external (and internal) factors cause changes in the organism's behavior, such a model would explain the way U, the system as a whole, unfolds over time." (2009, 25-26)*

Just as Chemero thinks that the agent/environment distinction is better captured by one, nondecomposable, system U, my intention is to argue that the components traditionally treated as separate by the Nature/Nurture framework are not separable, and instead comprise a single, nondecomposable developmental system D.

Now, I want to be clear. I have no explicit intention of making radical embodied cognitive science part of this project. That would be the work of an entirely separate dissertation. However, the way in which Chemero discusses agent/environment coupled systems is quite useful for the sort of work I want to be doing in my own project, and his discussion helps situate the types of literature I am drawing on to be able to employ a notion of coupled interaction. Astute readers should keep in mind Chemero's quip about how it is only for convenience and habit that we tend to conceive of organism and environment as separate, when they are better interpreted as parts of a larger system. This sort of move is exactly what I have in mind, though I'm working in a slightly different domain from Chemero.

What sort of argumentative work do coupled interactions offer me? First, this kind of interaction emphasizes the connections between entities within a system and how they cannot be broken down. Given my end goal is to use the metaphor of irreducible interaction to make sense of development, Chemero's approach to coupled systems puts forward a well-understood sense of irreducibility. In more practical terms, one needs to know the values of both variables in order to understand the meaning of their coupling. In

subsequent chapters, I will discuss some examples of how I think this works. I take this kind of interaction to be very important for understanding development well, as it helps dissolve the gene/environment dichotomy in favour of a larger system.

Finally, let us apply the standard analytical framework that I have been using for the other kinds of interaction thus far. Like with the exchange and dependence interactions, coupling involves multiple entities or variables standing in a particular relation to one another. In this case, that relation is co-constitutive, as each variable's value is bound up in the value of at least one other. Thus, by definition the coupled relationship is not empirically separable because of the way the variables are connected. Nor, I would argue, are they conceptually separable in a meaningful sense. This, I think, was part of Chemero's point when he remarked that one can by force of habit separate and distinguish the parts of a coupled system, but really the right way to think about matters is to recognize the larger whole. In such cases, the 'meaning' of one variable cannot be determined just by looking at that variable. Not when it stands in such a co-constitutive relation with some other variable, whose 'meaning' likewise depends on the variable in question. There may be heuristic ways to separate the parts of the system in thought, but we are better served when we recognize that these relations are irreducible, and thus not conceptually separable.

2.2 – Wrapping Up

We've covered a fair bit of ground in this chapter, so I want to take a minute to summarize and make the connections clear. We have discussed four different kinds of interaction: causal, exchange, statistical dependence, and coupled systems. Each of these distinct kinds of interaction sees uses in largely different areas of science, ranging from quantum mechanics, to economics, to cognitive science. Consequently, each kind of interaction does slightly different work in terms of describing how the various interactants come together and stand in some sort of relation to each other. This also means that, for the most part, these distinct kinds of interaction behaved slightly differently when subjected to the simple analysis that I have been using. For instance, with simple causal interaction, the parts are generally all empirically and conceptually

separable, but the same cannot be said for coupled systems, where the values of its variables are co-constituted.

In addition to describing some candidate notions of interaction, I have also alluded to the sort of work that I think they can contribute to my project. For present purposes, this means I again want to emphasize the usefulness of dependent and coupled interactions, and the reader can expect that I will be alluding to these throughout the project. Statistical dependence, for example, gives us a well-understood way to discuss how a range of outcomes can be modulated by other variables. In short, it's a non-mysterious way of explicating the notion of dependence. Similarly, coupled systems offers a clear way to describe the way in which something is irreducible (or nondecomposable, to use Chemero's term). For my purposes, I have seen attempts in the postgenomic literature to describe development as emergent, but many of such attempts may not have the right language to appeal outside the literature. Coupled systems offers a way to potentially bridge this gap, by putting forward a well-understood sense of irreducibility.

There is a pragmatic element worth highlighting here. How we talk about, and model, development is subject to several considerations. For instance, one could choose simplistic terminology and abstract away from the details in order to place emphasis on particular relationships between elements (i.e. favour simplicity as an epistemic virtue) or one could favour breadth, depth of explanation, or something else entirely. Different research approaches might favour different epistemic virtues and strategies, leading to a difference in how some phenomenon is modelled and used to generate knowledge. This is a pragmatic element of modelling choice; sometimes concepts or strategies might be chosen based on what they can do for an approach, given specific interests or goals that constrain the possibility space.

Development, especially in the broad way I use the term, is sufficiently broad that it is not always easy to experiment on a phenomenon in a holistic way. As such, different modelling choices may be made in order to make the problem more tractable for inquiry, and in so doing, such models may prefer to foreground simple causal or statistical

interaction, instead of coupled interaction. Indeed, by focusing on different kinds of interaction, it might be the case that different results are obtained. I am not trying to block this possibility, which is part and parcel of how science approaches a complex problem. Indeed, though I have reasons to favour focusing on coupled interaction, I am not demanding that everyone must only ever think in terms of coupled interaction. I recognize that complex problems can be broken down in various ways; even parts of a coupled system could theoretically be treated as separate as a simplifying assumption for further inquiry if so desired. There is a substantial literature on how even strictly false models (i.e. idealized or abstracted in some sense) can be productive in advancing our knowledge on various matters (i.e. Bokulich 2011/ 2012; Weisburg 2007; Wimsatt 1987). So why am I focusing so strictly on coupled interaction, and beating that drum so readily? Primarily because of the focus I have chosen, which is partly theoretical and partly practical. The positions that I favour in philosophy of biology, such as developmental systems theory, tends to view development in a very holistic way, such that there is relatively little difference between the organism and the environment. Such a background helps explain why I might go searching for something like coupled interaction, and choose to favour it in how I represent and model development and developmental phenomena. This is the theoretical element of my response. The practical part, however, is that a central focus of this project lies in finding a way of getting out from under the shadow of Nature/Nurture; to move beyond the standard 'two origin' story. There are different tactics one could employ in the face of this problem, but my focus is on denying that the standard distinction between phenomena typically construed as Nature or Nurture is meaningful. In so-doing, there is a push towards a more holistic view of development, and as I've already alluded to, that's not a coincidence given the sorts of positions I tend to endorse. One is free, of course, to think of development in other ways. But given the worry that I mentioned at the start of this project – that we keep finding ways to recreate some problematic aspects of the Nature/Nurture framework – my goal is to find a way forward, around this recurring pattern of Nature/Nurture. I think the view of development I espouse in this project is less prone to recreate these issues, though by virtue of emphasizing holism it may come with other tradeoffs. I invite productive inquiry around development from a multitude of angles. However, I tend to think that the price of such

pluralism is that we ought to pay close attention to whether or nor some approach contributes to some tacit recreation of Nature/Nurture – ie 'the price of metaphor is eternal vigilance'.

As I mentioned in the introductory chapter, I take the big battlegrounds in developmental science to be between different kinds of interactionists, since most people tend to agree that interactionism is a desirable school to endorse. So if I'm going to be rejecting one form of interactionism (which I have called 'conservative interactionism'), then another form must take its place. I take most of conservative interactionism to be telling a causal story, where gene and environment respectively offer distinct causal forces that additively determine the phenotype. I'd be willing to bet that some people also had something like statistical dependence in mind, where different variables (genetic history, environment, etc) mutually modulate development. What I am proposing instead is that the gene/environment system is better thought of as one larger, coupled system. The details of that argument will be fleshed out in subsequent chapters, but for the reader, the general thread goes in the following fashion. Coupled systems are an appropriate type of interaction to describe development. To make that case, I describe how we can understand the genome as extending beyond the body in important ways. Afterwards, I describe how there is no real difference between acquisition and inheritance, since both occur in the same way, which in turn is part of the ontogenetic niche – this organism-environment system where we are both (partial) product and (partial) creator of the world around us. The emphasis in these chapters is on destabilizing old dichotomies, and showing that there really isn't much difference between the parts. From there, I argue that since all this behaves like a coupled system, the Nature/Nurture dichotomy is incoherent since its components are best understood as mutually-dependent and co-determining. The developmental system is irreducible precisely because its parts are co-determinining. So it is not a small thing to say that interaction as coupled equations is foundational for this project. But one thing at a time. The next step is to apply the work I have already done to the genetic context, and argue for the Extended Genome Thesis.

Chapter 3: Genetics and the Extended Genome

In the last chapter I discussed four different kinds of interactions that can be found across various areas of science. I think each kind of interaction will likely have some part to play in development, but for present purposes there are two that I wish to focus on: statistical and coupled systems interactions. In the characterization of development that I am moving towards, statistical interaction offers a well-understood way to characterize dependence, and coupled interactions offer a way to understood claims of irreducibility and emergence. These themes will crop up again and again throughout these chapters, so it is worth keeping in mind that I am explicitly thinking of interaction in these terms.

In the next couple chapters, I apply the discussion on interactionism to topics in development and biology. This chapter is concerned with genetic interactions; the following chapter looks at inheritance and acquisition.

This chapter is comprised of two main sections, with both sections looking at different views of interactionism in genetics. The first section offers the received view regarding genetic interaction, and the second section looks at cases that are hard to capture on the received view (but easier to capture on my view). The upshot, I argue, is that the evidence seems to warrant a much more radical notion of interaction than the received view offers or allows for. It is worth noting that my conclusions in this chapter will become a premise for the overarching argument that I make later in Chapter Five. The forthcoming structure works in the following way: in this chapter, I argue that genes are not simple causal agents in the way they are often talked about, and instead the genome seems to behave reactively (rather than entirely proactively as the received view suggests). If the genome is reactive in this way, and it can be shown that it is reactive to the environment (as I claim in this chapter), then we begin to have reason to treat the gene/environment dichotomy as a larger coupled system. Among other things, in Chapter Four I support the claim that not only does the genome react to the environment, but the environment is changed by the interaction as well, and this gives us more reason to

reconsider the gene/environment dichotomy as a single, coupled, developmental system. I say all this to help place the present chapter in the right context and give a sense of how present arguments will be integrated into the larger project. To reiterate: Section 1 of the current chapter will examine the received view, and Section 2 will argue for the shortcomings of the received view. Those shortcomings will form the basis of my argumentative push towards adopting a more radical understanding of interactionism in genetics.

3.1 – The Received View; 'Genetics as Imagined'

To adequately characterize the received view, I want to approach it from a few different angles and analyze each in turn. First, I appeal to the characterization found in the postgenomic literature, which focuses on where the received view thinks information is 'housed'. There is a great deal to unpack there. Following this, I appeal to Dawkins' metaphor of a recipe as away of characterizing the themes exhibited through the discussions on information and the HGP. This metaphor of a recipe is something I will appeal to multiple times throughout this project, because it offers a quick way to understand many gene-centric aspects of the received view. In light of this, I draw on Barker's characterization of positions within biology, and make use of her term 'conservative interactionism' as a name for the received view. Finally, I want to look at the influence of the Human Genome Project (HGP) on the received view. Specifically, prior to the HGP there was a sense of great achievement; a sense that we would get to map the fundamental building blocks of an organism. After the HGP, however, there was more a sense of humility as our frameworks had been shown to be inadequate; our understanding of the role of DNA to be flawed.

According to the postgenomic literature, the received view treats information as being housed in either genes or the environment. This leads to two main themes: information is in genes or the environment (ie nature or nurture), meaning they are separate categories since each contains its own information. Additionally, development just is the addition of the relative informational contributions of each category (Stotz 2008). For example, if we said that an organism had genetic information that specified phenotype A, and was in an environment that pushes toward outcome B, the organism's

development would just consist in the addition of A+B (the genetically-specified phenotype modified by environmental influences). As Stotz (2008) remarks:

> *"Today's received view of development attempts to reconcile both [the preformationist and the emergent] visions: a (multicellular) organism begins as one cell packed with 'innate' information of how to build the phenotype, from which the final form emerges in interaction with the 'acquired' influences from the environment." (Clarification added).*

A number of important themes follow from this picture. First, genes and environment are taken to be separate categories, each with their own information or potential influence. Second, though a few steps would need to be filled in, this is clearly a gene-centric approach, since the information for the fully-specified phenotype is innately in the cell since its earliest existence. The mere ascription of innate information need not entail that it is gene-centric if something else were to house the innate information, but anyone familiar with biology knows the central importance that DNA and genes often play in its discourse. DNA, and genes by extension, are doing the work of the innate information in this picture, since cells inherit copies of DNA from their parent cells, and thus DNA is also present in the cell from the beginning of its existence. Third, if genes contain the innate information for the phenotype, then the nature of the organism is characterized by its DNA. The environment cannot be part of this nature in the same way, since the environment is not part of the DNA of the organism and only serves to modify the phenotypic outcomes. This picture enables further characterization of the parts of development. Internal causes (DNA, inheritance, etc) constitute the nature of the organism through a kind of teleology (by virtue of the innate information in the DNA of the organism). By contrast, external causes (the environment) represent the change of an organism, its variance, and the modification of its nature. This kind of picture clearly relies on notions of nature and nurture; genes and DNA (internal causes and information) are part of a thing's nature, whereas various environmental influences constitute its nurture. Thus, the nature is internal, causal, stable and genetically inheritable, whereas the nurture is external, modifying, plastic and variable (Stotz 2008; Kitcher 2001). The view is interactionist insofar as it takes development to be the addition (a kind of

interaction) of a thing's Nature with how it is Nurtured. The positing of the interaction is the attempt to square the preformationist and the emergentist views.

Other scholars, such as Jablonka & Lamb (2014), take the received view to be an amalgam of molecular neo-Darwinism and Dawkins' (1976) notion of the selfish gene. DNA replication is the vehicle of hereditary transmission, and the DNA sequence is the unit of variation. Random DNA changes are the origin of variation, targets of selection include the gene, the individual and the group, and the unit of evolution is either the population of alleles in the gene (Dawkins) or the population of individuals (neo-Darwinism).

3.1.1 – Dawkins' Cake Metaphor

This builds on the brief characterization I offered above, and helps establish why the view is so gene-centric. As a metaphor, Dawkins used the idea of selfish genes to do (at least) two things. First, the idea of the selfish gene ensures that attention is paid to genes (and competition between genes) as a sufficiently stable unit for evolution to act on. Second, it establishes that there is a distinction between the gene and body, where the body is just a carrier or vehicle for the gene to be replicated. Some things that might improve the gene's chances of increased representation in the next generation may not always be purely beneficial to the vehicle. Dawkins uses this gene-centred focus to classify types of things: 1) replicators – any kind of entities which are copied (Dawkins 1982), compared to 2) vehicles - "any unit, discrete enough to seem worth naming, which houses a collection of replicators and which works as a unit for the preservation and propagation of those replicators." (1982, 114) This approach is thus gene-centred because it treats genes as the stable entities which are copied between generations. Changes to the vehicle (ie gaining muscle, or a scar) are not changes to the gene, and so are not replicated. This distinction between replicator and vehicle is well-represented by Dawkins' metaphor of a cake recipe. On this metaphor, the cake recipe (a gene-analogue) contains all the instructions to specify the final cake product. Copying the recipe would allow a highly similar cake to be made elsewhere. It's possible to make a mistake in copying the recipe – perhaps changing the amount of a given ingredient – and this functions as a mutation to the recipe. Most fundamentally, the 'developmental conditions'

of a given cake – oven temperature, cook time, etc – can affect the cake phenotype but never change the cake recipe. For example, leaving a cake too long in the oven will burn that cake, but will not change the recipe that specifies the instructions on how to make the cake. This means that subsequent generations of cakes will not inherit the burns of that particular burned cake, but instead will inherit the other properties of the cake specified by the recipe (taste, colour, etc). The recipe is thus also teleological: it aims at bringing about a particular end product. Copying fidelity and inheritance mechanisms generally ensure that subsequent copies of the recipe aim at a highly similar end product. There will be some predictable variance in the final outcome of various cakes, given different oven temperatures, cook times, etc. But notice how faithfully this metaphor recreates the trappings of the Nature/Nurture framework: the stability of the recipe across generations of cakes and the teleology of a recipe explains the similarity of cakes made using that recipe. Environmental conditions can modify the outcome, but are not part of the instructions, the nature, or the inherited aspect of the cake. The cake itself can be described as the addition of the specifications of the recipe plus the idiosyncrasies of the context in which the cake is prepared - an additive interaction.

Thinking about gene/environment interactions in this additive fashion is, I submit, the norm in many ways. Indeed, it is taken to be so normal that other discussions of interactionism in biology can face an uphill battle to be taken seriously (Oyama 2000a/b; Jablonka & Lamb 2014), since 'everyone is an interactionist these days'. It's true that everyone is an interactionist nowadays; the ubiquity of the Nature+Nurture additive understanding of development certainly supports this claim. As I will argue moving forward, however, this kind of interactionism is kind of cheap. It recapitulates the problems of the Nature/Nurture framework and does little to solve them. Instead, it glosses over the issues with the language of interactionism (Oyama 2000b). It is worth giving a name to this kind of broad position (other than 'the received view'), and I like Barker's (2015) characterization of the position as a kind of conservative interactionism. I will use this term frequently moving forward, so I want to say a few words about why the name is a good fit for the position.

3.1.2 – Conservative Interaction

Most obviously, the name is composed of a description (conservative) plus a base commitment to some kind of interactionism. What, then, is conservative, and what does it mean to be an interactionist? The interactionism part is fairly straightforward, since the position describes the ultimate phenotype as arising through a combination of internal (genetic) and external (environmental) causes. To be an interactionist is to commit to neither pure internalism nor pure externalism with regards to the source of causation for development, but instead to say that both have some role (Barker 2015). What about the conservative descriptor? Barker (2015) suggests that the view is conservative in its tendency towards internalistic explanation and in the role it allows external causation to play. On views of this sort, the majority of the heavy lifting for the phenotype is accomplished via internal causation, and the general role of external causes are to either inhibit or enable the body's ability to execute its 'internal blueprint'. For example, feeding a child healthy food may enable their growth, but starving them will surely interfere with the body's ability to develop normally, which is to say develop in the way its internal blueprint would specify if the child were fed. This blueprint also provides a 'preferred' target to aim at, and tries to keep the developmental outcome close to that target if possible. The upshot is that one requires very strong external causation to overpower the internal blueprint, and because of this tendency towards an internally determined target there will be strong limits on the degree to which outcomes can be extremely divergent. This kind of point is often made in pop science or folk biology around the topic of human nature: the idea that our nature has some sort of resistant core, such that there are strong limits on how we can develop because our genes/evolutionary history/internal blueprint push us strongly towards a certain range of outcomes. This sort of story thus makes primary use of internal causation, but reserves some relatively small (ie conservative) role for external causation. Because the end result is explained by both internal and external causal sources, the story is interactionist. Thus: conservative interactionism.

Given this emphasis on internal causation and the role of genes in development, it is also easy to see why the Human Genome Project garnered such attention and

excitement. After all, the HGP sought to map the entirety of the human genome. If genes and gene regulation are the keys to who we are, then why wouldn't a mapping of all our genes be as if we had a window into our souls? This type of overly-simplistic attitude (called 'gene astrology' by Jablonka & Lamb 2014), is also a view widely shared by the general public, who tend to view genes as simple causal agents. If genes did function as simple causal agents and code for specific proteins, then a mapping of all our genes and the proteins they code for would give us a complete view of the adult phenotype (barring inhibiting environmental factors). It's easy to see why such a project would hold such promise in the public eye.

3.1.3 – Genetic Astrology (But Not Really) & The HGP

At this point I would like to add a few qualifiers, since critics might (understandably) say I am oversimplifying matters. First, I have been focusing on the underlying assumptions of this sort of framework and less on the particular views of any one person. Moreover, even in the era of genetic astrology, biologists and geneticists were usually more careful with their wording. At the time, there could often be a distinction between popular discourse and scientific discourse, as one might expect. Where popular discourse was concerned with genes that 'governed adventurousness', according to Jablonka and Lamb (2004) the scientific papers tended to discuss genes that were 'causally related to novelty-seeking behaviour'. As such, it's very easy to over emphasize the simplicity of this framework while strawmanning the professed views of geneticists. It's highly unlikely that many geneticists actually endorse the genetic astrology view. However, sometimes they have talked as if they endorse a view like it. For instance, in 1995, Lodish was asked to give an interview to the journal Science as a leading cell biologist about his view of the future of genetics and development. Put briefly, he thought that if you got reliable information from the maternal DNA (where 'reliable' means both accurate and relevant), you could feed it into a supercomputer along with information about the environment,

> *"including likely nutrition, environmental toxins, sunlight, and so forth. The output would be a color movie in which the embryo develops into a fetus,is born, and then grows into an adult, explicitly depicting body*

> *size and shape and hair, skin and eye color. Eventually the DNA sequence base will be expanded to cover genes important for traits such as speech and musical ability; the mother will be able to hear the embryo – as an adult – speak or sing." (See Lodish 1995, p1609, in Ashburner 1995 "Through the Glass Lightly").*

This doesn't sound quite like genetic astrology, though of course the talk about perfectly knowing the developmental outcome and adult traits of an embryo does sound reminiscent of astrological claims. However, it admits that a lot of information about the environment is needed to build the model, so it cannot be the whole story of development to just know about the embryo's genes. In admitting the need for environmental information, it seems to commit to an interactionist stance. Specifically, it seems to commit to a kind of conservative interactionism, insofar as knowing the genome plus the likely environmental modifiers yields a perfect predictive model of the adult. That kind of thinking about development - where DNA as the inherited and internal driving force (nature) that is modulated by acquired and variable environmental factors (nurture) - is precisely the framework I am arguing against.

As we begin to pivot towards the second part of this chapter, returning to the fallout of the HGP offers a useful transition. As I mentioned above, there were a great many hopes that the project would unlock nearly all there is to know about a person. However, the project did not yield that outcome. Instead, we found that we had fewer genes than we had previously estimated (the first draft of the HGP said 35,000; more recent studies suggest closer to 20- 25,000 (Keller 2014; Mattick 2004), which are both far cries from some of the estimates made before the project started (many guesses posited between 60,000 to 100,000 genes. Collins, the director of the HGP, guessed 80,000. Guesses for fewer than 60,000 genes were quite rare (Keller 2014; Pennisi 2003, 1484). Moreover, the vast majority of genetic information (98-99%) does not code for proteins, but is instead so-called 'junk DNA' (Keller 2014). As such, it is unlikely that there is enough genetic information to fully specify the developmental outcome and processes involved. We learned how much we really didn't know about genes and

developmental systems, because the new discoveries were showing us just how complicated matters were (Jablonka & Lamb 2014). As Jablonka and Lamb note:

> *"the revelations of molecular biology cannot be neatly slotted into the existing framework of thought. They do not make the old genetics complete; rather, they highlight the simplifying assumptions that have been made and reveal vast areas of unanticipated complexity. Genes and genetics can no longer be thought of the same way as in the past. [...] One of the things that molecular studies have reinforced is something that had already been accepted by modern geneticists: the popular conception of the gene as a simple causal agent is not valid. The idea that there is a gene for adventurousness, heart disease, obesity, religiosity, homosexuality, shyness, stupidity, or any other aspect of mind or body has no place on the platform of genetic discourse." (2014, 6)*

The worry I am trying to motivate can be expressed in the following way. Namely, I worry that despite changing how genes were thought of, despite things like epigenetics becoming more a part of mainstream biology, the underlying framework and the way it draws on preformationism and the nature/nurture distinction has not changed all that much. By way of elaboration, consider the following common metaphor: turning genes off and on. Epigenetics are often discussed in terms of gene expression and regulation, usually in terms of methylation patterns that prevents the transcription of the methylated sequence (Jablonka & Lamb 2014). To 'turn off' a gene consists in the blocking of its ability to be transcribed. In discussing matters this way, the focus is still primarily on the gene and what it 'does', and it's treated as a simple causal interaction to 'turn it off' and so 'prevent it from acting'. We may have learned that the notion of the gene as a simple causal agent is not valid, but here again our way of discussing the role of epigenetics is to treat it as an enabler or force of interference for the active gene. Indeed, by using the metaphor in this way, epigenetics has a role very similar to the environment under conservative interactionism. I do not think that is accidental. Lewontin makes a

similar point in his preface to Oyama's Ontogeny of Information (2000), and I think it is worth heeding. To quote:

> *"Virtually all the language of genetics describes the role of genes in the formation of organisms. Genes are said to be "self-replicating", and engage in 'gene action', they 'make' proteins, they are 'turned on' or 'turned off' by 'regulatory' DNA. But none of this is true. Genes are certainly not 'self-replicating'. New DNA strands are synthesized by a complex cell machinery (another metaphor!) consisting of proteins that use the old DNA strands as templates. We do not speak of manuscripts 'self-replicating' in a photocopy machine. Genes 'do' nothing, they 'make' nothing, they cannot be 'turned on' or 'turned off' like a light or a water tap, because no energy or material is flowing through them."*

I have already discussed various forms of interaction in other areas of science. This chapter is, among other things, aimed at exploring and analyzing what exactly interaction in the genetic sense is. A brief foray into some history was needed in order to fully characterize and begin a response to this question. One response is something like the following. Under conservative interactionism, the genetic sense of interaction involves interplay between genes and environment, where the environment serves to enable or constrain the causal force and teleology of the genes. The metaphors and language used to describe this interplay read like simple causal interaction. And while simple causal interaction will of course be part of the picture, I think there is a different and more rich understanding of interaction in the genetic sense. I see it as irreducible, highly dependent, and resulting in an emergent property. In this sense, the 'meaning' of a genetic sequence is not determined on its own, but rather it depends on the 'meaning' of the environmental factor/ interacting counterpart(s). Fleshing out this picture will proceed in the next few chapters, as it requires a few intermediate steps for everything to come together. Thus far I have analyzed several kinds of interaction and alluded to how they might be used to make a more robust form of interactionism. I have also discussed the received view – conservative interactionism – and shown how its framework relies on the

nature/nurture distinction because it trades in familiar binaries such as innate/acquired, inherited/learned, gene/environment, biology/culture, etc. Discussing contemporary interactionism in this way was the 'interactions in genetics: as imagined' portion of this chapter. Next, I will turn to building a positive case for how I think interactions in genetics/biology actually are. My aim is to show that the evidence supports a much more complicated picture than conservative interactionism allows for.

3.2 – Towards My View; More Radical Genetic Interactions

In this section, I argue that the reality of genetic interactions is far more complex than the conservative interactionist picture allows for. There are a couple main threads through which I argue for this position.

For the first main thread, I argue that genetic interactions are not so easily isolated from matters external to the body. Instead, genes are bound up with local environmental conditions in the context of development, and shouldn't be treated as wholly separable. I have two supporting sub-points for this. As the first supporting point, I claim that the classical methods used to try and parse innate and acquired properties – such as deprivation experiments – do not actually give evidence that some property is independent from the environment. My second supporting point is that there is good reason to think that the so-called 'genetic context' extends beyond the body, as per an extended genome thesis. As such, a metaphor of a 'reactive genome' better matches the evidence compared to our classical notions of proactive genes with their genetic programs. One never has genes without an environment. Together, I argue that these two supporting points show that we aren't warranted in holding the classical framework where genes and environment are treated as largely separable entities, and conversely, there is good evidence that they are deeply interconnected. For the second main thread, I finish this chapter by using these conclusions to begin sketching a notion of human 'embeddedness'. To do so, I elaborate on how the examples I have raised in the first thread can be understood as operating as an irreducible system. I then expand on the irreducible interactions of coupled systems, the often-complicated ways in which systems are connected to each other, and how this supports my push for a more radical notion of interaction. I then finish by quickly sketching how these arguments fit into the larger

project, and how the subsequent chapter will expand on this theme by discussing the interactions between acquisition and inheritance.

3.2.1 – Genes and Environment are inseparable; Against deprivation experiments

If one holds that nature and nurture are discreet and separable entities, then the logic behind deprivation studies becomes easier to understand. I'm sure most of us, at some point in our childhood heard or had wondered what a person might be like if they were just raised in the woods by wolves, without any human contact or 'interference'. Experimentally speaking, this musing can be treated as a theoretical attempt to parse which traits would still emerge in the course of development and which would not. Supposedly, the implication is that if certain traits still emerged despite no participation in human culture, then surely those traits must surely be 'innate', or 'pre-determined' in some way that doesn't depend on socialization. This is the point of deprivation/ isolation studies: restrict the access of the developing organism to certain kinds of environmental resources (depending on what the experimenters are trying to control for). While it could be implemented for any species, studies of this sort have been fairly common for birds and bird song development (West & King 2008). As West, King, and others note, “Small cages or sound attenuating chambers were the typical laboratory environment but they structured, restricted or eliminated social interactions. Such isolate housing and the reliance on isolate song supported the dominant view of complete social isolation as being capable of revealing the innate blueprint” (2008, 385; Searcy & Marler 1987). On this way of thinking, innate blueprints are synonymous with a thing's nature, given that a blueprint implies a particular end goal to be aimed at. The blueprint's innateness means it is always readily available to the individual, and likely to the larger group as well by virtue of being innately available to individuals. The logic proceeds in the following way. If we control environmental influences to limit possible learned behaviours, we might arrive at something like an internally-driven developmental outcome. As one might expect from conservative interactionism, if we limit the ways in which environmental factors might inhibit or change the outcome specified by the internal blueprint, then the final product will likely give us a better understanding of which traits are decided by

internal causation and which ones require external causation. In other words, it gives a better shot at seeing something like the default outcome as far as internal causal forces are concerned; the innate phenotype. And given the central dogma of biology where information flows in a single direction from DNA to proteins to the body (Mattick 2004), attempting to find the innate traits is synonymous with attempting to find the traits specified by an organism's genetic programs and/or evolutionary history.

There are at least two related issues with the approach described above, and I will argue that these issues explain why a deprivation framework doesn't succeed in its goal of finding traits that are independent from the environment. The first problem is that it's incredibly unclear what being independent from the environment amounts to. With more complex organisms, you never have a genome without an environment of some kind. Deprivation studies just change the type of environment that the genome is responding to, and the types of developmental resources available for the organism to leverage. Similarly, acting as if the results of deprivation studies showed innate traits only serves to confuse innateness with artifacts, and nature with reliability. This is because the experimental setup treats reliability as evidence of innateness, and if some trait is held to be innate then we are supposed to have a good idea of what that means due to the connotation of innateness (genetic programs, etc). However, a deprivation setup just means that an organism is responding to a strange environment, not an absence of an environment, and (along with the possibility of experimental artifacts) this sort of setup does not shed any light on the actual developmental pathways of a trait (Stotz 2008). At best, one would have a result that is developmentally particular to that experimental setup. And given the difference between the experimental setup and the organism's usual environment, it is hard to see how the experiment sheds much light on how the trait(s) might develop in a more standard setting.

3.2.1.1 – Cowbirds Case Study

West and King's studies on brown-headed cowbirds are useful in highlighting my points about deprivation experiments. When deprivation studies were used with cowbirds, certain behaviours seemed reliable: female song preference for song from their natal region was robust, mate recognition seemed to not require previous experience with

conspecifics, etc (King & West 1977). In the literature, this was taken to be evidence for some sort of innate safety net that had been posited for the brood parasite. However, hand waving an innate explanation is not particularly satisfying, as this doesn't help specify why and how these findings arise. Subsequent studies on cowbirds revealed that female song preference can be remarkably plastic (West, White & King 2003; West et al 2006); female preference for song from her natal region can be erased (West, White & King 2003); males exhibit remarkable macrogeographic plasticity in their song production and will adapt their repertoire based on social cues (West & King 1985; 1988); and males can indeed pursue the wrong species in courting attempts (West, King & Freeberg 1996), etc. Moreover, these subsequent studies highlighted the developmental pathways, which rely strongly on social interaction and flock dynamics (West & King 2008). In all these cases, female-female social interactions were the drivers of fixing a female's song preferences for a given mating season; male-female social interactions shaped the songs produced and the social skills exhibited within the flock, etc. Social interactions and flock dynamics were the drivers for all these effects; the 'innate safety net' is replaced with a rich exogenetic inheritance model, where the reliable behaviours emerge from underlying social interactions. It was only by changing investigative frameworks and looking to sociality as fundamental for development (eschewing the deprivation method) that the actual developmental pathways were highlighted. The nature/nurture framework served to obscure the actual points of interest, and actively interfered with our ability to gain reliable knowledge about the species. By making efforts to get outside of this framework, new inroads were made and better explanations were obtained. At this point it goes somewhat without saying that I don't think it's an accident that better results were obtained once innate explanations were ditched and interaction was taken seriously instead.

Merely arguing against deprivation-style experiments is insufficient on its own to make my larger case. At best, the argument against deprivation serves to advance a few ideas. First: a creature's nature is not revealed through environmental deprivation. Second: we should recognize that there is no such thing as a lack of an environment. No genome is without an environment, and typically vice-versa (at least on Earth). But

claiming that an environment is always present is not yet a claim about the role of the environment in development. That, then, is what I will turn to presently.

3.2.1 – The Extended Genome Thesis

To make a more forceful claim about the role of the environment in development, I will argue for what can be called the Extended Genome Thesis. This is my second supporting point for the overarching claim that the genetic 'domain' is not easily isolated in the way conservative interactionism pretends.

The Extended Genome Thesis is effectively what it sounds like – namely that in some important sense our genome extends beyond our body. Typically, this also involves some sort of claim that, since the genome is extended in this way, it is likely coupled to the environment in some important way. Before defending this position, I want to briefly discuss what it does for my overall argument. If the genome is extended beyond the body and coupled to the environment in some important way, then this becomes an argument that the supposed distinction between genes and environment is merely heuristic. If the distinction is merely heuristic, then the conservative interactionist picture begins to look quite suspect, since it relies on the addition of separate categories to characterize development. Additionally, the gene-centric character of the view begins to fall apart if the genome and environment are coupled and interconnected in this way. A new, more radical kind of interactionism would thus be required to account for the evidence.

Admittedly, the extended genome sounds like a strange idea to many when they first encounter it. My defense of it is primarily borne out of the postgenomic literature, and contemporary attempts to patch the holes we found in our understanding of genomics in the wake of the shortcomings of the Human Genome Project. My arguments below include the following points. First, given what we know from the HGP, there are not enough protein-coding sequences to fully specify the human form as an innate property of DNA. As Keller (2014) remarks, we don't have many more genes than the worm *C. Elegans*, but obviously we are substantially more complex organisms. Thus, something else must be involved beyond just mere gene action. Following Keller (2014), Stotz (2006, 2008) and others, I will pivot to the notion of a reactive genome that uses many

factors -including environmental cues – to regulate gene expression and genomic products. The argument for this pivot is that the genetic 'environment' involves a number of factors both internal and external to the body. Development consists in the interaction of these factors to mutually regulate and construct the genomic product. Since the environment is involved in the construction and informational specification of the genomic products, the genome is thus sensitive to the environment. This sensitivity is such that the environment co-specifies the genomic product. Thus, the genetic context extends beyond the body, in line with the Extended Genome Thesis. A more fleshed out defense of the above is presented below.

3.2.1.1 – The Reactive Genome

Increasingly in the postgenomic literature, the metaphor of the active gene is being replaced with the notion of the reactive genome (Keller 2014). This is certainly a change from the early days of molecular biology, but in both cases a similar understanding of the word 'gene' is at play. The informational content of the gene is understood differently, however. For early molecular genetics, genes were entities that made proteins, which is to say that they carried the information necessary for the construction of polypeptide chains out of amino acids. In this context, “information referred to protein-coding sequences, DNA was made up of genes, and genes 'acted' by making proteins.” (Keller 2014, 2425). While the meaning of the term 'gene' has been difficult to specify, the most common usage in contemporary biology still refers to protein-coding sequences (Keller 2014), though of course not all genes code for proteins.

In the context of a reactive genome metaphor, genes retain their status as protein-coding sequences. However, they are seen less as independent, stable entities and more as products of the reactive genome itself: “[genes] are “things an organism can do with its genome” on the spot to create a template resource for a product a cell may need at any particular time” (Stotz, Bostanci & Griffiths 2006, 195). This new metaphor reflects how DNA sequences alone do not specify their products, but rather how the same DNA sequence could be used for many gene products. It is possible for the same DNA sequence to be involved in multiple products, or different sequences might produce identical products. These facts, says Stotz (2006), are evidence that all the requisite

information for a given gene product is not merely contained within the DNA sequence. Supplementary specification is needed, whether this be from transcription, splicing or editing factors. Genes themselves are also contingent entities:

> *"What counts as a gene – where it begins and where it ends, which sequences it comprises – is determined by the genomic, cellular, and extracellular phenotype at each point in the organism's developmental trajectory. The whole determines what counts as a part." (Stotz 2006, 906).*

Genomic reaction and gene expression are thus central to development on the postgenomic approach. Gone (for the most part) is talk of straightforward causal interaction that treats genes as simple causal entities. In its place, dependence and coupled interactions take centre stage, as shown when Stotz discussed how 'the whole determines what counts as a part' – implying at least multidirectional causation.

3.2.1.2 – Co-determination of Genomic Products

A number of interesting findings lend support to this framework. First, the main genetic output in eukaryotes (98% in humans (Mattick 2004; Mattick 2001)) is genetically active but noncoding RNA, (as opposed to proteins). Rather than just be 'junk DNA', as it had been traditionally characterized, this noncoding RNA is now taken to have an important regulatory role. For example, is now hypothesized that this ncRNA/ intronic RNA "feeds genetic information into the regulatory network of the cell." (Mattick 2004; 2001). Moreover, ncRNA dominates the genomic output of the higher organisms and has been shown to control chromosome architecture, mRNA turnover and the developmental timing of protein expression, and may also regulate other functions like transcription and alternative splicing (Mattick 2003). One of the ways it might do this is because "eukaryotic genes seem to express two kinds of information in parallel: proteins and 'efference RNA signals' that can communicate with other genes or gene products independently of the biochemical function of the encoded protein of the host transcript." (2004, 319) The point here is that proteins are not the only thing that the genome can do, and information does not exist only in the DNA to protein pipeline of the

central dogma. Generating multiple kinds of information helps the genome be flexible in its reactivity.

These examples with RNA show one dimension of a regulatory network. However, this network is not a mere background condition. It is part of the genetic 'environment' that affects what counts as a gene and how the gene is expressed. As Stotz (2006, 906) notes, there are two main components of this genetic environment. First, there are "(1) regulatory and intronic sequences that are targeted by transcription and splicing factors (proteins and noncoding RNAs) that bind to them and (2) the specific environmental signals that cue these factors or otherwise influence the gene's expression." Much like how I argued above that deprivation experiments never show a genome without an environment, Stotz's point is that these regulatory sequences are similarly coupled with the environmental factors that cue them. There is not one without the other. And since regulatory mechanisms are coupled to environmental cues, the claim can be made that this wider context of both genetic and environmental factors jointly co-specifies the genetic products. It is exactly this sense – the co-specification of genomic products – in which the claim of the Extended Genome Thesis is relevant.

There are several examples of how this environment can help co-specify gene products or change things up such that entirely new products are created if needed, especially by reshuffling or editing the original DNA sequence to produce wholly new protein templates. One way of doing this is by splicing together separately transcribed sequences to create a single mature RNA (Stotz 2006). There are a few ways to do this, depending on whether the spliced exons are from the same gene or different ones. (Exons are the parts of a gene sequence that are expressed into amino acids. This is contrasted with introns, which are noncoding sequences that occur in between exons. As such, exons are expressed, and introns are in between). Separately transcribed exons from a single gene can be combined in different orders or repeated within a transcript (Flouriot et al. 2002; Takahara et al. 2002). Alternatively, exons spliced from separate genes can create a protein with an amino acid sequence that isn't part of the standard DNA expression (Blumenthal and Thomas 1988; Finta, Warner, and Zaphiropoulos 2002; Zhang et al. 2003).

Both of the above examples undermine the standard gene to protein pipeline of the central dogma, but in a subtle sense. It shifts the emphasis from 'what genes do' to 'how the genome regulates the products' in the sense that a gene sequence need not create a single given product. Different gene products can be created by shuffling and recombining either parts of the same gene sequence, or parts of different sequences. But the point is that the regulatory network oversees this, and it is highly responsive to the environment. Thus, even if someone wanted to emphasize straightforward transcription of exons to amino acids, those exons might not create their standard product because the cell machinery might recombine them in different ways based on the regulatory network. The point isn't to say that proteins and exons have no role; the point is to highlight the ways in which the gene-centric approach fails to aptly characterize the ways in which both internal and external factors can jointly co-determine developmental products.

A second example of how this regulatory network can co-specify products is through RNA editing. Editing in this way is particularly noteworthy for Stotz, because:

> *"RNA editing disturbs [the correspondence of the primary structure of exon and gene product] by changing the primary sequence of mRNA after its transcription. The creation of 'cryptogenes' via RNA editing of the gene's pre-mRNA is therefore a very extreme mechanism of genomic information modification, which can be rather extensive with up to several hundred modified nucleotides. Editing events occur in such diverse organisms as viruses, slime molds, higher plants, and mammals and have, among other things, profound effects on the function of transmembrane receptors and ion channels in mammalian neural tissues, in erythropoiesis and inflammation, in cardiovascular disease, in cancer, and on the life cycle of viruses." (2006, 909, emphasis in original)*

According to Samuels (2003), one of the functions of RNA editing (at least in mammalian APOBEC-1 proteins) is regulation of different sorts. This regulation enables

diverse protein production for the animal's genome, but it can also block protein expression from viruses by editing out the viral portion to be transcribed. As such, the regulatory mechanisms, via editing of this kind, can promote or inhibit different kinds of protein depending on what is needed. This showcases different kinds of potential reactivity, especially with the ability to edit out viral protein expression. The ability to react to invasive proteins and respond appropriately in virtue of the regulatory network shows how the final gene product can evolve and change at each stage depending on the previous stage and how the regulatory network responds to that previous state.

There are also complications for the central dogma involving epigenetic effects. To the National Human Genome Research Institute, Epigenetics are understood as changes to the function and expression of genes (ie the metaphor of turning them off or on) without accompanying changes to the DNA sequence. While epigenetic effects can come in different forms, they most commonly are associated with chromatin, histones, and DNA methylation. Chromatin is all the stuff associated with chromosomes: the DNA, RNA, proteins and other molecules so associated (Jablonka & Lamb 2014, 124). Histones are proteins that are part of chromosomes and chromatin, as regulated protein packages that house DNA and compact it. The way that DNA is packaged, the packing density of its surrounding proteins, etc all affect the rate at which the DNA can be transcribed by making the gene(s) more or less accessible (Ibid, 125). Methylation is a specific type of chromatin mark for a group of nucleotide bases that gets copied in transcription. As with the other epigenetic effects mentioned above, typically more densely methylated sequences are transcribed less often, but it can do this in a variety of different ways. I'll touch on more details as they come up in the cases, but for now I just want to have a broad overview of the fact that epigenetic effects can take several forms and tend to affect gene expression and transcription rate.

There are many ways in which genetic and various epigenetic factors can interact, but some involve the influence of epigenetics on genetic changes, especially mutations. For instance, an early sign of cancerous tumours can be a “change in heritable chromatin marks, such as an increase or decrease in density of DNA methylation.” (Jablonka & Lamb 2014, 244). It's thought that these changes affect the cell's ability to repair DNA by

blocking genes that are involved with products pertaining to cell maintenance. This leads to DNA damage accumulating, and since both the genetic and epigenetic changes are inherited by daughter cells, this leads to more widespread damage.

It also is possible for epigenetic effects to have a strong hand in germ line mutations, which goes against the distinctions in the received view. (To use Dawkins' recipe metaphor again, this would be like burning the bread becoming an heritable trait in some way). In maize, for instance, the plant seems to alter its genome in response to stressful conditions via 'jumping genes' (Jablonka & Lamb 2014). Contemporary work on these types of genes shows that a large part of which genes 'jump' depends on the plant's inherited epigenetic state (Raina et al 1998; Jablonka & Lamb 2014). Summarizing the findings, Jablonka and Lamb note:

> *"Transposability is correlated with DNA methylation: elements that are capable of jumping are less methylated than those that are inactive, which are normally highly methylated. The methylation marks of potentially active elements can vary in a way that depends on factors such as the cell's position in the plant, the sex of the parent cell from which it was derived, and various internal and external conditions. Stresses such as wounding, pathogen infection, or genomic imbalance (having too much or too little of some chromosomes or regions of chromosomes) can lead to substantial changes in methylation marks, followed by vigorous jumping. As mobile elements excise themselves and insert into new locations, they introduce mutations into both coding and regulatory sequences. Active chromosome regions are particularly inviting sites for the transposing elements." (2014, 245)*

What does all this mean? I take these findings to be important for two reasons. First, they help flesh out the highly dependent and interactive nature of these systems. It was already generally accepted that epigenetics was worth paying attention to in its own right, but these findings indicate it is important for genetics as well. (Indeed, it is worth questioning what is 'epi' about epigenetics and how the two intersect (Keller 2014)).

Second, I had discussed earlier the claim that multiple interactants co-specify the gene products that are produced, and I take these findings to lend some credibility to that. In this case, external cues (wounding, pathogens, etc) can lead to widespread epigenetic changes, which in turn affects which DNA sequences move around, which in turn affects gene expression. Different products may be constructed as a result of external cues, and the epigenetic methylation changes serves as a way to regulate gene expression and mutation. As per McClintock's (1984) claim in the 80's that plants can reshape their genome in response to stressors, we can see that the complex network that affects gene expression helps lend credence to the metaphor of a 'reactive genome' rather than 'active gene'. Additionally, given that the epigenetic changes can be driven by external cues, and epigenetics are one of several ways in which gene expression can be regulated, something like the Extended Genome Thesis has to be true. The implication is that the genetic 'domain' is expansive and difficult to separate from other networks, since multiple types of processes – including those originating external to the body - seem to be involved in the regulation of what genes get transcribed and what the genomic products are.

3.3 – Towards a More Radical Interactionism

I've thrown a lot of information in this chapter, and I want to talk about what it all means for my project. A list of central themes includes: co-specification/ co-determination, irreducible interaction, and the reactive (extended) genome. Additionally, I have claimed that the themes just listed undermine the plausibility of conservative interactionism. They push us towards a more radical understanding of interactionism. It's worth spending a bit of time to tie these threads together and make the case explicit before moving on.

To begin, it's worth remembering that I have a specific sense in which I am discussing irreducibility. That sense is based on interaction in terms of coupled, nonlinear equations, as discussed in the previous chapter. In such equations, the variables must be solved for at the same time since they cannot be isolated. They cannot be isolated because the variables co-determine each others' value; to isolate a variable is to change the value of the variable one is hoping to study. The variables are entangled, such that knowing the value of one part of the system need not tell you much about the state of the entire system.

This particular sense of a system being irreducible can be brought to bear on my examples in a couple ways. In particular, it is useful in showing how the 'meaning' of a gene sequence is determined on my account as opposed to conservative interactionism.

For conservative interactionism, the 'meaning' of a gene sequence depends on the information it carries. This information seems to be innate to the DNA, though still contingent on evolutionary history. As per the central dogma, evolutionary history shapes the information in DNA, DNA codes for specific proteins to express that information, and thus is the information made available to the rest of the body. Through this pipeline, there is a smooth and unidirectional flow of information from the DNA to the rest of the body. And because we inherit DNA from our parents, the information for our phenotype pre-exists us. Thus, this information is held to be innate, and gives the framework preformationist leanings.

However, that sort of picture doesn't seem to match the various examples I provided. Maize's response to stressors is highly contingent on the kind of stressor – it seems much more reactive than proactive. That picture also doesn't square with the way that genomic products depend on the regulation of a network, and the way that this network seems to be responsive to various factors. If my interpretation of those phenomena is correct, then it seems to be the case that both internal and external factors can jointly determine genomic products. This is a very different picture from the 'information pipeline' of conservative interactionism.

This jointness of product determination, this co-specification, is well understood by treating it as an irreducible system. As with the coupled equations, there can be multiple variables in play, and knowing the value of one is not sufficient to give you the full sense of what the system is doing. For example, if we take the case of RNA splicing or editing, knowing what a given gene tends to code for doesn't get you very far in assessing what the genetic products will be. This is because the products can be altered by other cell machinery; standard RNA could be combined or jumbled to create new sequences, or whole sections could be edited out. The notion of innate information

simply doesn't tell you much about a situation where the outcomes are reactive and contingent. When looking at the irreducible system, however, the 'meaning' of a gene emerges through the interactions that specify its product, and these interactants function as if they were coupled. The information does not pre-exist the reactive interaction, since the regulatory network and cell machinery can alter the genetic product to be produced in response to some cue. That cue and the regulatory response thus jointly determine the product and any information it may contain. The whole system, through its reactions, will determine what counts as the relevant parts of the reaction. As Stotz (2006) and Robert (2004) put it, the developmental process interactively constructs the informative-instructional content of genes. “Epigenesis is constitutive”; it “does not reduce to gene regulation, for genes themselves do not pre-exist developmental processes” (Robert 2004, 74).

This system is irreducible in the sense that there is an extended developmental system that can have both internal and external factors coupled together. Through niche construction and our ability to engineer (even self-engineer) an ontogenetic niche, the influence of these factors can be bidirectional. (I will say more on this niche construction aspect in the following chapter). The point to emphasize, again, is that knowing one variable does not provide insight on the whole system. Knowing a creature's genome is only one part of the co-determining coupled system, since genes are not simple causal agents. It is through the interaction of mutually-influencing coupled variables that information is constructed and made available to the system.

As we reach the end of this chapter, I again want to emphasize the change in what kind of interactionism we ought to be endorsing. The version I am advocating for is not merely a revision of conservative interactionism that also stresses the importance of the environment. Nor am I intending to claim that the environment reigns supreme since the genome is reactive to it. My aim is much more radical than that. By stressing the interpenetration of variables in a coupled system, and the way they can co-determine genomic products, I am attempting to break down the divide between genes and environment. If they are mutually-influencing in the way I have described, then it is a mistake to be too gene-centric in our approach to understanding development since many

other factors will play a role and can have a hand in determining the genetic response. This is not to say we cannot draw heuristic differences between genes and the environment. But it is a much more radical step to partially dissolve the divide between the two, and show how the reactive nature of the genome serves to construct information based on the interactions involved. In Chapter Five, this partial dissolution of the gene/environment dichotomy will be part of my argument for the incoherence of the Nature/Nurture framework, since multiple instances of the dichotomies that the framework relies on are irreducibly interactive in the way I've described above. But before that larger argument can be made, another aspect of the conservative interactionism pipeline must be examined: the difference between acquisition and inheritance. This will be the subject of this next chapter, before tying all the threads together into a larger argument for the incoherence of the Nature/Nurture framework in Chapter Five.

Chapter 4: Acquisition, Inheritance, and Ontogenetic (Re)Construction

In Chapter 3, I made the argument that the genetic and environmental 'domains' strongly overlap to the point where there is co-determination of genomic products. Since heavy overlap and co-determination is the case, the Extended Genome Thesis (or something very like it) is true. Because this thesis is true, the traditional gene/environment dichotomy can be understood as a heuristic distinction, but nothing stronger. Instead, as Chemero had noted with dynamical systems and radical embodied cognitive science (2011, 25-6), we should think of the developmental and genetic contexts and the environment as forming a larger, mostly nondecomposable, system. This argument was made by showing how the genome can be thought of as responsive and coupled to local environmental conditions, and how multiple subsystems overlap and co-determine genes and gene products. Many of these systems are sensitive to environmental conditions, and so the environment is part of the systems that govern gene expression and products. Given the genetic 'context' can, in this sense, extend beyond the body and include the environment, the gene/environment dichotomy is better thought of as enmeshed systems. These systems function like coupled equations: knowing the 'meaning' of some gene doesn't get you very far without also knowing the meaning/values of variables associated with the regulatory networks.

Much like the gene/environment dichotomy, acquisition and inheritance are traditionally seen as separate categories. Both of those pairs can be seen as extensions of the nature/nurture distinction: nature is internal (ie genetic), inherited, and stable, whereas nurture is external (ie environmental), acquired, and a source of variance through modulation. My stated aim in this project is to characterize development as irreducible interaction, and to connect this with the idea of embeddedness within a niche that allows for the leveraging of developmental resources.

My approach in this chapter will mirror Chapter Three. I think the evidence suggests that inheritance and acquisition are not neatly separable, and moreover, I think there is substantial evidence that there can be a feedback loop between processes that are traditionally categorized as either acquired or inherited, such that activity in one category can drive how we classify matters in the other. (At times I refer to this as 'the categories interact' for brevity's sake, but of course do not mean that categories themselves literally interact. Rather, the behaviour of some processes can shape how we classify traits on this dichotomy, and there seems to be many opportunities for feedback effects between the phenomena). This additional layer of interaction further blurs the ability to draw clean distinctions between acquisition and inheritance.

Given the Nature/Nurture framework (and conservative interactionism's tendency to rely on clean distinctions between dichotomous categories, blurring the boundaries between categories undermines the framework). And in the context of my larger project, my argument in this chapter also helps undermine the Nature/Nurture framework since the extensions of each dichotomy are not distinguishable. That overarching argument will come to a head in Chapter Five.

The present chapter proceeds in three main sections. The first section introduces the standard understanding of inheritance and acquisition. I also continue the historical aspect of my approach by linking this standard doctrine to the divides between Darwinian and Lamarckian thought. There are, I believe, important points to be made here about why the Nature/Nurture dichotomies are drawn up the way they are, and why the distinction between acquisition and inheritance is so important. The second section offers some cases where, I argue, the boundary between acquisition and inheritance starts to fall apart. This section chiefly relies on examples involving epigenetic inheritance and niche construction. The third section outlines ways in which the feedback loops between the phenomena can affect how we categorize them, using examples involving genetic assimilation and the cascading effects of niche construction.

4.1 – Inheritance & Acquisition: an Introduction

It is probably best to begin with some definitions and background material. As such, the first part of this introduction is concerned with describing the received view as it pertains to both heredity and acquisition/learning. Additionally, because the distinction between inheritance and acquired characters has been tremendously important, the second part of this introduction will remark on the ideological framework that has been strongly involved in how this distinction plays out. Notions that respect this distinction are seen as properly Darwinian, but transgressing ideas invoke charges of 'Lamarckism' (effectively meaning poor biology or evolutionary theory by invoking a defeated competitor to Darwinian theory).

The concept of heredity has meant different things to different people at different times throughout history. Given the increased molecularization of biology, this is probably unsurprising. For present purposes, however, I am concerned with a contemporary neo-Darwinian or Dawkins'-esque conception of heredity. According to Mosini (2013), the standard received view involves the information conception of heredity (Crick et al 1957). This information – sometimes called 'Crick information' – involves the specification of the linear sequence of amino acids in a chain (Thieffry & Sarkar 1998). This notion stemmed from the discovery of the double helix structure of DNA, since Watson & Crick thought this might suggest a possible copying mechanism (Watson & Crick 1953b; Mosini 2013). As part of this information conception of heredity, “the specificity of a piece of nucleic acid is expressed solely by the sequence of its bases, and this sequence is a (simple) code for the amino acid sequence of a particular protein.” (Crick 1958, 152). Nucleotide sequences, insofar as they code for amino acid components of proteins, are thus understood as information *for* that product. To have a sequence be copied and have the copy be made available to the daughter cell is just for the daughter cell to inherit the protein-specifying sequence.

Crick information quickly grew in popularity and became part of the central dogma (Mosini 2013). As a result, it is hardly surprising that the information conception of heredity (and frameworks that rely on it) tends to be quite gene centred. There are several examples of this. First, for Selfish Gene neo-Darwinians, the DNA sequence is

explicitly the unit of heredity (Jablonka & Lamb 2014, 39), with DNA replication being the key driver of hereditary transmission. Second, the three stages of the information conception all centre around the copying of DNA sequences. These three stages are: (1) replication of DNA, (2) transcription (understood as the transfer of information from a DNA template in the construction of RNA), (3) translation (the transfer of information from RNA to amino acids for protein synthesis) (Mosini 2013). Inheriting information is thus understood as the copying of existing, typically genetic, information and transmitting it to some new generation (of cells, creature, etc). With this in mind, it's quite easy to see the association between heredity and Dawkins' talk of replicators, since replicators are “anything in the universe of which copies are made” (Dawkins 1982, 83). This is to be distinguished from 'vehicles': entities which house replicators and works for their preservation and propagation (1982, 114). Copying DNA is thus central to this view. Change or variation is explained either by random mutation or through mistakes in the copying process (which is imperfect, after all). Given this focus on copying DNA and genes, Dawkins also explicitly assumes that genes are the only biological (ie noncultural) inheritance units. Moreover, by virtue of his definition of replicators and how things like bodies are taken to be just vehicles for the replicating DNA they house, Dawkins also explicitly assumes that development doesn't impinge on heredity. This is like the metaphor of the cake recipe. The recipe itself can be copied, but burning a single cake by leaving it too long in the oven (ie taking its developmental context into account) is not a trait that is necessarily passed on to future cakes. After all, burning an individual cake does not change the general recipe for a cake. As such, there are three main claims involved in the received view of heredity, and these claims have remained important as long as the information view has been influential. 1) Heredity involves copying some unit for subsequent generations; 2) DNA is the main (biological) unit that is copied; 3) DNA contains information, like a recipe, and so copying the same template across generations is taken to help bring about stability of product.

By contrast, acquired traits and characters can come about in a wide variety of ways. Scars, changes in level of athletic ability, the languages one can speak, learning to drive, etc, are all examples of properties or skills that an organism (typically human) can acquire during their life. This means that acquisition is bound up with a creature's

development, since it involves changes that take place during development. (This is opposed to having changes take place before development which are then inherited). One reason to think acquisition is so closely associated with development is because the set of dichotomies involved in the nature/nurture framework feature acquisition on the nurture side of things. Recall the typical paired and opposed dichotomies: inner/outer, stability/variation, nature/nurture, gene/environment, and inheritance/acquisition, where the initial terms all compose one group and the latter terms compose a second group. Acquisition is thus associated with nurture, change and variability, and the environment. Indeed, as one might expect from these sorts of dichotomies, sometimes the definition of one category is based on the negation of the other. In the case of acquisition and inheritance, there is the question of how an organism comes to have the traits and properties that it has. Some it ostensibly inherits, based around complex copying mechanisms, and these inherited traits have the additional property that they will be copied to subsequent generations. If the organism has any properties that were not inherited, then those properties are taken to be acquired. The source of the acquisition can vary: from parental effects, environmental idiosyncrasies, learning, etc.

On this dichotomy, acquired traits are distinct from inherited traits in that the former ostensibly do not have the property that they will be copied to subsequent generations – at least not in terms of biological mechanisms. A child raised in a house that speaks French will learn to speak French, and likely will pass on the skill of speaking French to any children it has. The transfer of this skill, however, will be via cultural methods rather than of passing on a 'gene for speaking French' or anything like that. Once again the cake recipe metaphor is useful for understanding how this dichotomy is understood via the received view. A recipe can be copied, using itself as a template. It contains information on how to make a cake, and this information can be copied for a number of different people. The cake product can be burned, cut, and ultimately devoured, but this does not change the recipe on its own. The burns, cuts, style and colour of icing (assuming the recipe doesn't specify) are all acquired traits, and changing any of these values does not alter the recipe itself. Even if someone didn't have a cake tin and chose to use a muffin tin instead, this would by analogy be a cake with strange

developmental conditions, and different acquired characteristics as a result. (Hence the association between acquisition, variability and the environment).

It is also worth mentioning the ways in which the above framework is connected to an overarching conceptual stance, and how this stance purports to distinguish good and bad biological theorizing. This stance (and resulting effects) stems from the historical competition between Darwinian theory and Larmarckism. In short, Darwinian theory was the victor, and so our theories should aim to be Darwinian and not Larmarckian. Jablonka and Lamb summarize the popular understanding well:

> *"Lamarck, [people] have been told, put forward a theory of evolution fifty years before Darwin but got the mechanisms all wrong. Foolishly (somehow, Lamarck is always made to seem foolish), Lamarck believed that giraffes have long necks because their ancestors were constantly striving to reach toe leaves on tall trees, stretching their necks as they did so. They passed on these stretched necks to their young, so that over many generations necks became longer and longer. Lamarck, the story goes, saw evolution as the result of the inherited effects of use (or disuse). His big mistake was to assume that "acquired characters" - changes in structures or functions that occur during an animal's life – could be inherited. Fortunately, the story continues, Darwin showed that natural selection, not use and disuse, is the cause of evolutionary change, so the idea that acquired characters can be inherited was abandoned." (2014, 13)*

Jablonka and Lamb go on to explain that, despite its popularity, this popular story is wrong in many regards. Darwin did think that the effects of life's conditions on a creature did affect heritable variation, and Lamarck didn't invent the notion that acquired characters can be inherited. Instead, this was apparently a very popular notion at the time, and was certainly present in Darwin's thinking. Versions of neo-Darwinism and neo-Lamarckism came and went, with several prominent scientists such as Herbert Spencer, Samuel Butler, and George Bernard Shaw espousing tenets and ideas of neo-Lamarckism

present in their time. Development was still taken to be important for heritability. However, this changes around the turn of the 1900s, as the Modern Synthesis comes into being, pairing Weismann's neo-Darwinism with Mendel's laws (2014, 24). Corroboration of Mendel's laws in 1900 by Hugo de Vries, Carl Correns, and Erich von Tschermak boosted its popularity, and so 1900 was taken to be the birthdate of the field of genetics. With the focus now on genes, development and acquisition was seen as less important, since heredity was now understood as the transmission of genes through the germ line, and the genes contained information about the characters of the organism.

My point here is not to fully analyze the complete history around these events, as that would be an entirely different project. I have offered this admittedly extremely brief and curtailed version of events because I think it is useful for two reasons. First, it helps remind us that Lamarckian-style ideas have often been popular in biology, and have been recurring figures in biological discussions. Second, this brief history is helpful in understanding the implicit framework of good vs bad biology. Remember, on the popular understanding of the history, foolish Lamarck thought that acquired characters could be inherited, but Darwin set the record straight with the focus on natural selection. Moreover, the pairing of neo-Darwinism with Mendel's laws put the focus centrally on genes as the inherited units, with acquired traits not being a part of this picture. In terms of popularity and proliferation, this was a massive win for neo-Darwinism and the Modern Synthesis. Doing Good Biology now means operating within the neo-Darwinian paradigm, and focusing on genes as inherited units. This means that maintaining the dichotomy between acquisition and inheritance is part of doing good neo-Darwinian biology; by contrast, attempts to blur the line between the poles invokes the spectre of Lamarckism ('Bad Biology'), since that would mean suggesting that acquired characters could be inherited.

My emphasis on this framework of Darwinism vs Lamarckism – seen as Good vs Bad biology, respectively – is also intended as a way of forestalling possible charges that I am oversimplifying matters. After all, surely everyone admits we inherit environments. But there is a difference between merely inheriting an environment, and having this inheritance factor importantly into one's theory. Also, my earlier suggestion of Dawkins -

where I mention he assumes genes are the only biological inheritance units – does not preclude the possibility of cultural inheritance. Of course there can be cultural inheritance, and Dawkins is not blind to this by any means. But my focus on the centrality of DNA as the main heritable unit is not to suggest that no one ever discussed cultural inheritance or attached any importance to it. Instead, I am trying to show how the biological side was the primary focus, where heredity is primarily associated with genes and internal causation. Indeed, the simple combination of biological plus cultural inheritance bespeaks the kind of conservative interactionism I've been discussing and criticizing. Biology and culture are heritable as entirely separate units, with each category contributing something (separately) towards the organism's development, implying that development just is the addition of nature and nurture as discrete and separate categories.

4.2 – The Distinction Blurred

I am not at all convinced that there is such a hard and fast distinction between acquisition and inheritance. As with the distinction between genes and environment, I suspect there is substantially more interaction and co-determination than the distinction traditionally allows for. My argument towards that end proceeds in a couple steps. First, I outline the idea that there are multiple inheritance systems – genetic, but also epigenetic, behavioural and symbolic (Jablonka & Lamb 2014). I think there are likely other forms of inheritance as well, but for present purposes I will focus on these four. The point of emphasizing multiple inheritance systems is to show that a larger range of phenomena might be of interest than is traditionally thought. Additionally, there are straightforward ways in which these inheritance systems might overlap or mutually influence each other. Behaviour, after all, allows us to effect a great many changes in the world around us. And I think by examining these sorts of things more closely, we see the boundaries between acquisition and inheritance begin to dissolve.

First, while we already understand genetic inheritance and its role in the central dogma, I should say a few things about other inheritance systems. I will begin with epigenetics, followed by behavioural and symbolic inheritance systems – thus following the same ordering used by Jablonka & Lamb (2014) in their discussion of these matters.

For now I will merely introduce each system. The discussion of their import will follow after.

4.2.1 – Epigenetic Inheritance Systems

Epigenetic Inheritance Systems (EIS) are cellular systems that are responsible for the maintenance and transmission of both gene activity and expression, as well as structures and states of cells (Jablonka & Lamb 2014, 115). According to the National Human Genome Research Institute, epigenetics is the study of changes in cell function that are heritable, but whose causal power is not attributed to changes in the DNA sequence. Similarly, other researchers such as McGowan and Szyf (2010) characterize epigenetics as long-term changes in a gene's function that do not also involve corresponding changes in the gene's structure or sequence. However, this can also apply to cells, as the differentiation of cell kinds is a classic topic in epigenetics. For example, when liver cells split during mitosis, the daughter cells remain liver cells (and not, say, lung or kidney cells); there is something about being one kind of cell and not another that is heritable. The structure of these three types of cells are different, but their DNA is identical. Thus, this is a kind of structural difference that is heritable (ie liver cells produce liver cells), and not attributed to a difference in DNA (since the DNA is identical).

Additionally, Epigenetic systems can have far-reaching effects across generations, which helps convey their importance. For example, famine and environmental stress can alter methylation patterns, which are heritable, and can greatly affect gene expression. This allows things like effects of trauma to persist through multiple generations. As such, epigenetic changes are one way that the genome and extended regulatory systems can respond to the environment – by altering patterns of gene expression in response to some environmental cue (or behavioural cue, as I will endeavour to show later in this chapter).

A classic example of this involves the epigenetic inheritance of the effects of deprivation. Growing up in a lower socio-economic class (SEC) can be correlated with a wide variety of psychological, behavioural and biological effects. Miller et al (2011) proposed a few mechanisms by which early-life stressors can have uptake within 'biological' factors (as opposed to behavioural or psychological). They also found that

blood DNA methylation profiles are more strongly correlated with childhood SES than adult SES. These methylation profiles also correlate more strongly with earlier childhood adversity than later adversity (Borghol et al 2011; Epsosito et al 2016; Pepper & Nettle 2017). Put briefly, (as I will expand on the import of all these inheritance systems later), some scholars (Pepper & Nettle 2017) suggest this evidence suggests there are epigenetic mechanisms that correlate early developmental conditions and stressors with the 'live fast, die young' form of life-history theory. The heritable nature of these effects has implications for the intergenerational inheritance of metabolic disease (Godfrey 2010), and so some scholars talk about the feedback loops that can embed the effects of deprivation and reinforce them over generations (Pepper & Nettle 2017).

4.2.2 – Behavioural Inheritance System

The second exogenetic inheritance system that I want to draw attention to involves behaviour. Behavioural Inheritance Systems are ways of passing on information to subsequent generations, primarily through social learning and the inheritance of traditions (broadly understood). This bears more than a passing resemblance to notions of cultural inheritance, though the focus is slightly different by emphasizing behaviour as opposed to culture in general. (Calling it 'behavioural' instead of 'cultural' also sidesteps tangential arguments about whether nonhuman animals have a culture or not, since they do exhibit behavioural inheritances regardless of one's sentiments about calling this 'culture'). A great example of this involves the changing habits of Japanese Macaques on the island of Koshima. When they were being studied by primatologists in the 1950s, the researchers wanted to observe them more easily, so they used sweet potatoes to lure them from the forests to the shore (see Jablonka & Lamb 2014, 176-177; Hirata et al 2001 also describes these techniques). One of the young monkeys (named Imo) started washing her potatoes in a stream, which was effective in getting the sand and soil off. This technique was soon picked up by the other monkeys, who also started washing their food. This later carried over to other types of food as well, such as wheat, and the monkeys also started washing their food in the ocean once they discovered that they liked the salt it provided. The practice of washing food meant the group spent more time around the water, which lead to a roster of new play behaviours: diving, splashing, and jumping around in the surf.

It also led to the acquisition of new food items: older males began scavenging fish, and the group would collect octopus and other food items from tide pools. Thus, an intervention technique led to a roster of different behaviours exhibited by the group, and the behaviours were mutually reinforcing. The monkeys seemed to enjoy playing in the surf, which meant they spent more time there and thus tended to scavenge more seafood, and wash their food. And washing the food increased the chances the monkeys would come to enjoy playing in the surf. These changes were both heritable and persistent. Jablonka and Lamb (2014, 176) note that, despite only getting sweet potatoes from researchers perhaps twice a year, the food-washing and play patterns initiated by Imo have remained and are carried on by subsequent generations since Imo has passed away.

4.2.3 – Symbolic Inheritance System

The third and final inheritance system that I wanted to mention involves symbolic inheritance. Unsurprisingly, symbolic inheritance systems rely on symbols to copy or transmit information across generations. In alluding to this system, I have no intention of trying to give a precise definition of what a symbol is. That topic can be quite the intellectual quagmire, and ultimately is simply too broad a topic to be included here. However, most of the examples that I make use of in this chapter pertain to behaviour or epigenetics, so there is minimal risk of relying on a suspect notion of symbols. Mercifully, there are so many paradigm cases of symbolic inheritance that even without a precise definition, this inheritance system can be approached in an intuitive way. Language (both written and spoken) offers the most straightforward example of this. Stories, myths, beliefs, etc can all be transmitted to subsequent generations via language, whether it be through institutions like public education, books, or through family-related oral histories. Anyone who has ever heard of the Bible owes it to symbolic inheritance systems, as does anyone who has ever made use of the Arabic numerical system for math. Likewise, pop culture dances can persist through time as symbols: most folks in North America (perhaps further afield as well) recognize the Macarena, even if they don't remember all the moves, who the song is by, or many of the lyrics. Likewise, many would likely understand a reference to Alfred Hitchcock's film *The Birds*, even if they haven't seen the film. With technology (Cds, mp3, etc) we can transmit songs, text and

cultural information to people on the other side of the world, or again to subsequent generations provided that medium of transmission is still in use. Importantly, these systems also allow the transmission of latent information, unlike behavioural inheritances. For example, if no one in your family is a carpenter, you can look up information about basic woodworking. You do not need to learn this information by observing or imitating someone who is actively doing carpentry.

4.2.4 – Implications of Multiple, Overlapping Inheritance

My goal in bringing up these other inheritance systems is not to claim they are brand new additions to the conversation. But there is a difference between a thing having been discussed and its implications realized. Given the type of interactionism I am advocating for, highlighting these multiple (and non-genetic) inheritance systems is useful in fleshing out the multiple avenues by which a developing organism might access developmentally-relevant information. It is also possible for inheritance systems to overlap in many ways – for instance we might talk to a child while demonstrating some behaviour for it to copy. These two notions - the multiple avenues of developmental information, plus the potential for the inheritance systems to overlap in ways where it is hard to tell where one stops and another begins – help give a sense of what I mean by 'embedded organisms'. Emphasizing the different kinds of inheritance an organism can have access to within its developmental niche helps highlight the messiness and complexity of the world, where different kinds of inheritance can theoretically interact and be leveraged in different ways. Before we get to multiple inheritance systems interacting, however, I want to discuss a couple examples to help show why I think the existence of multiple inheritance systems blurs the distinction between acquisition and inheritance as the divide is traditionally thought of.

Epigenetics offers numerous examples of an acquired property becoming heritable. Primarily, this is because many epigenetic effects are quite sensitive to environmental conditions, especially stressors. As I discussed above and in previous

chapters, often the epigenetic response comes in the form of DNA methylation or chromatin marking. These responses in turn can alter the transcription rate of genes, and so affect gene expression. Since methylation patterns are heritable, one can thus try to argue that there are strong grounds for some environmental property leading to a new (and thus acquired) property of the organism. Behaviourally-driven properties could similarly be acquired and then inherited by subsequent generations. But if this acquired property is then heritable (where it is reliably reconstructed in ontogeny), then there is a case to be made that the distinction between acquisition and inheritance is quite blurry and indistinct. To make this argument, I rely on three examples: stress in mice, rabbit food preferences, and environmental stress in children influencing the cycle of poverty. The first two examples are intended to showcase relatively 'pure' examples of a given inheritance system (epigenetics and behavioural inheritance, respectively). I've chosen relatively simple cases that can discuss the target system in isolation, while still showcasing the developmental importance of the systems. However, the case of stress and cycle of poverty is more multifaceted. While there are important epigenetic components involved, there are also behavioural patterns (to say nothing of structural social issues) and so the case is a bit more messy. It does, however, show the complex ways in which these inheritance systems can overlap and influence each other.

4.2.5 – Heritable Behaviour in Mice

The first example involves mice, stress, and inherited behavioural phenotypes. The experiment is based around early-life separation of pup and dam, with additional sources of stress added as part of the separation condition. During the first two weeks of the pup's life, they were separated from the mother for a couple hours each day (Franklin et al 2010). The timing of the separation was made unpredictable, and when the pup absent the mother would be stressed in additional ways (either by taking a cold swim or by being briefly restrained). As with similar experiments involving rats and stress (ie see Zhang & Meaney 2010), the behaviour of the stressed mice changed, and the mothers spent less time nursing and grooming their pups. Their offspring - especially males - tended to exhibit depressive and anxious behaviours when they became adults. For present purposes, the most interesting outcome involves what happened when the stressed males

reproduced, since they seemed to pass on their stressed behavioural profile. Importantly, the pups still retained this stressed and anxious behavioural profile even when they were raised with normal (ie non-stressed) mothers. Even more crucially, the pups retained this profile despite never having contact with the fathers. Researchers then looked at the methylation patterns in the sperm of the separated males, and in the brains of their descendants. In both cases, they found changes linked with genes associated with neural disorders.

Another point worth noting is that, for the stressed mice, the epigenetic change was found in the sperm cells (See Franklin et al 2010; Jablonka & Lamb 2014). This is worth emphasizing because of the way it undermines the firmness of the germ/soma distinction that the recipe metaphor relies on. A property that emerges in development but also reaches the germ line is like burning a cake but also having the recipe structurally changed in such a way that future cakes will also be burned.

In short, these experiments around stressed mice show a couple important things. First, it shows that future generations of mice can inherit an epigenetically-influenced behavioural profile without ever having contact with the parent from whom the behavioural profile is inherited. Second, the epigenetic changes are, in turn, induced by stress during development, which seems to 'biologically embed' (Aristizabala et al 2020) the effects of the stress via epigenetic mechanisms. This is a big deal, because the implication is that some acquired property (such as stress and its epigenetic effects) can both drive the development of a behavioural profile for the individual in question, but can also be inherited by offspring even if they don't have behavioural contact with the specific parent. This offers a case where one cannot easily pass off the development of the behavioural profile as the mere effect of learning.

4.2.6 – Behavioural Inheritance in Rabbits

Next up we have an example that more explicitly revolves around the behavioural inheritance system. A set of experiments were performed on young rabbits, looking at how food preferences were acquired. Rabbit mothers tend to be away from the nest most

of the time, and so the young only encounter her for about five minutes each day for nursing. The pups are also weaned when they are four weeks old, so there is not much time for teaching or extended maternal contact. There are a few ways in which the mother can instruct such young pups on what is good for them to eat, however. A study fed pregnant rabbits juniper berries (which can be part of their wild diet). Once the young were weaned, they clearly preferred juniper berries to standard lab food, though they'd had no direct contact with juniper berries in any way (see Jablonka & Lamb 2014, 160-62, citing Bilkó et al 1994). It seems that enough chemical cues reached the young while in utero (or while suckling) to help communicate that these berries were edible. Similar results were found when you took the pups of a regularly-fed mother (non-juniper diet), and were given to a foster mother who was fed juniper berries. During the weaning period, these step-pups were given a choice of standard lab food or juniper berries, and they preferred the juniper food. So it seems there can be both in-utero and suckling pathways for the transmission of dietary information. Another mechanism seems to involve the breath and saliva of the mother (especially in rats), and the mother's fecal matter. For rabbits, after the weaning period, the mother will deposit a few fecal pellets outside the nest before leaving. The young eat these pellets, which also contain cues about what she has been eating. When the above experiments were repeated, the fecal pellets of normally-fed mothers were replaced with pellets from a juniper-fed mother. The young again expressed a preference for the juniper diet.

All of these results showcase behavioural inheritances, and again show how an acquired trait can become heritable. (It is behavioural because the behaviour of the mother seems to be what is driving the availability of dietary information to the young rabbits). For the mother, prior to the experiment beginning, she would have been fed standard lab food. The change to juniper berries is thus an acquired property for her, but its one that her young seem to inherit through some combination of uterine chemical cues, suckling cues, or from eating the mother's fecal pellets. Regardless of the transmission vector, some of the mother's behavioural patterns (either 'standard' or acquired) are clearly inherited by her pups. Indeed, information about what to eat is vital to their survival, and so adapting to new areas or new sources of food depends on the ability of an acquired trait to become heritable.

4.2.7 – Stress in Humans; biological embedding

As Aristizabala et al (2020) put it, early childhood experiences can impact future developmental outcomes through what they call 'biological embedding'. This means that an organism's experiences contribute in some way to make changes to the function of some system, where these changes have consequences for development, health, behaviour, and the like. This process of biological embedding has been studied with respect to early childhood stress, deprivation, and the effects of low socioeconomic status (Pepper & Nettle 2017). However, unlike the previous two examples of 'pure' inheritance systems, this example is likely much more messy, with various feedback loops and overlapping aspects. This is because there are not only epigenetic effects associated with early-life deprivation; there are (statistically speaking) behavioural, endocrine and epigenetic differences associated with major stress in early childhood. Many aspects of the resulting 'behavioral constellation of deprivation' has been extensively studied, such as the tendency to save less money and have higher debt ((Blanden & Gregg 2004; Chowdry et al.2011), earlier sexual activity and pregnancies (eg., Imamura et al. 2007; Johns 2010; Nettle 2010a; G. D. Smith 1993), and less general involvement with their children such as reading to them, breastfeeding, etc (eg (Hango 2007; Kiernan & Huerta 2008; Kohlhuber et al. 2008; Nettle 2010a).) As such, there is clearly more than one factor that is different between a family experiencing deprivation effects and one that is not. However, my current efforts are not to give an exhaustive account of childhood deprivation. Rather, my intent is to point to known factors within this web (especially epigenetic and behavioural factors) that serve as examples for how some acquired phenomenon (early childhood deprivation) can have long-lasting and heritable effects.

One of the developmental effects of early deprivation emphasized by Pepper & Nettle is the suggestion that deprivation leads to a 'live fast, die young' form of life history theory as the adaptation to local environment during the course of development. (Importantly, Pepper and Nettle are not claiming that the behavioural constellation of deprivation is always adaptive in contemporary society). This ontogenetic calibration of the life history can take many forms. One such example is behaviours of impulse control, where immediate outcomes tend to be prioritized over more distal ones (Pepper & Nettle

2017). The suggestion is that immediate outcomes are prioritized because folks in lower SES tend to have less control over their surroundings, and so cannot ensure that they live to reap deferred rewards. This extends beyond the individual to the neighbourhood, assuming the deprivation is shared. Deprived neighbourhoods also tend to exhibit future-limiting characteristics, such as lower levels of trust, social capital, cooperation, and the like (Pepper & Nettle 2017; see also (Drukker et al. 2003; Drukker & van Os 2003; Hill et al. 2014; Schroeder et al. 2014). But since society tends to value deferred rewards (ie education, ability to save money, etc), the cycle of deprivation can be behaviourally reinforced through participation in these types of behaviours that are not rewarded in the larger social framework, thus limiting future social movement.

Even if social movement is achieved, and one's adult SES differs from one's childhood SES, it may not be smooth sailing just because one has achieved a higher SES. Childhood SES (cSES) shows a strong statistical link with adult health outcomes, especially the more one ages. (ie Nettle & Bateson (2017) found that the parameter estimate for cSES to adult health was larger at age 42 than it was at 23). There was also a 'silver spoon' effect, where high childhood SES seemed to protect against the potential adverse effects of adult SES. Miller et al (2011) have suggested that early life stressors generate proinflammatory tendencies, poorer health behaviours, which drives chronic inflammation and later-life disease. They identified three mechanisms by which early-life stressors can be biologically embedded in this way: 1) epigenetic changes, 2) posttranslational modification, and 3) tissue remodelling (2011: 975-78). The epigenetic changes include cytosine methylation and chromatin remodelling, which tend to affect the rate at which a methylated gene gets transcribed, and thus influences gene expression. Posttranslational modifications are chemical changes to proteins after they have been synthesized, typically by having additional molecules such as phosphate (a salt) or ubiquitin (a protein) attach to one or more of the amino acids that make up the synthesized protein. These additional molecules can alter all sorts of things, such as accelerating the degradation of the protein, change its ability to pass through membrances, or alter how it binds to other molecules (2011: 976). For example, the phosphate attachment to amino acids on the GR gene has an influence on cortisol-mediated signalling, making cells much less responsive to cortisol signals. This type of

inhibited cortisol signalling is also a characteristic of childhood stress, and is often seen in people who experienced trauma at a young age. Finally, there is some evidence to suggest that when exposed to stress, certain tissues undergo a restructuring that can change functional implications over time. One such example is exposure to cigarette smoke, with tissue remodelling being a driving part of airway disease (Miller et al 2011; Prescott 2006). Overeager immune response can also feature as part of this process, whereby local injuries from smoke trigger an immune response that tries to repair the damage, but instead remodels the tissue in a way that reduces resting lung function and makes the airway more responsive to stimuli and irritants in the future. Miller et al also point to findings that show similar tissue remodelling in the brain of rats that had been separated from their mothers, and they suggest that childhood stress could also influence the functional connections between the autonomic nervous system and the lymphoid organs that produce immune cell and respond to microbes. Thus, by drawing on existing research programs (using both animal and human models), the authors suggest that there are at least three ways that we have seen the effects of stress get biologically embedded in an organism, such that it makes a functional difference to the life of the organism.

The main point in emphasizing these three processes of embedding lies in the way they can affect health outcomes later in life. When stress gets embedded in monocyte cells (white blood cell) this way, they mount excessive inflammatory responses to microbial triggers, and moreover they are less responsive to hormonal inhibitory signals, which fosters a state of chronic inflammation in the body, which in turn is central to a number of chronic diseases of ageing (2011: 965). And critically, Miller et al noted that studies of the relationship between childhood SES and adult morbidity “need to establish that childhood SES is not simply rendering people more or less advantaged in adulthood” (2011: 962). Given their model, however, Miller et al do not think this worry obtains. Instead, they suspect these features of embedded stress are part of a predictive adaptive response (PAR), which is drawn from behavioural ecology. The function of a predictive adaptive response is to change and organize physiology such that the organism is better able to meet the ecological demands of its local niche. As such, the genome and other developmental processes are responding to local environmental cues in order to tailor the

phenotype, which offers another way in which the gene/environment system jointly produces certain outcomes.

4.2.8 – Summary & Wrap-up

The stated aim of this section has been to show three things. First, that we can understand organisms as possessing multiple kinds of inheritances beyond genes. Second, recognizing that organisms have multiple, reliable systems by which developmental information can be inherited allows us to see with greater clarity how overlapping and intertwined all these systems can be. Third, these exogenetic systems (and especially their interactions) do not respect the traditional acquired/inherited framework. This disrespect can be seen in two ways. First, some forms of acquired traits can go on to become inherited. This implies that, even if there were a hard distinction between acquisition and inheritance, that distinction is primarily temporal since yesterday's acquisition can become tomorrow's inheritance (so to speak). Second, and perhaps more crucially, no phenotypes or phenotypic traits are inherited wholesale; they all develop contingently in ontogeny using the sets of interactants made available to the organism via multiple inheritance systems. In this sense, both acquired and inherited traits are (re)constructed during ontogeny, and the development of either kind of trait is dependent on the ways that the various inheritance systems present themselves within the developmental ontogenetic niche, and the way they could potentially scaffold development. But if development is contingent in this way (and we're not sneaking in some sort of preformationist notion of inheritance), then all traits are contingently (re)acquired during ontogeny, and there is not a big difference in how each type of trait comes about. And even if we hold to some sort of historical view to preserve the distinction, where there is a difference between some trait that has been replicated across several generations and some entirely new trait, it is unclear that this move would accomplish much. If yesterday's acquisition can be tomorrow's inheritance, but both types of traits emerge in ontogeny through interactions within the niche, then even a temporal approach does little to separate the two in a non-heuristic way. In such cases, thinking temporally would, at most, be a tool for researchers to track how long some trait has been present in a given population, assuming we are interested in trait stability or emergence at the population

level. For the purposes of studying how traits develop, there is no practical difference between acquisition and inheritance, since both make use of the same developmental systems and are contingent in the same way. There will be more to say about this argument in the subsequent section where I will examine how the feedback loops between things traditionally categorized as acquisition and inheritance seem to interact. However, I allude to it here in order to help convey where my overall argument is going.

4.3 – Feedback Between Components of Categories

To help make the discussion of categories a bit less abstract, I again want to focus on a few examples. So as to not merely beat the same drum again and again (at least not quite so much), I am making use of different examples from the previous sections. There are two cases that I have in mind for this section: 1) genetic assimilation, and 2) adaptation by mutation. I take the case of genetic assimilation to show the interconnectedness of the many inheritance systems that typically comprise the ontogenetic niche. It shows, I argue, that an acquired trait can become supported within the genome and so enhance the reliability of the trait's (re)construction during ontogeny. Thus, the examples shows that the acquisition of some property can drive what is available to be inherited, and in turn the inheritance systems can make the trait easier to acquire in subsequent generations. The case of adaptation by mutation acts in a similar fashion. To make that case, I highlight research that indicates mutations are not always random as previously thought, and instead may be propagated for an organism when in stressful environments. Acquiring new mutations as a means of spurring adaptation still relies on the extended inheritance systems to pass on the relevant interactants and construct the trait in question. Both examples point to the two categories interacting as a form of adaptiveness. If the processes and properties categorized as ‘acquisition’ and ‘inheritance’ are interactive in a co-determining way, this helps explain why the thought that nature maps onto inheritance and nurture maps to acquisition goes so far awry.

4.3.1 – Genetic Assimilation

First, let's start with genetic assimilation. Genetic assimilation is an old term put forward by Conrad Waddington in 1942, which refers to the ability for an acquired trait

or character (as responses to environmental challenges) to become inherited thanks to natural selection. This is making the same functional point as the Baldwin Effect, which is also concerned with how natural selection could enable learned responses to become inherited. (Waddington apparently maintained that there was a conceptual difference between the two, but in my argument they serve the same functional purpose, so I'm less concerned about the differences between them). There are two points to be made here. The first is that both genetic assimilation and the Baldwin effect suggest that acquisition can drive inheritance, given stable selection pressures. That is to say, when a new challenge is faced (and is a persistent selection pressure), the organisms that learn how to respond appropriately will be more likely to survive. Over time, however, it is costly for the organism to have to learn the appropriate response every time, so the response might be canalized such that very little impetus is required to bring it about in the future. The learning and acquisition comes earlier, and over time the mechanisms of inheritance allow the genome to 'catch up'. On this model, the behavioural component drives the genetic component, resulting in the genome reducing the cost of the behaviour and freeing up that energy for different behaviour (recall the metaphor I introduced last chapter about the reactive genome). The capacity for more behaviours to be added over time as the genome reacts and reduces the cost of learning is called the assimilation-stretch principle by Avital and Jablonka (2000). The second point is that for all of this to work, the categories of acquisition and inheritance must interact. Indeed, in the face of constant selection pressures, adaptation requires that they interact, since requiring constant learning just to face one challenge is very costly. The implication here is that today's 'nature' could be yesterday's 'nurture', so to speak. If the survival of a population depends on acquisition and inheritance interacting, then it becomes less and less clear how nature and nurture are supposed to separately map onto these categories in a meaningful way.

Some examples may help make this more clear. Waddington's (1942) own example involved the ostrich and its ability to form a callus on its underside (for when they squat). He argued that previous ancestors couldn't form a callus as easily, and so young birds would suffer more than contemporary ostriches along this metric. However, organisms that could callus more quickly would have enjoyed an advantage, and so over

time this response became canalized such that only a small stimulus is needed to induce a callus. The selection pressures remodeled the epigenetic landscape to canalize this response in the face of persistent selection pressure. This reached a point when no stimulus was needed, and the assimilation was complete; the acquired characteristic had become part of an inherited phenotype. In this case, again we see behaviour (squatting) driving what becomes incorporated into the ostrich genome through inheritance systems, with the genome serving a reactive role.

A second example involves lactose intolerance. Durham (1991) has done interesting work looking at genetic changes in response to culture, and the coevolution of genes and culture. Many places have used milk products as a source of food, but relatively few have relied on fresh milk as a food source. Using fresh milk as a food source has costs, however: you need the enzyme lactase to be able to receive nutritional benefit from it, and for most of the world there is relatively little lactase in their intestinal tract. People who are able to drink milk can do so because they have a variant allele of the gene associated with producing lactase, and the regulation of this gene means they keep producing lactase into their adult years rather than have production slowed as they left childhood. Durham associates this genetic change with cultural changes; these genes show up in populations that tended to rely on dairying (mostly northern Europeans, and parts of the Middle East and Africa, along with their local or overseas descendants). However, not all dairying communities developed this allele, since a dairying society can rely on milk products for various foods (cheese, yoghurt, etc, which require little lactase to be able to digest) without also drinking milk. The main driver of this difference seems to be cultural and historical, as the cultural circumstances will influence how milk and milk products are treated as food sources. For instance, milk products are very common in the Mediterranean region, but primarily come in the form of cheese and yoghurt due to their cultural histories. By comparison, roughly 90% of Scandinavians have this gene, and there's several reasons why this might be the case. First, lactose helps facilitate calcium uptake in the same way vitamin D does. The further north you go, the more common it is to have periods with less sunlight and more incentive to cover exposed skin against the cold, which reduces vitamin D absorbed from the sun. So drinking milk would have helped these communities prevent calcium-related diseases that they otherwise

might have been prone to (as well as being a good source of energy). This is compounded by having local myths and folklore involving cattle, as Durham discusses how cattle become more important to northern-European folklore the further north you go. They even feature in creation myths, where they were the first animals of creation, and survived to create milk, which was a source of nourishment and strength the giants and gods alike. Having cultural access to this mythology highlights milk as an important food source for the young generation, which helps increase the odds they will drink it. This means you have biology and culture co-evolving, as each drives change in the other. Food scarcity and ease of access to milk as a potential food source in a dairying community means that those who could digest milk enjoyed an advantage over those who couldn't. As a result, the gene for lactase production spread and became assimilated, and folklore helped entrench the cultural importance of milk in those communities, meaning the gene was unlikely to be weeded out while the population frequently consumed milk. What was initially an acquired trait of drinking fresh milk (a behaviour) lead to an heritable genetic difference for populations that engaged in this behaviour, along with a complementary culture that values this behaviour for both nutritional and cultural reasons. The categories interact precisely because of the developmental context and the way the extended inheritance systems overlap with each other. A child in a milk-drinking culture can draw on all of the mythologies and values attached to a given behaviour, but also inherits all the genetic interactants to ensure the stability of the phenotype.

4.3.2 – Developmentally-Induced Mutations

To round out this chapter, there is one more case I want to discuss where acquisition and inheritance interact in interesting ways. The case is mutation, especially adaptation by mutation, where a mutation is a change in a DNA sequence that is heritable if it takes place in the germ cells. This final case comes from a relatively recent book by James Shapiro (2011). In his book, he presents an argument that targeted, developmentally-induced mutations are a rule rather than a exception. This is important and a stark divergence from the standard view on mutation. For positions like the selfish gene, mutations are taken to be random, and specific to DNA rather than influenced by developmental processes (since this would break down the recipe metaphor). Suggesting

that mutations might be targeted in some way evokes spectres of both Lamarckism and some sort of Intelligent Design. However, there are examples of things that seem to be targeted (and heritable) mutations, which serve as examples for how some acquired trait can be heritable in very important ways.

The first example involves P element targeting in *Drosophila melanogaster* (the fruit fly), and is drawn from the work of William Engels (2007). A P element is a kind of transposable element, which means it's a DNA sequence that can move from location to location within the genome, copy & paste itself into a new sequence, and thus behaves somewhat like a virus. These transposons are virulent enough to the degree that they have taken over all wild strains of the fly. More to the point, while P elements are found in all wild strains of *D. Melanogaster*, they are not found in lab strains. This is because the P elements are a relatively new occurrence, and probably only arrived in wild strains some 100 years ago. (Jablonka & Lamb note that the vector by which they arrived is unknown, but likely was a virus or mite (2014, 390)). Lab strains, however, were started in the early nineteenth century, meaning their cultivation began prior to any exposure to P elements.

Crossbreeding wild and lab strains shows remarkable results. If a wild male is bred with a lab strain female, the resulting offspring exhibit what is called hybrid dysgenesis. As the P elements from the wild strain begin jumping all over the place in the offspring, they cause mutations at a much higher rate, and rearrange various chromosomes, leaving most of the offspring sterile as a result. (The sterility, higher mutation rate and chromosome recombination are all collectively symptoms of the hybrid dysgenesis syndrome). Moreover, the P element's ability to target host sites is also remarkable. As Jablonka and Lamb (2014, 391) remark:

> *"The researchers [Cheng et al 2012] have found that P elements insert preferentially into the control regions of genes, but, although there are a few hot spots, most insertion is relatively nonselective with regard to the genes involved. The exception is when P elements carry regulatory sequences from certain genes that are involved in laying out the fly's body plan. Then, they frequently insert in the vicinity of the same or a*

> *related resident gene. This "homing" process is based not on DNA homology but on the chromatin structure in the region of insertion. Consequently, the chance that the inserted gene will become involved in the regulation of the resident gene is increased: the insertion is not only geographically semi-targeted, it is also functionally semi-targeted."*

The point here is that the P element is able to preferentially target some sites, and shows some sensitivity to certain genetic functions and chromatin structures. Cheng et al 2012 found that homing is mediated by interactions between proteins that are bound to the homing fragment and proteins that are bound to the targeted region. The implication is that, because the P element exhibits this preference, it's not always merely random in where it strikes; once it can bind to a small fragment of DNA it seems to be able to use that to facilitate further targeting in a preferential manner. That means the accompanying mutations it causes are not random either, since they are the result of targeted strikes.

This is not the end of the story, however. If P elements were this dangerous, then surely no wild strains of fruit flies would remain. Luckily, there are epigenetic evolved responses that control the ability for P elements to proliferate, and these responses also exhibit targeting.

In the above example of hybrid dysgenesis, a wild male bred with a lab female produces sterile offspring. However, the converse is not also true. A wild female bred with a lab-strain male produces fertile offspring. The reasoning is that in the former case, the lab female does not have the evolved response of the wild population, and so she is vulnerable to the P elements of the wild male. The difference seems to lie in the epigenetic inheritances both mothers bequeath to their offspring.

To stay alive against P elements, the wild flies have evolved piRNA. piRNA (piwi-interacting RNA) is generally used to convey epigenetic information across generations, especially certain kinds of defenses. piRNA is quite active in the germ line, and its primary function is to silence transposons that would otherwise cause damage to future generations. The wild flies have lived because their piRNA is able to silence

transposons like the P elements, and thus prevent their harm. Given that the lab-strain flies are so vulnerable to P elements, it's clear that their ancestors didn't have the piRNA system (at least not with the ability to target transposons in this way), which means it's come online since P elements started spreading in the last 100 years or so, and it's able to target P elements well enough to keep the population alive and fertile. The lab-strain female doesn't have access to this inheritance, so her offspring are vulnerable. The wild-strain female, however, passes on the piRNA and corresponding ability to suppress harmful transposons through her eggs.

Both of these examples (the homing capacity of P elements, and the relatively rapidly evolved piRNA defense system) are made more effective through their ability to target. For P elements, their ability to use small fragments of DNA (at least 1.6 kb of regulatory DNA (Cheng et al 2012)) to home in on a vulnerable target has helped them become present throughout the whole wild population. Likewise, it is the targeting capacity of the piRNA defense that has allowed the wild flies to survive and remain fertile. And given the short time period between the estimated onset of P elements and now, for such an effective defense it came about quite quickly. For Shapiro (2011), Jablonka and Lamb (2014), examples like this strongly suggest that "targeted, developmentally-induced mutations are the rule rather than the exception" (2014, 390). For my purposes, examples like this stress two things. First: the importance of development within this overlapping and mutually-determining system that I am trying to describe. Second: it highlights that for the fruit flies to have survived, the line between acquisition and inheritance must be blurry. It must be blurry in the sense that while we can understand acquisition and inheritance as separate categories, organisms rely on their interaction, and on the ability for acquired characters to become heritable (especially in a way that targets a solution to some novel and persistent selection pressure). It would have been far too costly for each generation of fruit fly to have to mutate its own defenses against transposons, but precisely because some acquired traits can be heritable, the population was not wiped out. This outcome has been great for the population of D. Melonagaster, but it is worse for the Nature/Nurture distinction, since the survival of the species seems to have depended on the distinction between acquisition and inheritance being very thin (if not practically absent). And that theme, where there is effectively no difference between the opposing

parts of a dichotomy, forms the main thrust of my argument in the next chapter, where I attempt to tie all these threads together in a way that dissolves the Nature/Nurture distinction by demonstrating its incoherence.

4.4 – Summary & Moving On

This chapter has looked at the acquisition and inheritance dichotomy, from the standard mode of thought that has contrasted these as opposites, to the way that some forms of adaptation and development seem to require significant overlap between these ostensibly-contrasting categories. The traditional opposition of acquisition and inheritance, I have argued, is a part of how Good Biology and Bad Biology are separated; proper Darwinian thought from foolish Lamarckian, respectively. Drawing on Jablonka & Lamb, I have showed that there are many inheritance systems – epigenetic, behavioural, and symbolic – and there are multiple cases where the inheritance systems overlap or work to allow the inheritance of some newly acquired property. As such, I have argued that there is strong evidence that acquired characters can be heritable under the right circumstances. Additionally, I have argued that since acquisition and inheritance overlap in the ways I have described, there is no strong mapping from nature to inheritance and nurture to acquisition in the way that the nature/nurture framework implies. As with previous chapters where I argued for the dissolution of the dichotomy, there seems to be little practical difference between traditional acquisition and inheritance, as traits are not inherited wholesale, but rather are constructed in ontogeny. Thus, for the developing organism, the traits come into being as a part of development, regardless of whether the traits are supposed to be inherited or not.

Far from the recipe dictating the form of the cake, the metaphor seems to work the other way around as well. Following the Central Dogma, the recipe metaphor assumes a one-way causal flow of information, where development is (partially) a downstream effect of upstream information. However, many of the examples I gave showcase the reactive genome, where it is behaviour and development that drives the contents of inheritance and genetic assimilation. Given the opportunity for feedback effects in this way (which in turn can be reconstructed in future generations), acquisition and inheritance seem less of a dichotomy and more parts of a complicated, overlapping

developmental system whose parts can be difficult to parse into wholly distinct categories. In the next chapter, I will have more to say about all of these overlapping systems discussed in previous chapters, and how their extensive overlap and co-determination shows us that nature and nurture are incoherent categories.

Chapter 5: Dissolving Nature/Nurture; Objections and Replies

Let's review. A lot of ground has been covered in the preceding chapters, so Section 5.1 will quickly re-articulate what I take to be the main threads from the preceding chapters. Afterwards, the focus of this chapter is to tie these threads together, with two central aims. First, in Section 5.2 I make the case that organisms are embedded entities, with niches that are co-created by them and, in turn, help shape the organism. This notion of embeddedness is part of my pitch for the metaphor of irreducible interaction, where 'irreducible' is understood in the sense of nonlinear coupled equations, as articulated in Chapter One. Arguing for these metaphors of embeddedness and irreducible interaction is the first main conclusion of this chapter. For the second central aim, I argue in Section 5.3 that these dichotomous domains (ie nature/nurture, gene/environment) I have been examining are irreducibly interactive, there are not clean mappings and distinctions between nature and genes, nature and inheritance, nature and wholly internal causation. The same can be said for nurture and the properties it tends to get grouped with. The point is that these are not discrete categories, but rather they overlap in many ways, and co-determine each other. Given this co-determination, nature and nurture are incoherent concepts on their traditional way of being construed.

After the argument for incoherence, I will address the most likely source of counterarguments to my overall project: twin studies. In Section 5.4, I discuss what twin studies are, and the insights they purport to offer. I follow the discussion of these details with a critique, and show how the logic of twin studies depends entirely on conservative interactionism and its wrong-headed understanding of development. Finally, Section 5.5 offers some supplementary observations about understanding the Nature/Nurture framework and what we might make of it in the postgenomic era. In short: Section 5.1 offers a chapter summary, and Section 5.2 advances metaphors about embedded organisms and how that pertains to the kind of interaction I'm interested in. Section 5.3 puts forward my main argument as to why the Nature/Nurture distinction is incoherent;

Section 5.4 addresses describes and critiques twin studies, both as a source of knowledge and as a source of potential counterexamples to my project. Finally Section 5.5 rounds out the chapter with some supplementary comments on the Nature/Nurture distinction and where we might go from here.

5.1 – Chapter Summaries

In Chapter 2, I noted that there are many kinds of interaction discussed across the sciences, and each can do its own argumentative lifting. Categories of interaction that I examined include: 1) causal (ie billiard balls); 2) interaction as exchange (quantum mechanics); 3) statistical interaction (especially two-way interaction of independent variables); and 4) interaction as coupled equations (ie dynamical systems). Each of these kinds of interaction has different implications for whether the interactants are empirically and conceptually separable. While I think it likely that all these kinds of interaction will be involved in development as a whole, I am particularly interested in the statistical and coupled kinds of interaction since they offer two important conceptual points. First, they allow us to formulate a well-understood sense of dependence, and second, they offer a sense of what it means for a system to be irreducible. (While being irreducible also implies a certain kind of dependence, I think it is still worth including and discussing statistical interaction because two-way statistical interactions are understood well in statistical terms.) Special focus on statistical and coupled interaction is something I rely on going forward, because of how they help us understand dependence and irreducibility.

In Chapter 3, I discussed the idea of genetic interaction and the 'meaning' of genes. Specifically, I pushed back against conservative interactionism's gene-centred approach and pointed to evidence that suggests two related conclusions. The first is called the extended genotype thesis, which holds that the 'genetic context' (regulatory systems, cues to which the overall genome responds, etc) extends beyond the body since many regulatory systems depend on environmental cues. My second conclusion was that since these regulatory systems are so environmentally sensitive and reactive to external cues, the gene/environment dichotomy is best thought of as behaving like a coupled system. Given that coupled systems are irreducible (as discussed in Chapter 2), this also applies to the gene/environment system as a part of development. (The system is irreducible in

the sense that knowing the value of one sub-variable – such as the genome - does not get you very far in determining the overall value of the other coupled variable(s) (or in solving the coupled equation, to put the same point in slightly different terms). All of the variables/equations need to be solved together, and not in isolation. This means that the emerging phenotype is dependent on the values of the sub-variables, which is to say the specific way in which the interactants actually interact co-determines the emergent phenotype.

In Chapter 4, I examined the putative distinction between acquisition and inheritance. Over the years there have been a few attempts to adopt new terms instead of acquisition and inheritance, but despite any terminological changes the underlying structure has remained. This structure treats inheritance as being tied to inner nature, while acquisition is just a product of variable nurture (Oyama 2000a/b). My work in that chapter aimed at establishing two conclusions. First, it seems like the crossover and interaction between acquisition and inheritance is necessary for the survival of species in the face of persistent selection effects. Behaviour drives the acquisition of new skills, but many mechanisms and pathways allow for the acquired property to become inherited. This points to a way in which the causal force can flow from 'outside to inside' (behaviour to genetic change), as opposed to the standard conception under conservative interactionism, which emphasizes 'from inside to outside' causal force (from genes to behaviour, genes to traits, etc). Second, based on the available evidence, the acquisition/inheritance distinction does not to match the complex ways that traits can become available to an organism and reconstructed across generations. Moreover, the way that the distinction breaks down seems to threaten the separability of Nature and Nurture as distinct categories.

5.2 – Metaphors & ‘Embeddedness’

In my view, irreducibility comes as a result of our embeddedness and integration. It is largely because of the latter that the former arises. For instance, when discussing what it means to be irreducible, I relate it to coupled equations and dynamical systems (both of which are highly integrated). In these examples, multiple equations or factors are conjoined in a way that they cannot be solved separately because the values of each

equation, variable, etc are co-determining. The co-determination of variables means that the removal of one variable also serves to alter the values of the other variables involved, and likely the system as a whole if the variables in question are important enough. Thus, whether or not some system is irreducible is contingent on its setup. If it is set up such that there is extensive overlap in its parts, which results in co-determination of given variables, etc, then the system is also irreducible in precisely the sense I have discussed. Ultimately it is an empirical question whether a given system is coupled in such a way that its parts are co-determining.

As described, for a system to be irreducible is for its parts to be coupled in such a way that they are co-determining. It is also worth discussing the language involved in describing a system in this way, as I have chosen the terms 'irreducible' and 'embedded' deliberately. I chose these two terms because I think they have useful connotations which help frame the overall picture in helpful ways. Ideally, I would prefer to see these terms (or terms that accomplish the same sort of lifting), at least instead of other metaphors that I think are problematic and undermine our ability to well-understand biological systems. For instance, throughout this dissertation, I have rejected the language of blueprints, hardwiring, etc, on the grounds that these terms do not match the empirical reality. They also carry insidious threads of preformationism into our conceptual frameworks, which helps recreate the Nature/Nurture framework that everyone agrees we should be moving beyond. By contrast, in earlier chapters I have spoken positively about metaphors of response and the reactive genome, because I think these metaphors help illuminate rather than obfuscate. I bring this up because I do not think that metaphors *simpliciter* are a problem in scientific thinking. I think it likely that they are necessary in science, as a way of helping to scaffold how we think about the world around us. However, to paraphrase Lakoff & Johnson (1980), the price of metaphors is eternal vigilance with regards to how they shape, distort and enhance our views. I've chosen the term 'irreducible' interaction specifically to resist the attempt to reduce traits and behaviour to 'mere' downstream effects of genes. Similarly, I do not think it helpful or illuminating to associate some traits primarily with genes and yet others primarily with the environment. As with the example of brown-headed cowbirds (West & King 2008), fixation on a set of behaviours being associated primarily with a putative closed genetic loop led to methodology that

obscured the actual developmental pathways that underpin mating behaviour. Investigations that took interaction seriously, and treated development as sensitive to the niche in which it takes place, have succeeded where a gene-centric approach has failed.

I do not mean that one cannot use reduction as a heuristic to make complex systems more tractable for scientific investigation. However, I hope that the metaphorical emphasis on the inability of a system to be meaningfully decomposed will still serve as a guiding principle even where some form of reduction is of practical use. Ontogeny requires that the systems are coupled, and my choice of language is meant to reflect that.

Similarly, I use the term 'embeddedness' to reflect the state of the parts of a system (or set of systems) that heavily overlap and co-determine their products. Given the evidence presented in the last two chapters regarding different (if often-overlapping) sources of inheritance, coupled with what we know about parental effects, niche construction, etc, I take the sum of this information to be evidence for the conclusion that humans are embedded in a niche that features many overlapping systems. The specific choice of the term 'embeddedness' is meant to convey two main sentiments. First, the colloquial usage of the term can mean that multiple objects are now part of a larger whole, and cannot be easily disentangled. Second, 'embedded' is usually a term that implies a second object or entity for the former to be embedded in: 'ie X is embedded in Y'. And sometimes, given how some things are embedded, we stop thinking about the components as though they were separate. I think this is a useful connotation, as I hope for it to help direct attention to the question of what sorts of things are we part of, namely extended systems of inheritance, our ontogenetic niche. This is a metaphorical attempt to help shift focus in the same way as the rhetorical question 'ask not what's inside your genes, but what your genes are inside of' (paraphrasing West & King 1987, 552). This choice of term is thus intended to reflect our inseparable ties to the overlapping systems that we create, participate in, and rely on. There are no genes absent an environment, and likewise no environment absent genes worth speaking of in terms of development. Recognizing this makes it imperative that we see the connections between things and how they form a larger system that cannot be cleanly decomposed as if it were modular or merely additive (ie nature contributes A, nurture contributes B, resulting in phenotype AB).The

Nature/Nurture framework (and conservative interactionism) both make the mistake of treating development as additive when it seems to be emergent. I am hoping that explicitly choosing metaphors that reflect this emergence will be a first step in moving forward; moving away from the mistakes of Nature/Nurture and towards a better understanding of developmental systems.

5.3 – The Argument for Incoherence

Thus far, my focus has been on irreducible interaction, and organisms embedded in niches within overlapping systems of inheritance and other developmental resources. This picture is clearly heading towards understanding development as the set of interactions that take place within the niche, such that development is an emergent property of the interactants involved. This is because the systems involved in the developmental interactions are irreducible in the sense that they are like coupled equations through their mutual sensitivity and co-determination. This picture is very much opposed to the implicit Nature/Nurture framework in the received views I have discussed. Additionally, given the evidence I have presented in the previous chapters for the extended (and reactive) genome, multiple sources of inheritance, how acquired traits can become inherited, and the way in which behaviour can drive inheritance, I think there is substantial evidence that the nature/nurture framework is not merely incorrect, but that it is incoherent because of all this overlap. In what follows, I will flesh out my claim, and say why I think this distinction is incoherent.

The argument for incoherence, put shortly, goes something like the following: since the empirical work suggests that developmental systems are extended and entangled (for example, the reactive and extended genome), they cannot be divided along internal/external lines in the way the nature/nurture framework suggests. The upshot is that any attempt to reference genes or inheritance as Nature will also 'spill over' the traditional boundaries of the Nature category to also reference entities and processes traditionally conceptualized as Nurture. The entwining and co-determination of the extended genome and environment is one such example. In making a more technical argument for this claim, I am going to need to draw on some philosophy of language and that sort of structure. The rest of this section is aimed at substantiating this argument, and

is split into two parts. First, I argue that the Nature/Nurture framework reads as two contrasting natural kinds, where each sub-kind or sub-level of the kind is related to the overall category by virtue of shared properties. Second, I appeal to Dummett's anti-realism of meaning. Through Dummett's work, I argue that the truth or falsity of Nature/Nurture claims relies on the methods we have for checking, and the sort of evidence that can be brought to bear since the categories are taken to have empirical extensions. Given the complex overlap of systems that I have been describing, traditionally-construed Nurture-type evidence may sometimes be drawn upon to evaluate a (traditionally-construed) Nature-type claim, and vice-versa. Since Nature/Nurture is traditionally construed as a dichotomy involving opposites, the effect is that neither category can shore up its boundaries against its opposite. The distinction thus collapses, and appeals to the framework (as traditionally conceived) are incoherent.

5.3.1 – Contrasting Natural Kinds

It is worth stating up front that I am not claiming outright that Nature and Nurture are full-on natural kinds, nor that they are explicitly thought of as natural kinds. That seems too strong a claim to me, and I do not need to make a claim that strong in order for my argument to still work. Instead, I am making a more specific claim about the way the terms are used in the context of their framework. My claim is that, even if no one explicitly claims nature/nurture are full-on natural kinds, they operate as if they were kinds (or at least are reasonably kind-like) within the framework and literature.

Let's be honest: I think it likely that a category named 'Nature' is probably often explicitly thought of as a natural kind. It's in the name, after all. For the purposes of my project, however, I think the way the terms are used is of much more importance than investigating how individual scholars explicitly thought of them. Thus, the claim in question to start matters off is the following: the use of Nature/Nurture (and related terms) is at least implicitly kind-like (ie is used in a kind-like way, regardless of whether it is explicitly considered a natural kind or not).

There are, of course, a number of ways of conceptualizing natural kinds. I do not intend to cover them all here, but will focus on weak realism. I have a few reasons for honing in on weak realism specifically. First, weak realism is a sufficiently popular position (Bird & Tobin 2018) that it doesn't invite accusations that I have cherrypicked some niche position just to suit my purposes. Second, given the large literature on realism and anti-realism in science, a recurring theme is that one of the aims of science is to make real claims about the world (whether this regards entities, structures, theories, or something else). Certainly, many scientists act as if their explicit aims are realist. As such, I think it is most charitable to interpret the claims about the Nature/Nurture framework as if it were aiming to make claims about the real world. I also think the argument in the following section(s) can be applied to an anti-realist framework, so I don't think the project is entirely contingent on an assumption of realism.

On a weak realist (or 'naturalist') understanding of natural kinds, one holds that there are certain groupings of things that are genuinely natural in some way (as opposed to being arbitrary, or arising because they track human interests). This view is not committed to the claim that we have located the right natural groupings, or even that all our current theories track these natural groupings. However, according to Bird & Tobin's (2018) summary of weak realist/ naturalist positions, there are often several criteria that one might have in order to think something qualifies as a natural kind. Potential criteria include: 1) members of a kind should have natural properties in common; 2) inductive inferences should be permitted by the natural kind; 3) where possible, natural kinds should participate in laws of nature; 4) there is a hierarchy of natural kinds; 5) kinds should be categorically distinct.

Of course, some of these criteria might be seen as more contentious than others, but others seem relatively intuitive. Even with an intuitive pull, folks may disagree on the precise details of the criteria. For instance, there may be some quibbling about what 'natural properties' the objects should have in common. For example, 'white objects' probably don't form a kind (Mill 1884), but objects that have a certain atomic number (such as hydrogen) arguably do. For the purposes of my argument, the criteria I wish to highlight are those involving hierarchy and categorical distinctness (as many people

recognize that applying natural laws to biology can often be difficult, and I don't want to assume the Nature/Nurture framework to be explicitly committed to that).

First, a few comments on natural kinds and hierarchy, since this will be important for my argument later. Ellis (2001; Bird & Tobin 2018), holds that asking for a hierarchy of natural kinds helps establish relations between concepts. If two kinds are overlapping in some way, then one must be a sub-kind of the other (assuming the two kinds are not simply identical). This helps preserve the distinction between kinds. Similarly, the requirement that kinds be categorically distinct implies that there cannot be smooth transitions between kinds (Ellis 2001), as this would (allegedly) make it more likely that the boundaries have been drawn by us rather than nature. Chemical elements make for a good example of this requirement: for neighbouring elements on the periodic table, they are classified by their atomic number, and each number is distinct. It is not as if one can have a smooth transition from, say, seventeen protons to eighteen (ie chlorine to argon). There is a sharp difference in number of protons, and this difference is reflected by the fact that we think of these as separate elements and (likely for a weak realist) are different kinds. In this case, the difference in kind-ness is tracking an intrinsic property of the two objects, namely the atomic number.

These criteria can be applied to the way that the traditional Nature/Nurture distinction operates, and how the various sub-categories within the distinction come to be aligned with one side or the other. First, I noted in earlier chapters that these two categories and their associated dichotomies are oppositional and exclusive in their definitions. The main crux of the matter is, I think, an internal/external distinction with regards to the location of (potential) causal force. Traditionally, Nature is taken to be internal, stable, and associated with genes and evolutionary/genetic history. The causal force is internal, with the internal teleology-as-genetic-blueprint aiming at certain favoured outcomes or expressions. The notion of having preferred outcomes, or ends that are aimed at, also lends a sense of stability to the range of potential outcomes. Genes, inheritance, and indeed the overall category of Nature thus have several important properties in common. First, they are either sources or describe sources of internal causation. Inheritance is taken to be a consequence of genetic activity and

replication/transcription mechanisms (which are also traditionally seen as internal to the body). Genes are also taken to be discreet entities (ie see the Human Genome Project's estimate of 25,000 distinct genes), with distinct combinations of nucleotides/ locations, meaning there isn't a smooth transition between entities (ie genes are distinct rather than blended together).

Recall the weak realist's criteria for natural kinds. First, members of a kind should have properties in common. Above, I argued that a central way of organizing the important properties in common for the Nature/Nurture distinction is based on where the components are located: either internal or external to the body. Location relative to the body, and being a source of causal force in development serves to unify the subcomponents. The kinds are also (traditionally) categorically distinct, in that they offer opposing sources of potential causal force. The components of each categorical kind are also related to each other in a quasi-hierarchical, and sometimes constitutive, fashion. Below, I also discuss ways in which I think there are also hierarchies or levels within each kind, which is related to another criteria for kind-ness.

For instance, the environment is, broadly speaking, everything around and external to the body: local conditions, availability of food, parental care, community and culture, etc. One's environment is deeply involved with determining what can be learned or acquired (ie hard to learn a given cultural practice if no one can teach it, etc). The term 'nurture' is the broad categorical name given to this collection of components – it picks out things external to the body that can influence development (typically care-related), and since there are several ways this can occur, the category that captures them all is fairly broad by necessity. Drought and food scarcity would be categorized as Nurture, but so would oral histories, parental attention, and the like, despite clearly being quite different sorts of thing.

A very similar list can be offered for the nature side of things, though with a slightly more limited scope (as not all internal processes and causes might directly relate to genes and inheritance, ie breathing, circulation, etc). For example, inheritance is an effect of gene activity, gene activity is a locus of internal causation that expresses some

sort of phylogenetic teleology, and the term 'nature' captures and refers to these things. Or, in terms of causal ordering, the story would be something like the following. Natural selection determines what information is encoded in the genes. And since information flows outward from the gene, inheritance is the ramification of goings-on at the genetic level (ie which allele(s) are part of fertilization, etc). There is a clear hierarchy of levels implied in this story, and a clear direction of causal force. Additionally, by virtue of nature being treated as a different kind of thing from Nurture, conservative interactionism seems to treat references between levels of a given category as operating entirely within the same kind. That is to say, referencing genes and transcription activity to describe the effects of inheritance is to just reference different levels of the same abstract kind.

For both categories, there is a way of ordering the sets of terms such that the relationship (broadly speaking) is as follows: category –> unifying property or properties –> main entity of interest –> effect of that entity. On this ordering of terms, Nature/Nurture are the categories; their unifying properties are internal/external sources of causal force; genes/environment are the main items of interest; inheritance/acquisition are effects of the respective entities of interest for that category. Given this grouping, using either category to make a claim about the world will involve explaining how the within-kind subcomponents apply to the phenomenon such that we explain how the phenomena obtains. For instance, when various evolutionary psychologists claim that human nature places constraints on the types of societies that we can easily live in (ie absent totalitarian force, etc), the claim is that something internal to us – our phylogenetic history and the continued conservation of these internal causal forces – somehow explains the notion that there are 'natural' ways for us to live. In this sense, the meaning of a claim about Nature or Nurture is an appeal to the components within the kind to point to something like a (probably causal) explanation. Invoking human nature to explain some contemporary phenomenon is just to appeal to the history of the information within our genes, and the ways our development aims at expressing this information. As a hypothetical example, competitive behaviour (or selfish, altruistic, etc – pick your favourite example) might be traditionally construed as natural (and thus belonging to Nature) if we think that this behaviour was adaptive at some point in our phylogenetic history, such that our development aims at producing competitive behaviour

due to how our phylogenetic history would have favoured genes that predispose this kind of behaviour.

5.3.2 – Dummett's Anti-Realism of Meaning

So I have made a case that Nature and Nurture are treated as different kinds of things, primarily based on an internal/external divide. But there is more to say about references within the category, and how this supports my argument for incoherence. To this end, in this next section, I introduce and make use of an account of reference and meaning, namely Dummett-style anti-realism. I have chosen this theory because I am interested in how the terms are used, especially across frameworks as the frameworks change over time. As such, a theory of meaning that takes a 'meaning is use' angle seems a natural choice, though ultimately I think my argument could work with a different theory of meaning as well. My argument for incoherence proceeds in the following way. Given the evidence I have presented in previous chapters for irreducible interactionism, I take many of the things traditionally associated with Nature and Nurture to be instead coupled together in a single, extended, developmental system. Since Nature and Nurture seem to be treated as distinct kinds in the way they are treated as opposing sources of developmental causation, and given the evidence that these categories are coupled in this 'interpenetrating' (Keller 2014) way, I argue that there simply isn't a stark divide between these 'kinds' in the way that the traditional Nature/Nurture framework presupposes. As such, attempts to reference the contents of the Nature category in any way beyond a mere heuristic will also reference the contents of the Nurture category (and vice-versa). Since these categories are opposed in their definitions, the resulting attempt at reference picks out both sides of an opposing dichotomy, and is thus incoherent because the evidence for each side of the dichotomy ends up dissolving the dichotomy. Now, that is several different kinds of claims in rapid succession, and so one might reasonably ask: 'is the point that the claims don't refer, that they don't pick at true natural kinds, or that their claims cannot be verified in some way?'. My response is that it is all of the above, since I think all of those claims stem from the same argument that I am making regarding incoherence. I will say more on this presently, but for now I see the matter in the following way: The central issue is that where the Nature/Nurture framework

traditionally sees two sources of developmental causation, much of my work in this dissertation has been to lay the groundwork for the claim that there are not two categories, but one. In collapsing the two categories into one, there is also a corresponding change in the way the sub-components relate to each other. Traditionally opposite categories are no longer opposites, but are instead related in a different, interpenetrating, way. In light of these new connections between components, it is more difficult to refer to within-category elements in the same way, since the components stand in a very different relation to each other. We can understand what a gene is in a vacuum, but the 'meaning' of a gene is understood completely differently. Where on the traditional framework, the meaning of a gene lay in its information (ie what it codes for), the evidence for irreducible interaction suggests that the 'meaning' of a gene depends on a host of factors, some of which extend beyond the body. Thus, an attempt to refer to genetic meaning no longer respects the traditional categories: it picks out elements that are both internal and external to the body, and our methods for checking this also do not respect the traditional divides of Nature/Nurture. That is my point: recognizing that the elements of the traditional Nature/Nurture divide are actually part of a larger, coupled, developmental system means that previous attempts to carve up the world have missed the mark in some important way. As such, the claims and meaning of the Nature/Nurture framework do not refer to the things they were previously thought to, but rather the empirical reality of the claims rely on components from opposing categories (ie Extended Genome and genetic 'meaning'). Thus, the difficulty for the traditional categories to refer, and to pick out only the elements traditionally associated with the respective category, are all based on the evidence that there is effectively little practical difference between the traditionally-opposing categories. I will say more on this below. For now, I turn to Dummett's work and to describe how I will incorporate it into my project.

Michael Dummett was a British philosopher, who published widely on philosophy of math, language, logic, metaphysics, and is especially well-known for his commentaries on the philosopher Gottlob Frege, as well as for introducing an anti-realist theory of meaning. Such a theory is, straightforwardly, to be contrasted with realism about meaning. Put briefly, realism about meaning holds that every sentence is determinately true or false. Importantly, this can be understood in an evidence-

transcendant way (Devitt 1983; IEP & Murphy n.d.; Wright 1981). This is to say that there is some fact of the matter about whether a sentence is true or false, even if we do not have the means to verify this fact (ie the truth-value transcends the evidence we have for determining it). By contrast, the anti-realist holds that there might not be a determinate truth-value for a given sentence, and instead, that recognizing the meaning of a sentence is to recognize what would count as evidence for it (IEP & Murphy, n.d.; Devitt 1983). This is a 'verificationist' approach, where the truth-value of the sentence is tied to its ability to be verified. There is, of course, a great deal of history around this divide that I am simply going to ignore in this project, as I am far more interested in making use of these positions than I am in resolving any outstanding disputes.

One might reasonably ask, however, why I am appealing to an anti-realist position instead of a realist one, and whether my argument turns on this choice. I do not think it does. And, as alluded to above, I think it is possible to make my argument work using a realist framework, though aspects of the argument might have to change. The main thing driving my choice of an anti-realist framework is that I think it is in line with my focus on use, rather than explicit intentions. For instance, in the section on natural kinds above, I mentioned that I am less interested in how individuals explicitly think about the Nature/Nurture distinction, and more interested in how the distinction has been used, and the sorts of argumentative lifting it is used for. Dummett's anti-realism has a distinctly Wittgenstinian flair (Devitt 1983), and uses a similar line of argument to Wittgenstein's famous declaration that 'meaning is use'. (After all, Dummett did consider himself a Wittgenstinian in his early career, and so incorporated some of this flavour into his own work (Dummett 1993)). As such, given my focus earlier was on the use of a framework, I think it makes sense to examine the framework's broader meaning in light of its use. Additionally, I think it is important to remember that I am not merely discussing language in general, but rather a framework that informs the contents of scientific theorizing. Verification is often taken to be an important part of science (ie Popper's work), and so I think it starts to look more reasonable to emphasize how the claims stemming from the framework might be justified (ie verified). I am not aiming at mere 'the cat is on the mat' linguistic statements, but rather at how claims about genes, development, and the like are understood. I think that is an important difference.

What, then, are the relevant details of Dummett's anti-realism, and what argumentative work do they do for my project? Again, let us return to the verificationist stance. In this sense, the meaning of the term is based on our ability to know when it has been confirmed or refuted. Additionally, for the anti-realist, the truth or falsity of the assertion is constituted by the methods we have for checking. As Murphy puts the matter: "For the anti-realist [...] there is no gap between what makes an assertion correct, and the most direct means we have of checking that assertion." (IEP & Murphy, n.d.). Dummett also holds that indirect means might be used as well, as we can apply our best methods available at some hypothetical time. Alternatively, another way of putting the same point is to say that to understand a sentence is to be able to recognize what would count as evidence for or against it. This can obviously be sensitive to time, place, and framework throughout history, since we might have different methods for checking at different periods in time. This is a semantics grounded in justification – to say that a sentence is true is to show that one is able to (and has the general ability to) recognize when a sentence has been verified or refuted. The verification or refutation, then, comes down to one's ability to parse what would count as evidence for either outcome.

5.3.3 – Overlapping Categories Leads to Incoherence

So that is a very brief overview of anti-realist semantics, but how does my own project intersect with all this? I submit that my previous chapters have catalogued evidence that developmental systems are not as simple as we once thought they were, and that they behave differently than was first thought. Jablonka and Lamb certainly seem to concur on this point, as much of *Evolution in Four Dimensions* (2014) points to how the results (both positive and shortcomings) of the Human Genome Project showed us the inadequacy of the received view of genetics. This means the overarching theory and structure of the framework by which we understand development is changing, as these inadequacies become recognized and addressed. If constructs from previous frameworks (ie Nature, Nurture, and their subcomponents) are going to maintain their place in the changing framework, then we need to understand what they mean. To know what they mean, we should know what sorts of empirical extensions they have, and (this is where

the anti-realism kicks in) thus what counts as evidence for a true statement about them. Previously, it might have been thought sufficient to appeal to an organism's genes or phylogenetic history as evidence for some behaviour or trait constuting part of its Nature. However, this evidence is no longer acceptable in this same way. We know that genes are not simple causal units, and the genome is better characterized as reactive rather than proactive (Keller 2014). Given the evidence I have put forward in the previous chapters, I submit that we also have good reason to think development consists in irreducible interaction, where systems transcend the body/environment divide. This means that different evidence needs to be provided (to use Dummett's phrase) in order to evaluate the truth of some sort of Nature claim. In this 'postgenomic' framework, we understand gene activities to be governed by mechanisms of gene expression. Some of these mechanisms for gene expression, however, include environmental cues or conditions, epigenetic changes, and the like - ie things that may transcend the body/environment divide. Thus, in the postgenomic framework, evidence for the truth of a Nature claim may also include the environment, developmental conditions, etc, since those factors contribute to any genetic 'meaning'. Thus, things traditionally-regarded as part of Nurture are now part of the truth conditions for Nature, and vice-versa. Acquired and inherited influences can, in turn, affect both gene expression and the Nurture side of things, which means they may be fair game as evidence for the truth of Nurture claims (ie licking and grooming behaviour in rat mothers affecting subsequent generations). The interpenetration of the traditional categories means that, given how we understand matters now, each category may need to rely on claims about its opposing category in order to determine whether a given claim is true.

Quite obviously, this picture I just sketched of interpenetrating categories is distinctly different from - and in conflict with - the traditional way of understanding the Nature/Nurture framework. As discussed in the section on natural kinds, the traditional understanding of Nature and Nurture is of sharply bounded categories that functioned as opposing kinds – each offering different causal force to explain traits and developmental outcomes. Given the evidence we have access to, I think traditional Nature/Nurture claims are incoherent in light of how we think development actually works. This is because the empirical reality seems to reflect that a traditional Nature-style claim cannot

be made without implicitly invoking its opposing category to determine the meaning and truth of the claim. The conditions for understanding such a claim transcend the category and depend on its opposite. To invoke one category is just to invoke the other as well, because the evidence suggests that there is no stark divide between the categories. The meaning of the respective categories, as traditionally understood, didn't survive the new evidence. Of course, we might understand in a heuristic sense (as a historical tidbit) what Nature or Nurture claims used to amount to, but under the contemporary framework their truth conditions will, often enough, include the opposing category in a way that renders the claim incoherent on the traditional framework. By including the opposite category in the truth conditions this way, the old categories are dissolved in favour of a larger one: the developmental system.

5.4 – The Twin Studies Objection

Thus far in this chapter, I have summarized the contents of my previous chapters, discussed the metaphors that I think are useful to adopt, and put forward an argument for the incoherence of the Nature/Nurture dichotomy. The next step is to respond to an objection that I anticipate my hypothetical critic has been muttering for the past several chapters with increasing irritation. Namely: 'but what about twin studies? Aren't they a good counterexample to the claims you're trying to make? '. For the sake of responding properly to this objection, I want to make sure I am examining the topic properly. I will first discuss what exactly twin studies are, and what sorts of insights we generally draw from them. Given the putative insights they offer, this motivates the objection being raised, and I want to be clear on what the nature of the objection is. However, I think that criticism based on twin studies ultimately fails to discredit my arguments, because I think the foundational logic of twin studies is flawed in precisely the same way that conservative interactionism goes astray. For that reason, I argue, we should be skeptical of twin studies, and any doubt that my arguments have raised against conservative interactionism equally counts against twin studies since they are based on the same underlying assumptions.

5.4.1 – So, What Are Twin Studies?

Twin studies are an experimental method that attempts to leverage the difference between monozygotic and dizygotic twins (identical and fraternal twins, respectively) in order to examine the relationship between heritable phenotypes and their genetic/environmental components (Nielsen et al 2012). The idea is to pick some trait(s) and compare frequency of phenotypes with this trait between monozygotic and dizygotic twins. The key difference is that monozygotic twins share 100% of their DNA (by virtue of being from the same cell), whereas dizygotic twins share 50% on average, in the same way standard siblings would. Since twins are often raised together and are the same age, they also putatively share the same environment in a way that siblings of different ages may not. Classical models of twin studies formulate an heritability calculation for the trait(s) being studied by comparing the two kinds of twins, using a three-part formula: additive genetic effects (A), environmental influences that are shared by both twins (C – common to both), and environmental influences that are unique to a given twin (E) (Nielsel et al 2012). Other (typically nonadditive) effects that can be searched for include dominance in genes (D), and epistasis (I), though these variables have some overlap with others and so that will need to be accounted for by the study design in some way. Whether one is using the ACE model, the ADE model, or something else, the aim is to produce an heritability calculation for a given trait.

Heritability, in turn, "has a precise technical meaning in biology: it is a measure of the proportion of the visible, phenotypic variation in a particular trait, at a particular time, in a particular population, living in a particular environment, which arises from genetic differences between individuals. It is a population measure, *not* a measure of the relative role of genes and environment factors in individual development." (Jablonka & Lamb 2014, 357). But even with those caveats, folks who endorse twin studies will often suggest that doing an heritability calculation offers a putative way to compare genetic relationships between phenotypes that exhibit the trait of interest.

5.4.2 – What sorts of Insights Do Twin Studies Offer?

Twin studies as a methodology purport to offer interesting insights, though of course individual studies can vary in their import. By offering a look at how phenotypes for some trait differ across populations with more or less similar DNA, researchers are invited to ascribe more or less causal force to genetic or environmental causes (as competing alternatives!) in order to explain phenotypic variance. Additionally, for highly similar (correlated) phenotypes, some researchers think that twin studies can give us an estimate for the degree to which the correlation is mediated by genetic factors common between the phenotypes (Nielsen et al 2012, 332). In stronger cases, where similar genes are involved across all cases of variance one is interested in, such studies may lead researchers to conclusions about underlying factors that connect the phenotypes. For example, Nielsen et al (2012, 332) perform a review of twin studies and heritability regarding pain phenotypes, and cite Williams et al's (2010) work that "a twin study including five anxiety disorders revealed that most of the genetic variance was explained by a single underlying genetic factor." While researchers are often honest about the fact that insights like this can only get one so far (after all, heritability estimates are tied to specific contexts), it is still an impressive and important result to potentially say one has located a single genetic factor that mediates correlated phenotypes. This is straightforwardly useful in motivating more in-depth research on the findings, and gives us a sense of getting at the internal cause of such matters.

Other twin studies are a bit more humble in their reach, and may have a more exploratory aim. By that I mean they are simply aiming to establish some sense of genetic involvement in a given phenotype, which can be explored further in future work. An example of this is Alford, Funk & Hibbing's (2005; 2008) work on whether political orientations are genetically transmitted. They also respond to critics in a 2008 paper, mostly focusing on the Equal Environments Assumption, but it's clear that part of their rhetoric involves trying to argue that genetic research on political orientations is legitimate. For example, they are sensitive to the limitations of twin studies and heritability analyses, but insist that similar findings merit further attention. As the authors mention:

> *"Twin studies are population-specific, meaning the results hold only for the populations studied [...] twin studies on their own ignore important confounds such as assortative mating and gene by gene as well as gene by environment interactions. No single coefficient can accurately capture the precise global influence of genetics on political attitudes, but we can say this: The large variety of empirical studies conducted with different samples in different countries that all report substantial (often in the 0.4 range) levels of heritability for political attitudes indicates that the genetic contribution to variations in political orientations is significantly greater than zero and may even approach 0.5. So saying hardly makes us determinists but it does encourage us to call for a reorientation of the common (and unsubstantiated) social science assumption that the only valid independent variable is an environmental variable. [...] for a variety of reasons an exclusive reliance on twin studies would be limiting. We fully support the use of multiple methodologies in this and other social science research. Twin studies establish that there is a genetic component to political orientations. This empirical evidence of a role for genetics gives us cause to look further at the nature of that role using other methodologies."* **(2008, 795)**

5.4.3 – My Response to the Objection

As such, twin studies mostly seem to be understood as important, albeit limited, tool in a researcher's toolkit. They offer a way to putatively poke at the different genetic and environmental correlations between phenotypes, and so have their place in a multi-method approach to giving some kind of more in-depth, causal story for some phenomenon of interest. Even with those caveats in mind, I think there is still something fishy about the underlying logic of twin studies and the way heritability is calculated. I

also think that this 'something fishy' can be well-explicated by the themes I have been discussing, especially pertaining to similar pitfalls I have been discussing around conservative interactionism.

I have three related concerns regarding the underlying logic of twin studies. First, the equations associated with twin studies and heritability explicitly break genetics and environmental causes into separate categories that can be added together. Second, by breaking the system into additive components in this way, the attempt is to locate the source of change in one category or the other. Third, by forcing this gene/environment dichotomy, there is reason to believe it greatly overestimates the importance of genes in producing the phenomenon of interest, which is consistent with the gene-centred focus of conservative interactionism that I have been arguing against. I'll address each concern separately.

5.4.3.1 – The Equations Imply Additive Development

I am aware that there are variations on twin studies that one can do, often involving additional comparisons to other family members. But as described above, there are three main terms involved in heritability calculations based on twin studies. The classical ACE model focuses on additive genetic effects (A), common environmental effects shared between the twins (C), and unique environmental effects to each twin (E). From these terms, one can flesh out the variables used in a broad-sense heritability calculus, which involves the variance in a genotype divided by the variance in a phenotype (ie Var(G)/Var(P)). Phenotypes are taken to be modelled as G+E (Genes plus Environment). Where variation in a more general population might be more difficult to describe, the assumption of twins' shared environment and overlapping genome helps simplify matters by ostensibly allowing us to describe variance in terms of categories A,C,E (or D, I, etc, depending on the structure of the study). By holding some variables constant (as per the Equal Environments Assumption), variance in phenotype can thus be associated with variance in either genotype or unique environmental effects. In this sense, difference in some variable is used to account for difference in phenotype.

A central issue with all this is that it is assumed the phenotype is just the addition of genetic effects plus environmental ones. Modelling matters this way treats genes and environment as discrete entities that can be simply added together. This is exactly in line with the mistakes of conservative interactionism, where parts of a complex system are treated as separable and distinct. This picture is bereft of the co-determination and interpenetration of parts that I have argued for in previous sections. If the genetic system extends beyond the body to include the environmental cues that it is coupled to (as I argued in Chapter 2), that coupled system cannot be decomposed into genes + environment without changing the very values of the variables in question. Coupled variables must be solved together as they mutually influence each others' values, and so to treat this system as decomposable into discrete terms (genes + environment) is precisely to miss the nuances of the coupling.

5.4.3.2 – Separate ‘Source of Form’

By decomposing the developmental system into separable, additive parts, the methodology of twin studies makes an attempt to locate 'the' source of causation within the separated parts and not their conjunction. My point here is not to try to argue that differences in environment or genes are irrelevant to developmental outcomes. That does not seem a position worth taking seriously, as it would commit to a strange fatalism about development and contravene the available evidence. However, it is important to think about the type of role we are assigning to these potential differences. Given our historical obsession with genes, it is perhaps unsurprising that there have been repeated attempts to locate the source and causal power for some phenotypic difference within the genes. Conversely, one could try to do the same regarding the environment. Regardless of whether one is focusing on genes or the environment, the key is that difference is often taken to explain difference. Sufficiently different developmental conditions might be used to explain differences in twins' phenotypes. Similarly, differences in gene/trait frequency between monozygotic and dizygotic twins may also be used by researchers to try and explain differing phenotypes. In both cases, the difference in question is taken, to use Dennett's turn of phrase, as a difference that makes a difference. And in describing it

as such, one locates the causal force within that entity rather than in interactions. It treats the entity as a simple causal agent, rather than a coupled system.

Additionally, this manner of analysis associates likeness with likeness, in addition to difference with difference. Recall, for example, Nielsen et al citing Williams' work that cited shared genes as mediating between similar pain phenotypes. So sometimes similarities can explain similarities, and differences explain differences. That might well be the case sometimes. But we also know that development can be well canalized in some instances, such that differences in conditions do not produce a difference in phenotype. Gene knock-out experiments sometimes do not result in a different phenotype. As such, a difference in the broader developmental context will not always result in a corresponding difference in phenotype. It is worth questioning what sort of explanation a difference offers us. It may be a valuable piece of information for us, but we should be careful not over-emphasize it.

Indeed, focusing too intently on difference as a source of causation can obfuscate matters rather than illuminate them. By analogy, lets assume there are two groups of students are working on school projects, but the two groups share a member (person 'B'): AB, and BC. Each group turns in a project. Why would we seek to explain the differences between the projects entirely by appealing to the unique members of each group? That would only work if student B had the same role in both groups, and had no input on the parts of the project that differed. Alternatively, we could see both projects as products of the labour of both students, where differences in group composition affected the overall result, but the 'phenotypic' differences are a result of the group rather than unique elements.

To relate matters back to twin studies, taking developmental systems seriously would imply resisting locating causal force as purely within one element or another, and instead looking at the broader interactions and how the parts are coupled. Finding genetic difference correlated with a phenotypic difference only tells us so much, as the genetic differences don't exist in a vacuum where they are uncoupled but still somehow causally potent. The same can be said for environmental differences. Forgetting this fact raises the

shadow of Nature/Nurture, of preformationism, where things somehow have their effect locked in long before the interactions that bring about their emergent properties.

5.4.3.3 – Forced (and False) Dichotomy

Finally, there is the issue of heritability involving a false dichotomy. The equations track genes and environment, and heritable variation is assumed to be genetic (Jablonka & Lamb 2014, 357). But there is more to heritability than just one's genes. Adding terms for cultural factors, epigenetic factors, and behavioural inheritance systems could decrease heritability significantly since we have reason to think that there's more going on than just genes and environment. As Jablonka and Lamb note, “heritability in humans is always somewhat doubtful, but by failing to distinguish genetic and nongenetic pathways, this makes [heritability] estimates even more shaky as it overrepresents genetic contribution to variation. There is more to heritability than just genes.” (Jablonka & Lamb 2014, 357-8)

This is not merely a concern that the specific numbers of a given heritability equation might be off. After all, we already know that heritability estimates can change given a different sample, or at a different time for the same sample. The point is that it purports to measure something in a way that it simply cannot, and the additional use of overly broad categories helps lean in to our gene-centric history. We do not advance our understanding by repeating our mistakes, by cleaving to a framework that perpetuates the Nature/Nurture categories. If the issues I have raised are serious in the way that I think they are, the methods of twin studies simply lead us right back to a conservative interactionist framework, recreating the assumptions of Nature/Nurture all over again.

5.5 – Supplementary (‘Wrong Contrast’) Argument

There is an additional, albeit short, argument to make here. This argument adds to the conclusion that the nature/nurture framework is incoherent, but for different reasons. The central claim is that nature is not even a proper contrast with nurture, and thus the framework is at least misguided in making such a contrast. The claim that it is misguided, however, can be upgraded to a claim of incoherence by expanding on the claim of poor

contrast. Ultimately, this rides on the claim that Nature is not merely a poor contrast with Nurture, it is a product of Nurture, and thus hardly an opposite for the sake of contrast.

Some definitions need to be changed in the process of making this argument, however. For this argument to work, one must be on board with the sort of expanded notion of inheritance that I have discussed. According to Oyama:

> *"Ontogenetic means are inherited; phenotypes are constructed. [...] I am only making explicit what is routinely taken for granted. No one claims that genes alone are sufficient for development, or denies that environments, organic and inorganic, microscopic and macroscopic, internal and external, change over organismic and generational time. What is missing from most accounts is the synthetic processes of ontogenetic construction. Inheritance is not atomistic, but systemic and interactive. It is not limited to genes, or even to germ cells, but also includes developmentally relevant aspects of the surround – and 'the surround' may be narrowly or broadly defined, depending on the scope of the analysis." (2000a, 71)*

The point to be made here is that if one wants to maintain a connection between inheritance and Nature, then some definitions have to change. Inheritance, for one, needs to be expanded, for reasons I have articulated in Chapter Three, and for reasons Oyama gestures at in the quotation above. But given the disconnect between Nature and inheritance, "Inheritance can be identified with "nature" only if it embraces all contributors to that nature, and nature does not reside in genes or anywhere else until it emerges in the phenotype-in-transition." (Oyama 2000a, 71-2). A thing's Nature, then, is just synonymous with the phenotype, and the phenotype continually emerges from interactions within the niche of the embedded organism. And if Nurture is understood in its traditionally broad and vague sense of nearly anything that takes place during the development of the organism, then Nature (in a deflated sense) is just the product of this Nurture-as-continuous-niche-interactions. This is a deflationary way of thinking about both Nature and Nurture – deflationary Nature because it is synonymous with the

phenotype and thus a product of development. This new Nurture is also deflationary in the sense that it is maximally broad. It sees development as an ongoing process that is inseparable from the actual living conditions experienced by the organism, which is part of Nurture as traditionally understood. These deflated categories are not contrasting opposites, and certainly not 'sources of form'. They are at best broad descriptions of goings-on within development and the emerging products (phenotypes).

I am admittedly leery of repurposing these terms, given their value-laden history, so I'm not sure how far I want to push this supplementary argument. For the most part, I am mentioning these new uses of the terms in the name of completeness. Perhaps one day someone will find a less risky use of these terms, and this usage will also be useful in helping elucidate matters within this sort of interactionist frameworks. For instance, while the traditional Nature/Nurture framework is incoherent, taking some of its parts seriously (ie inheritance) both corroborates the incoherence of the traditional framework, but also suggests that broadened versions of the concepts could possibly help illuminate certain kinds of relationships. That may be valuable for future work, but at present I am quite skeptical that such a project would illuminate more than it would simply lay a path for accidentally importing aspects of the Nature/Nurture framework. I think that, if anything, a central message of this dissertation has been how difficult it has been for us to actually move beyond traditional Nature/Nurture in a meaningful way. As such, I think we import these sorts of terms at our own epistemic peril.

5.6 – Wrap-up

This chapter has attempted to tie together several threads from previous chapters. My central claim has been to argue that the nature/nurture framework is incoherent. I have done this. I have also made a first pass at further explicating a picture of how I see development in terms of the metaphorical framework. Organisms are embedded within niches that make available developmental resources. Some of these resources are inherited (in the sense that they were also available to the previous generation), some resources might be newly offered based on changes in the world or (importantly) the activity of the organism within the niche. The interactions of these means of ontogeny are irreducible, in the sense that the active organism is coupled to many developmental

systems (or a single, larger developmental system depending on grain of analysis). There is more to say about where I think the study of development should go, however. This will be the focus of the final chapter, where I highlight instances of effective research and try to make a case for how the type of framework I've been discussing could be used in practice.

Chapter 6: Now What? Next Steps & Implications

The previous four chapters have largely comprised the negative project portion of this dissertation, by making the case that the Nature/Nurture framework is incoherent. However, I don't want this project to be strictly negative, for a mix of idealistic and pragmatic reasons. First, I prefer that my contributions not merely consist in tearing down the work of others. Second, given how steeped in the nature/nurture framework we are, I worry that merely tearing down the old framework without making suggestions on replacement parts will simply lead to a substantively similar framework being used in its place. After all, some of the perniciousness of the nature/nurture framework lies in how readily it is tacitly adopted, and how difficult it is to eradicate its influence. Despite seemingly honest desires to be rid of it, the distinction has persisted in contemporary biology.

Still, I laid the groundwork for a positive program in the earlier chapters. After all, I did spend a fair bit of time discussing how coupled interaction is supposed to work, and why development in biology should be thought of in this way. So I do not think that I have merely torn down some old framework while offering nothing by way of replacement. That being said, I still think it is worthwhile to spend some time discussing implications of my view, and where we go from here.

To that end, the main content of this chapter will consist of three suggestions for moving forward. Some are explicit suggestions for what I think will be useful, others pertain to areas of enquiry that I think will be worth watching in light of the framework I have suggested. All of these suggestions are aiming to promote meaningful interactionism as a core value in the science going forward. In order, my suggestions are: 1) focusing on regulatory systems and factors of gene expression; 2) expanded inheritance with the aim of investigating how developmental resources (broadly understood) are made available within the niche; 3) committing to 'no zero-state' developmental outcome and reconsidering how we want to think about teleology in biology.

Before we move on to these areas of interest, it is worth quickly returning to a thread I discussed previously (way back in Chapter 2!): namely, the pragmatic element of choosing how to make complex systems experimentally manageable. My return to this topic offers something in between a caveat to my own project and a scope clarification, since I want to avoid the risk of overstating matters now that we have seen the vast majority of this project. Specifically, I have spent a great deal of effort in this project cheerleading for coupled interaction, holistic/irreducible developmental factors, and the notion that additive conceptions of development run the risk of recreating problematic elements of the Nature/Nurture framework. I stand by these conclusions. However, there are some limits to them. I am not trying to claim that everyone must always treat development as irreducibly interactive in all contexts until the end of time. Development is a complex subject, and there are theoretically many ways that we might choose to carve up the problem space to make it more amenable to inquiry (i.e. see Longino 2013). As Longino notes, these different ways of carving up the problem space will all have their own limits, simplifying assumptions, and associated scope. And due to the complexity of the topic of interest, it is better to have a pluralistic approach within the community since each approach necessarily investigates only a select portion of the problem space. (For example, see Longino (2013)'s discussion for an overview on how behaviour genetics, molecular genetics, social/environmental approaches, neurobiological, and interactive approaches (including developmental systems theory) all approach the study of behaviour and development with different assumptions and scope). As such, each can bring something different to the table, and it seems likely that we have a better picture of the world in light of this pluralism in the scientific community. This conclusion somewhat mirrors that of Tabery (2014), who argues that we are split over what kind of interactionism to favour: 1) variance-partitioning, a conservative interactionist style of approach where development is additive; and 2) a mechanism-elucidation approach, where interaction is taken to be ubiquitous in development. Moreover, he argues that these projects are more or less incommensurable, but that there can be an explanatory bridge from one to the other. We need both, he claims. Variance-partitioning is useful for detecting patterns in the population, but cannot tell a causal story. Mechanism-elucidation, on the other hand, is useful for articulating the

developmental mechanisms involved in the trait production, but given its more holistic approach, can sometimes struggle to make the original problem space tractable (Tabery 2014). Pluralism of approaches, both Longino and Tabery suggest, is essential for progress.

My project is not aiming at blocking this pluralism, nor in claiming that reductive approaches can never be fruitful in any regard (even if I think they are misguided in some way; again, false models can still be epistemically useful in various ways (Wimsatt 1987)). Rather, my aim in this project has been to analyze a certain set of concepts (ie the Nature/Nurture distinction and associated framework), discuss the ways in which we keep recreating problematic aspects of this distinction in contemporary theory (despite our efforts to avoid this problem), and also to show how a more holistic approach to development can both help us get around this issue and elucidate the mechanistic complexities of developmental trajectories. While that is valuable, other researchers may choose a different approach for a variety of reasons. While Longino has a point regarding the value of pluralistic approaches, my hope is that my warning of the epistemic risk regarding the perniciousness of Nature/Nurture serves as a jumping off point for how investigative efforts are organized, how concepts are chosen, and how results are shared within the wider community. I think investigations of development would benefit from the holistic approach that I have advocated, both in terms of better understanding the mechanisms involved in shaping developmental trajectories, and in mitigating the epistemic risk of recreating Nature/Nurture. Both of those outcomes strike me as epistemically valuable. However, there are circumstances where it may be appropriate (and fruitful) to choose a different approach, and hopefully mitigate any associated risks by leveraging the pluralistic nature of the broader scientific community. Time will tell how this plays out. And while such pragmatic concerns were worth repeating, my intention with the rest of this chapter is to highlight areas of study worth keeping an eye on. So let us now turn to that.

6.1 – Gene Expression & Regulation

I am not the first to suggest that a focus on factors affecting gene expression and regulation will be a key route forward (see Stotz 2006, for example). However, I think it

is worth emphasizing again. One of the central issues that I have highlighted throughout this dissertation is the lack of meaningful interactionism being employed due to a gene-centric focus, and the problematic treatment of genes as if they had teleology within them. It might seem slightly strange to emphasize a gene-related phenomenon when part of my diagnosis of the problem is that we have been too gene-centric in our approach. However, I have two responses to this worry. First, no one thinks that genes are unrelated to development entirely, so surely they are part of the interactive picture in some capacity. This means they won't be excluded from the way forward. Second, my hope is that the focus on gene expression and regulation, despite giving genes yet more attention, will serve to change how we think about the role of genes for the better.

Given the discussion in Chapter Two, it seems that many factors can be involved in gene expression – including environmental cues. As such, a call for research looking at gene expression could include anything from methylation patters that affect when and how a gene is transcribed, to environmental cues that lead to the epigenetic change. As such, despite the focus being oriented around a gene and its expression, there are still many possible areas of fruitful research. Additionally, research that highlights how genes can be expressed in different ways, implicated in different developmental tasks, etc, serves to highlight that genes don't have simply one thing that they do. This, in turn pushes back against the notion of genes as simple causal units, and it becomes harder to talk about their specific telos if they are involved in a variety of processes.

I also hope that an increased focus on the ways in which gene expression can be altered, or how regulation occurs will additionally generate an interest in how interactants become available to the developing organism within the ontogenetic niche.

I will say more on this in the next section, but I think the connection to be made here is something like the following. If there are reliable factors in a given gene expression, then we can also ask how that particular expression might be found (or 'reconstructed') elsewhere. In other words, if genes can be expressed in a variety of ways – and that seems to be what the evidence is pointing to – then we can also ask how the

constituent interactants of that expression become available to other creatures in other contexts. Putting the question this way serves to highlight not only the inheritance systems we have access to, but also the ways that we might shape our niches to bring about certain developmental outcomes. (I expand on this example in the next section, but language is a good example: language use is ubiquitous in our niches, and we reliably shape them to cultivate language acquisition in children). As such, the emphasis is not merely on extended inheritance systems, but the dynamic way that we can change, modify or enhance access to some of these systems (or the contents of the systems) through behaviour. Linking matters in this way serves to position a normally gene-focused topic (ie gene expression) within the broader developmental system.

6.2 – Expanded Inheritance & Useful Next Steps

A second area that I think will be important in the future involves an expanded notion of inheritance. By 'expanded' I am referencing Oyama's understanding of inheritance that I mentioned earlier, where inheritance just is some developmental resource being made available to subsequent generations. This is explicitly agnostic about whether the resource is genetic, epigenetic, behavioural, symbolic, or some combination. It deliberately transcends the internal/external divide to recognize that a) the inheritance/acquisition distinction was flimsy and doesn't match the way these categories interact, and b) there are many ways we can receive developmental resources, and sometimes it may be the manner of receiving it -based on how the niche is constructed – that can offer insight into the reliability of some developmental outcome's reconstruction.

There are two main reasons I think this expanded conception will be important. First, I think it helpfully draws attention to the fact that organisms can receive many types of developmental resources. By recognizing and emphasizing this within a framework that highlights interaction, I hope it will spur research into what types of things might function as developmental resources and how these things are made available to subsequent generations. The 'what' and 'how' are distinct but related questions, and I'm open to the answers varying from organism to organism. The variance from species to

species also offers plenty of research opportunities, both in terms of what counts as a developmental resource, as well as how it might be 'gated' or not. Gating refers to the way in which some resource becomes available to a population where it otherwise might not be. For instance, in West & King's studies of brown-headed cowbirds, they found that flock structure gated both song structure and song preference, and made some types of songs 'bio-available' (2008). In this sense, there is a difference between having some developmental resources 'merely present' in the niche as opposed to being salient or relevant for the developing organism. In the case of cowbirds, this distinction tracks how the bird might try and imitate some things but not others, when there are many sounds that it could try and imitate. For instance, for birds kept in a large aviary, they can hear all sorts of songs around the aviary from wild birds, both from conspecifics and non-conspecifics. But merely having songs around an aviary was not enough to get the flock interested in the song. Instead, the social structure of the flock determined what sorts of things would receive attention and what would not. Older females might pay attention to different male song across different mating seasons, but younger females were highly sensitive to the attention of their elders, and so the type of songs that the elder females paid attention to would gate the types of songs that the flock tended to respond to. This was the case for both female and male birds, since males tended to adjust their songs based on the social responses of the females. For some song to be available in the right way (such that it would be imitated by males and preferred by younger females), the song needed to attract the attention of the elder females. If the song did not attract elder attention, then it was significantly less likely to be imitated by males or preferred by younger females. The structure of the flock and the responses that follow based on the social relations between individuals thus determines whether some developmental resource (song structure) is made available to the flock as a whole or not. Behaviour can thus gate learning in particular ways, and investigating the ways in which learning and developmental resources can be gated or made available within the niche will help us to understand the ways in which we are connected with the world around us.

I've mentioned a number of areas that I think will have promising research, but I also want to highlight some of the ways that these types of recommendations are already being implemented. In addition to the bird examples above, this is something of a 'credit

where credit is due' section, briefly showcasing a couple research efforts that I think show useful results.

6.2.1 – Mosini on Chaperone Role of Proteins

The first example is Mosini's (2013) work on the chaperone role of proteins in heredity, where she is using these results to push back against the gene-centric informational theory of heredity subscribed to by Neo-Darwinists. A bit of a history of science paper, she traces the way in which Crick's (1958) oversimplification of protein folding became enshrined in biology, until some key experiments showed that not all proteins are arranged in patterns that would accord with thermodynamic control. Crick's assumption was that the folding arrangement of proteins was simply a result of its bases, meaning the sequence of nucleic acids implied a certain structure of the resultant protein. (It's also worth noting that Crick later mentions that he made this assumption consciously in order to reduce the problem of protein synthesis from a three-dimensional problem to one-dimension (Crick 1970, 561)). There are some proteins for which this assumption works perfectly well. However, it is an oversimplification, and is far from being all-inclusive. This is where Mosini's emphasis on the chaperone role of proteins comes in.

Some proteins, such as groEL, groES, and stress proteins, can assist in folding other proteins in various ways, such that the resulting proteins do not end up in a state that could be described according to thermodynamic control. As Mosini (2013, 60) puts it:

> *"Such assistance may be performed through a number of mechanisms (Langer et al. 1992): from sequestering the substrate protein in a protected, encapsulated, environment ('cage') (Ellis 1987, 1996, 2006), to inhibiting competing reactions (Liu et al. 2010), arresting 'translation' from RNA which may lead to interferences, or unfolding mis-folded states that might have arisen as a result of the transient exposure of hydrophobic or charged sections during polypeptide synthesis or after cross-membrane transport or recovery from stress (Hemmingsen et al. 1988; Peralta et al. 1994)."*

As mentioned, these folding states are not always thermodynamically controlled, but rather are kinetically controlled by the proteins (Mosini 2013). Point being, there are some folding states where Crick's assumption simply doesn't hold, and proteins can be folded into other shapes through complex interactions with chaperone proteins and the overarching translation machinery.

Chaperone proteins have other roles, too, beyond translation. They are also involved in transcription, hormone signalling pathways, and other such factors. According to Mosini (2013, 63):

> *"Take, for instance, the steroid receptor function involved in 'transcription'; it has been found to be mediated by the stress protein and molecular chaperone Hsp90 (Bohen and Yamamoto 1994). Other stress proteins, notably Hsp56 and Hsp70, as well as molecular chaperones DnaK and DnaJ, have been found to regulate the oligomerisation state of various transcription factors (Craig et al. 1994), affecting gene expression and, therefore, the transfer of information from DNA to RNA. The stress protein Hsp90 has been singled out as ''a capacitor for morphological evolution'' (Rutherford and Lindquist 1998, p. 336)."*

Point being, proteins are involved in all sorts of processes, and their involvement complicates the simplifying assumptions made by Crick that helped reinforce a gene-centric view of such things. But many such biological functions – trancription, translation, etc – can depend on both content and shape. And so, Mosini's argument goes, proteins have a much more complicated role in heredity than was originally imagined. The Neo-Darwinian approach originally confined proteins to the role of helping decipher genetic code (ie see Maynard-Smith 2000a/ 2000b). But even if one cedes that genes control the content of proteins, chaperone molecules seem responsible for their shape (and function). Since both properties are vital to various biological processes, the notion of heredity should be expanded from being gene-centric to include other things, such as chaperone molecules, since heredity cannot take place without their shaping effect.

6.2.2 – The Extended Evolutionary Synthesis (EES)

A second area that I think deserves highlighting is the 'Extended Evolutionary Synthesis'. Folks familiar with wider discussions on biology will be unsurprised that I am highlighting this approach, since it is very closely aligned with what I have been working on in this project. For those who are less familiar with the term, the Extended Evolutionary Synthesis (EES) is a term that is used in a few different ways, but in general it is understood as "[not just an extension of the Modern Synthesis, but] a distinctly different framework for understanding evolution" (Laland et al 2015, 3).

As a framework, the EES differs from the traditional Modern Synthesis (MS) approach in a number of respects, and these differences also point to differences in thinking about evolution. Laland et al's article (2015) is quite useful in directly comparing the respective positions on a different key issues. For instance, under the traditional MS, natural selection is king, in that nothing besides natural selection is needed to explain the fit between environment and the matching traits that organisms have. For EES, however, there is emphasis on reciprocal causation, where organisms shape and are shaped by their environmental niches. Developmental bias and niche construction therefore also have a role in explaining the fit between traits and environment. As I have addressed in previous chapters, there is also a distinction about whether we think of inheritance as a purely genetic process (as the classical MS does), or think of inheritance in a more inclusive way (as advocated by the EES). The MS posits that there is only random genetic variation; the EES argues that there is non-random genetic variation due to the way developmental processes have reciprocal causation between organism and niche. The MS holds that evolution on a macro scale can be explained by micro phenomena such as selection, gene drift, gene flow, and mutation. By contrast, the EES expands this list to include developmental bias and ecological inheritance, and argues that these conditions can also contribute to evolvability. In short, where the Modern Synthesis favours a gene-centric approach, the Extended Evolutionary Synthesis favours an organism-centric approach, including the interplay between organism and environment.

The EES is only an emerging framework for thinking about evolution and development, but given the differences highlighted above, it is an area worth keeping an eye on for the future as we gather more evidence and the Modern Synthesis might strain to account for everything. In addition to the Laland et al (2015) article, Jablonka and Lamb (2020) have a new book explicitly arguing for the EES. This is unsurprising, as Jablonka and Lamb's work over the years has steadily paved the way towards this type of theory. After all, *Evolution in Four Dimensions* (2005/2014) quite explicitly targets non-genetic inheritance systems as being critical for development; Jablonka & Raz (2009) have a seminal paper on transgenerational epigenetic inheritance, allowing us to flesh out ways in which such an extended framework might work in relevant ways. Similarly, Jablonka & Noble (2019) have a paper on the systemic integration of multiple inheritance systems. So the development of this framework has been a steady project for a few decades, and they of course are building on the work of those that came before them.

The position that Jablonka & Lamb (2020) develop in this new book is, unsurprisingly, a continuation of the types of arguments that they have made previously. Comprised of four sections, it examines the history of the Modern Synthesis, ways of characterizing inheritance systems, explicitly connects non-genetic inheritance with evolutionary implications, and finally, culminates with an overview of the philosophical implications implies by this shift in framework. As an inheritance-focused project, there are philosophical implications in the notion of information, the distinction between replicator and interactor, the notion of biological individuality and identity, among others (2020, 56). One point of emphasis, however, is that “The entanglement of developmental plasticity, developmental bias, niche construction and multiple systems of inheritance [...] requires a move away from the gene-centred MS view of evolution to a development-centred view (Laland et al 2014, 2015).” (2020, 71) In adopting this organism-centred view, the aim is to take seriously sources of evidence that are marginalized by the Modern Synthesis, such as niche construction and non-genetic inheritance. While this position is currently not the norm in biology, given its emphasis on development, this is very much an approach that I would like to see rise in popularity. But we shall see how matters turn out.

6.2.3 – Synthetic Biology

Finally, I wanted to round out this section with a few remarks about an area that I think will be interesting to watch for a number of reasons: synthetic biology. Unlike the previous two examples, I am less certain that the results of synthetic biology will engender a development-forward approach to biology that I think is needed. That being said, I think there are some points that are worth mentioning.

Let's start from the beginning. What, exactly, is synthetic biology? This is a surprisingly tricky question to give a full answer to, and many different definitions focus on different aspects within the broader field. For instance, the National Human Genome Research Institute identifies synthetic biology as “a field of science that involves redesigning organisms for useful purposes by engineering them to have new abilities.” (NHGRI, 2019). Moreover, they go on to discuss how synethetic biology is allowing us to harness the power of nature for our own purposes, and where they see the distinction lying between gene editing and synthetic biology (the scale of changes made, in short). By virtue of the language used, this approach clearly likens synthetic biology to a kind of engineering, such that we can build new creations (or repurpose old ones) that are in line with our purposes – whatever those might be. The listed examples of application include: 1) engineering microorganisms to clean pollutants from our environment; 2) rice modified to produce beta-carotene, with the aim of reducing childhood blindness through decreasing the likelihood of vitamin A deficiency; 3) manufacturing yeast to produce rose oil, enabling a more sustainable rose-scented product for luxury perfumes. As such, the practice of synthetic biology can clearly be both research and product-oriented. Additionally, there is a short section on the relatively recent history of synthesizing an organism's entire genome: done first in 2002 with the simpler genomes of viruses (not the least of which involved creating a polio virus from scratch, raising plenty of ethical questions about bio-weapons), and in 2008 the Craig Venter Institute successfully synthesized the genome of a bacterium, Mycoplasma genitalium.

Nature magazine offers a slightly different definition of synthetic biology – and, for what it's worth, one that Western University's interdisciplinary reading group on synthetic biology found more palatable. On this definition, synthetic biology is “the

design and construction of new biological parts, devices, and systems, and the re-design of existing, natural biological systems for useful purposes." (Nature, n.d.). This definition has a lot in common with the previous: both emphasize the utility of outcomes in the field, both stress an element of design, etc. There is a difference in the degree to which an aspect of engineering is explicit, and indeed this has been a sticking point in the literature, in that there is sometimes a tension between emphasizing our supposed mastery over the world around us, versus emphasizing the sheer complexity of biological systems and how our interventions can and do fail. Regardless of the minutiae of these differences, however, the point is that synthetic biology is routinely taken to be a branch of science that actively intervenes in organisms, makes alterations to their functions, aims at creating useful outcomes or functions, and/or is interested in the creation of new (or 'synthetic') organisms and genomes, such as the synthesized virus and bacterium examples mentioned above.

The sheer applicability of this branch of science is a central reason why I think it is important to keep an eye on it, since the type of language used highly-regarded projects could have a strong impact on the way we talk about biological systems. And while not everyone agrees with the engineering emphasis of synthetic biology, my worry is that the successes of synthetic biology might reinforce the conflation of reliability with innate content. After all, part of the advertising of synthetic biology is that we can genetically-edit our way to a better (or more useful) world, and that focus is squarely on the sheer power and possibilities offered by genes. That is, I think there is a possibility that our ability to reliably produce certain functions, traits, etc in some organism through gene editing might serve to reinforce the Modern Synthesis's emphasis on gene-centrism and content that is selected for through natural selection. Publication bias may also contribute to this: after all, genomic editing can have off-target effects, and is not guaranteed to work. But it is less likely that the general public will hear about the failures compared to the successes. And insofar as the successes are framed as gene-driven outcomes, the general public might get the sense that some sort of bioessentialism is the case. That is, of course, partly a worry about science journalism and scientific literacy in the general public. But it is more than that, I think. The engineering approach that gets so often invoked could be taken as implying that biology can be modular, or just that we will

build a better world through gene manipulation. Such themes threaten to reinforce the gene-centric understanding of biology that we already have, and the successes of such programs may entrench such attitudes. So there are certainly epistemic risks involved as this branch of science grows in popularity. However, not all aspects of this topic worry me in this way, as I'll discuss presently.

A connected topic involves the use of synthetic biology in conservation, and like most of synthetic biology this discussion is often gene-focused. There are many facets to this conversation, but one thread I want to tease out involves the discussion of gene drives, their conservation potential, as well as risks and ethical implications.

For starters, according to Sandler (2020) and the IUCN (2019) a gene drive is a genetic modification that "increases the probability that a genetic trait or element will be inherited with greater frequency than the usual Mendelian ratio. Gene drives increase the rate at which a genetic modification spreads through a sexually reproducing population and could enable engineering genetic changes throughout wild populations (IUCN 2019)" (Sandler 2020). There are different versions of this effect. As Preston (2018, 104) puts the matter in his book *The Synthetic Age*:

> *"One version of a gene drive puts the CRISPR editing mechanism primed with the desired trait into the germ cells of a reproducing organism. If a doctored organism mates with an individual that lacks the beneficial trait, the CRISPR technology—now embedded in the germ cell—will edit the replacement trait into the chromosome that lacked it. The new individual now has the valuable trait present in both chromosomes and is ready to pass it on to the next generation, greatly improving on the 50 percent chance of inheritance that otherwise would have been present. Also passed on is the still functional CRISPR editing mechanism. The editing process and the valuable gene now will spread quickly through the wild population as it continues to breed with an almost 100 percent chance of passing on the desired genes."*

Both of these discussions clearly emphasize the notion of editing in specific genes *for* some specific trait. Sandler also mentions the inheritance of traits, and I have discussed in earlier chapters why I think this language is a mistake. After all, traits are never inherited wholesale, but rather are (re)constructed in ontogeny. That aside, however, I think these examples showcase both the immense potential for synthetic biology, as well as the way in which its focus on genes and genetic editing might serve to reinforce the problematic gene-centric aspects of the Modern Synthesis.

However, not all aspects of gene drives reinforce this worry. In fact, quite the opposite: some recent attempts at implementing gene drives have highlighted the ways in which organisms can respond to (and sometimes overcome) our attempts to modify them. For instance, there has been a great deal of work put into using gene drives to help eradicate malaria, stopping the spread of mosquitos and other pests, and so on. In separate lab tests involving fruit flies and mosquitoes, however, the organisms frequently developed resistance to the gene drive quickly through mutations (Scudellari 2019; Champer et al 2017; Hammond et al 2017). Often enough, the mutation would alter the sequence that CRISPR was targeting, which would prevent the edit from taking place. As such, if the frequency of the edits dies off, then the gene drive loses much of its potency over generations, rendering the targeted problem unsolved. These difficulties can be overcome, but I think it is important to highlight these difficulties and how one can use them to push back against a gene-centric narrative. The ability for organisms to respond to drives that would fundamentally alter the population, and even mutate themselves to be increasingly immune to the effects of the gene drive, highlights the complexity of organisms and how they can respond to their environment (broadly construed here). So while the focus on targeted changes might endorse a gene-focused approach, the resistance of organisms pushes back and might serve to broaden our considerations in useful ways. Given synthetic biology is a relatively new field, I do not have a guess for how these different threads will play out. There are some aspects that worry me, and some aspects that I think serve as useful reminders about the sheer complexity of biological systems. Time will tell how matters play out.

Additionally, there is the (hopeful) possibility that synthetic biology could be a gift that keeps on giving for developmental science, as we could manufacture odd developmental contexts just to see what happens. For instance, how differently does an organism with an entirely duplicated genome respond to its niche? How does the notion of a minimal genome intersect with the idea of extended inheritances? Does thinking about inheritances and the ontogenetic niche help clarify what a truly synthetic organism is (as opposed to one which is 'merely edited')? There are just so many possibilities for interesting and fruitful research, and I am not yet sure whether my hopes or my worries about synthetic biology will win out. But that uncertainty is precisely why I think it is an area worth watching.

6.3 – ‘No Mistakes’ Slogan & Teleology

We come, at long last, to the final section of this dissertation. Where previous sections have looked at areas of research that I think will be worth pursuing in the future, here I want to focus more on carrying certain themes forward. And, for the sake of symmetry, to again tie the epistemic aspect of biology with its political ramifications. As the project opened with such considerations, so too shall it conclude. I began Chapter One by highlighting some of the socially pernicious aspects of the Nature/Nurture framework. Now, in Chapter Five, I want to argue why I think there is some reason to believe that understanding development as ongoing irreducible interactions can have some ameliorative effects for the socially pernicious aspects. My main focus is on arguing that without the specific outcome-directed teleology of the Nature position, we can better understand that development doesn't make mistakes in the sense that there isn't a target that is hit or missed. Rather, there are sets of interactants, and while developmental bias might favour a certain ranges of outcomes, we can understand variation as an explicit part of biology. To be different is not to say that something 'went wrong' in one's development.

Recall the discussion of the Naturalist-style position discussed in earlier chapters. Put very briefly, the common thread across Naturalist-style positions is some commitment to preformationism. This is where, on account of our evolutionary history and natural selection, genes ostensibly have something like innate content that is the

result of the shaping effects of natural selection. Development is then the mere unravelling (with potential modification) of this pre-existing content. This is how you get discussions where developmental outcomes are attributed more or less to internal versus external causes; typically genes vs environment. Collectively, this means that there is a certain teleology assigned to genetic content and development, such that there exists targets to be aimed at (and thus either hit or missed, by implication). Nature thus is taken to have content, and that content aims at being expressed in particular ways. This is the version of Nature that I have been arguing against, practically ad nauseum.

To make my case, I want to return to the postgenomic understanding of 'nature'. As I have discussed previously, a popular understanding of nature within the postgenomic literature (see Oyama 2000; Stotz 2006, 2008) is that the nature of a thing is whatever results from interactions within the niche. As Oyama puts it, “Inheritance can be identified with "nature" only if it embraces all contributors to that nature, and nature does not reside in genes or anywhere else until it emerges in the phenotype-in-transition.” (Oyama 2000, 71-2). One of the ramifications of this way of understanding the topic is that Nature is not properly contrasted with Nurture; rather, the nature of some developmental outcome is simply a product of the interactions that give rise to it during development (some of which would traditionally be classified as 'Nurture'). In other words, on the traditional understanding of the words, Nurture (plus other stuff) is the cause of Nature, rather than have both Nature and Nurture be alternative explanations for some developmental outcome. The relationship between the two traditionally-defined categories is fundamentally different. Additionally, it is a fundamentally different way to understand what the Nature of some organism is. In light of this, I think it has importantly different political ramifications.

I think one of the most important ramifications of using the term 'Nature' in this way is that it dilutes a socially-pernicious, teleological element of the traditional way of understanding Nature. (For a more fleshed out version of my arguments for the social perniciousness, see Chapter 1). To very quickly rehash: performationist aspects of a Naturalist position imply that the organism's genes have predetermined content, often as the result of natural selection. And, while developmental outcomes can be modified by

the environment, generally speaking the role of the environment is taken to be enabling or constraining the predetermined 'inner plan' of the organism. If there's a predetermined plan for development, then there is a targeted outcome that is either hit or missed (to varying degrees). But the existence of some predetermined plan (and thus the traditional understanding of Nature) is contingent on genes acting like simple causal agents, where genes are effectively the most important aspect of development. (Not the only important aspect, of course – even genetic determinists are unlikely to go that far). Most of my efforts in this project have aimed at undermining the traditional understanding of Nature and Nurture, and some of the evidence that I rely on to make this argument is the fact that genes just are not simple causal units in this way. In light of this, the term 'Nature' is not to be understood in the traditional, preformationist, way. Instead, an organism's Nature continually emerges as part of the outcome-in-transition. This means that there is no predetermined developmental plan squirrelled away in our genes. In turn, that means there is not an inner genetic teleology that aims at specific outcomes. If there's no teleology of this sort, then it's much harder to speak of development hitting or missing predetermined targets in a meaningful way. Developmental outcomes don't 'miss', because there isn't a plan that aims. Can't miss a target if there's no aiming and no predetermined target.

I think it is hard to understate how socially important it is to say that a developmental outcome cannot miss its intended outcome in this way, because there is no internal predetermined plan for the fully constructed organism. The form does not precede the interactions that give rise to it. But what this means is that both reliability and variation of outcome are just part of developmental systems and how their parts interact with each other. Both the 'normal' and the 'unusual' outcomes are on equal footing as far as their natures are concerned: both are products of their constitutive interactions. Neither one hits or misses a target; both simply exist in relation to their constituent processes.

Why is this important? The primary importance, I think, lies in the way this approach pushes back against other socially pernicious research programs that push some form of bioessentialism in mainstream pop science. Here I have in mind such issues as: race science (ie see Charles Murray's *Facing Reality: Two Truths About Race* (2021))*;*

problematic investigations of brain-based and 'hardwired' sex/gender differences that are taken to explain behavioural differences (ie see Brizendine's *The Female Brain* (2006) and *The Male Brain* (2010), respectively); and arguments about how our human nature undermines many attempts at positive social change (ie see Pinker's *The Blank Slate* 2002). Understandably, we as a society are clearly sensitive to the ways that justification and/or explanation are sometimes offered for social stratification, and the corresponding ways that groups of people are values and ranked against each other. But moreover, I encourage the reader to sit and listen to the ways that developmental outcomes are sometimes described, especially if people are not neurotypical, or have a disability, serious mental illness, etc.

Many times I have heard some developmental outcome described using language such as 'something went wrong in development', 'that part of the brain didn't wire up correctly', or 'things didn't unfold as they normally should'. Now, I am of course aware that there are potentially multiple ways to interpret the precise claims made in these types of statements. But that aside, my point is less about the nitty-gritty details of any of these specific claims and more about the hostile language that can be used when some developmental outcome is deemed 'not normal' in some way. As such, to the degree that hostile comments of this sort have their root in some sort of preformationist teleology, the framework that I am proposing has a simple response to this: development doesn't “make mistakes”, so to speak. (At least not in a biological sense; I discuss later how the term 'mistakes' can be understood in a social or cultural way). Our developmental outcomes are not mistakes. They are continually-emerging products of interactions. Even in outcomes that we might have reason to not like (ie heavy metal poisoning), this is not development 'gone wrong'. It is just development. And the ability to tell people that they are not mistakes of development - they are not 'broken' in this way – seems to me a powerful, if limited, tool.

Having said all this, I am under no illusions that the view I have just articulated – that development doesn't make inherent mistakes – is a panacea for sweeping social problems. I also do not want my view to come across as too utopian, or too naive – that everything is possible and it's only Big Bad Society that ever holds us back. I merely

wanted to put forward and defend the notion that, based on how development works, I do not think it makes mistakes, and that saying this can be a powerful social tool. But since it is perhaps easy to over-emphasize this aspect of things, I now want to forestall some potential objections that could be raised against me, and take the opportunity to be maximally clear about the limits of my claims.

6.3.1 – The Limits of the Slogan

First, my slogan of 'no mistakes in development' is limited insofar as it is built to undermine preformationist teleology in development. By no means do I think that preformationist-style biology is the only other framework that might stratify developmental outcomes based on a variety of metrics. As such, I again emphasize: my slogan is intended to be socially helpful, and is necessarily limited in scope based on the argumentative path by which I arrived at it. Nothing I have said thus far blocks claims about the statistical likelihood of some outcome (ie 'X is unusual because it is infrequent'), or that some outcomes may be better adapted within a given niche.

But both statistical and fitness-based claims are contingent, post-outcome evaluations (ie they are evaluations that we make after some outcome has obtained in some set of circumstances). The contingency aspect is something worth emphasizing here, as I think that sets statistical and fitness claims apart from 'target-based' claims. For instance, if a person were born with blue hair, that would be unusual in a world where few other people have naturally blue hair. Conversely (and somewhat obviously), in a world where blue hair is the most common colour, being born with blue hair is neither unusual nor 'unnatural' in any statistical sense, since the claim is dependent on the contingent setup of the world. Similarly, claims of fitness are not made in a vacuum, but rather pertain to an organism's ability to exploit some facet of its environment such that it enjoys a comparative advantage over its conspecifics. Again, both the relational and comparative aspects of fitness claims highlight that they are dependent on the way the world currently is, and the match (or mismatch) of organisms within that world.

By contrast, claims of 'natural vs unnatural' or about 'intended developmental targets' pick out what the body 'should be', according to some set of criteria (be it genetic

history, natural selection, etc). The evaluation isn't as contingent, in that the implication seems to be that the target is inherent. For instance, if the thought is that my genes are supposed to somehow dictate how I am embodied (or behave, etc), then in some sense my current embodiment is being evaluated against the 'intended version' of me. That seems a deeply personal evaluation in a way that statistical and fitness claims aren't. Of course, that doesn't stop someone from trying to somehow weaponize either statistical-frequency or fitness claims, but my point is that there seem to be important differences between the types of claims. And if the reader is still with me at this point, hopefully agrees that my position is aimed at blocking the more inherent form of evaluation, but still leaves more contingent methods of evaluation. After all, there is only so much heavy social lifting I can hope my arguments enable, while not extending so far as to block other, potentially useful, forms of evaluation.

6.3.2 – Two Challenges to This Approach

My arguments above are intended to offer a kind of social liberation. Where there are any number of social pressures for our embodiment to take certain socially acceptable forms, I had hoped to offer an olive branch to those who felt left out, and reassure them that developmental outcomes are not inherently mistakes. I've made this point by emphasizing the way in which the Naturalist-style teleology simply isn't available to an irreducibly interactionist approach. However, there are some challenges that are associated with resisting this Naturalist-style teleology. To round out this chapter, I will address two such challenges in turn. First, the reliability challenge: the Naturalist could explain reliability of outcome because development aimed at certain outcomes. But if I am denying that there is a target to be aimed at, how does my position account for the (seeming) reliability of form? Second, developmental teleology offered an easy way to evaluate potential developmental outcomes and/or forms of social organization, based on what is 'natural'. If I am claiming that there are no mistakes in development, and we cannot appeal to developmental teleology to give us a 'natural scale' for ranking outcomes, then how do we decide between potential outcomes? Am I committed to valuing every outcome equally?

6.3.2.1 – The Reliability Challenge

First, the reliability challenge. If there are no developmental targets in the way I have described, then how do I explain reliability of form? After all, yes many people are quite different from each other (within certain bounds), but it does seem as if there were limits on how different folks can be (or if not limits on the range of outcomes, then at least there are only so many developmental outcomes that actually obtain). For someone espousing a Naturalist-style position, this reliability is fairly straightforward to try and explain. For instance, if we assume that natural selection shapes the range of genes that we have access to, and genes have specified content shaped by natural selection, then we should expect a relatively curtailed range of developmental outcomes if we assume that development is just the executing of that predetermined genetic plan. In effect, for the Naturalist, reliability of outcome is easily explained, but variance is more difficult to account for (at least on a straightforward preformationist/predeterministic story).

The way that we can account for reliability is through the developmental niche, inheritance systems, and especially behaviour. I am also aware that responding in this way may further limit the scope of my 'no targets in development' slogan. But while I resist the idea that genes are packed with innate information such that development has an inherent teleology, I am not sure it's so easy to exclude at least some forms of teleology from the rest of the developmental niche. At the very least, with behavioural inheritance systems, the purpose of these systems can be to ensure that certain skills or information are learned by the next generation of organisms. That is to say, the existence of inheritance systems of this sort can indeed aim at fostering traits in a population. In this case, the 'aiming' is done by us, through our behaviour. And insofar as behaviour and behavioural inheritance is a part of development, developmental outcomes can certainly be aimed at or steered towards in various ways. The widespread use of educational institutions is a good example of this. Clearly, we as a society have determined that education is important to us, and so we have created social institutions to shape the development of our children to include certain types of knowledge. The reliability of a population possessing certain knowledge (ie basic math, certain forms of literature, etc) is explained by the socially-based inheritance systems that we have constructed to ensure

that most people have knowledge of this sort. Whether through niche construction or mere niche changing (to steal Dawkins' (2004) term), we can set up social and behavioural systems with the aim - be it tacit or explicit – of engineering developmental outcomes within certain limits. Despite this being a form of teleology, I think it is (on the face of it) slightly less pernicious than the Naturalist's approach. This is because, while society certainly may value some sorts of developmental outcomes more than others, being an 'undesired' outcome in this case has more to do with a mismatch between society and developmental outcome, rather than suggest that the outcome itself is wrong. This leaves room for society to change what it values. However, I certainly acknowledge that this distinction I am pressing may be cold comfort for those who find themselves not valued by society but still able to claim they are not 'developmental mistakes'. That's fair enough, but one thing at a time. My central point is that social values (and especially the associated practices) can shape the way that developmental resources are made available to developing organisms within the niche. Reliability can be the result of intensive behavioural efforts. Additionally, it makes for interesting research to look at what happens when reliability within the niche breaks down.

6.3.2.1.1 – Theory of Mind Example

Language use makes for an interesting example in this vein. It seems clear that, as a species, we have found language use to be extremely useful, and so it is practically omnipresent. Many developing children will have conversations involving mentalistic language with their parents (such as during story time, where the parent might invite the child to consider what a given character is thinking about, how they feel, etc). Children using mentalistic language of this sort is part of how they pass a battery of tests for what's called 'theory of mind': the recognition that others have private thoughts and to intuit what others might be thinking based on what information they have access to (ie see Peterson 1995). A classic test for theory of mind is the Sally-Anne test (a version of a false belief task), which proceeds in some version like the following. Sally has a favourite ball, which she brings to her bedroom and puts in her play chest. She then leaves to go to a friend's house. While Sally is gone, her sister, Anne, comes in and takes Sally's ball out of the chest and puts it in a drawer. The question for the child taking the test, then, is

'where will Sally look for the ball when she comes home?'. Passing this test ostensibly relies on the subject recognizing that Sally has access to less information than they – the test-taker – does, and so answering based on the information that Sally would have access to (ie recognize that Sally would have a false belief about where the ball is located, as opposed to the test-taker just giving the correct location of the ball).

However, not all children have this ease of obtaining high-level linguistic input from their parents, especially if there are language barriers, such as with deaf children raised by hearing (but non-signing) parents. Various studies (Peterson et al 1995, 2000, 2005; Pyers & Senghas 2009) show that deaf children raised by hearing (but non-signing) parents exhibit a significant delay in passing theory of mind tests. This is not unexpected – there is, after all, a substantial communication barrier between parent and child in these cases. Even if the parents are learning how to sign, it's likely they will focus on the basics first, such as ways to check if the child is hungry, etc. Mentalistic language is simply unlikely to be a priority in the same way, which means that there is a breakdown in reliable exposure to mentalistic language for children in this situation. Thus, there is a delay in acquiring theory of mind (or at least a delay in passing the tests for theory of mind).

A mere delay does not imply that these children never catch up, however. Indeed, most of the child's vocabulary is learned from their peers when they enter school. As Peterson & Siegal (citing Power & Carty) put it:

> *"In fact, according to Power and Carty (1990, p. 233), deaf native speakers of Australian Sign Language, or Auslan, have a unique linguistic background in that "in 90% of cases (Auslan) is learned not from parents within a family setting, but from other deaf students, usually in school". Thus, until school entry, many of these children have very restricted conversational experiences at home with family members who typically lack sufficient fluency in Auslan to readily communicate their private thoughts or beliefs to the deaf child (Peterson & Peterson, 1990)" (Peterson & Siegal 1995: 464)."*

The point for the authors is that these deaf children do not have a dearth of access to linguistic inputs forever, merely a delay as the developmental niche struggles with a breakdown in reliability. After more experience with their peers and this higher-level language, the signing children begin passing the theory of mind tests.

Theory of mind is a deep topic that I am merely skimming over here. I have included the above case study for two reasons. First, I think it is an interesting potential case study for what happens when the usual reliability in the niche breaks down. Linguistic connection and input is usually available fairly early within the niche, but even if it's not available early on, it likely will be available later since we have shaped our social niches in a way that provides some redundancy (ie schools and education, not being solely reliant on parental teaching, etc). Second, it highlights how reliability can be achieved through the shaping of the niche and the types of interactions this begets, rather than merely attributing theory of mind to some sort of inner human nature or necessity. The former offers a substantially more detailed and interesting potential research area than the latter.

The point of discussing inheritance systems and breakdowns in niche reliability, then, is to give a more fleshed-out response to the reliability challenge. I think the interactionist approach I am advocating has numerous tools with which to respond to the reliability challenge, and moreover, there are interesting research opportunities available when we find that some usually-reliable aspect of the developmental niche is not making developmental resources available in the way that it often does. I think approaching the problem in this way also ultimately offers a more satisfying solution than relying on some sort of innate content to account for reliability. Both approaches allude to teleology, but in importantly different ways, as the extended inheritance approach can attribute the teleology of some system to our behaviour in constructing the system that way. I think that is ultimately a much less mysterious sense of teleology than innate content, and it also plays out differently in terms of social evaluation. Where preformationism's innate teleology locates the problem of unexpected outcomes within the individual (ie something 'went wrong' in their development), the inheritance system's approach emphasis the relation between social values and the individual. At the very least, I think

that is more useful for potential activism, philosophical thinking, and potential positive social change.

6.3.2.2 – The Challenge of Evaluating Outcomes

With the problem of reliability out of the way, we come to the problem of evaluating outcomes and deciding between potential alternatives. The preformationist's solution to this problem, in general, has been to compare whatever outcome obtains to the outcome that is assumed to be natural. In Barker's (2015) discussion of these themes, she notes that whether some outcome is considered natural also corresponds with assumptions about how much some outcome 'costs'. That is to say, an outcome that is 'more natural' is assumed to be easier to obtain than one which is less natural, since the more unnatural outcomes must also overcome the developmental inertia of the developmental system aiming at the intended outcomes. Pinker makes use of an argument like this in his book *The Blank Slate* (2002), where he argues that some systems of social organization, government, and social intervention may only be possible through the use of overwhelming and excessive force, since we would then be trying to force a more unnatural outcome that is less compatible with human nature, and therefore is more costly to implement and maintain. But this approach can also give reasons to marshal certain kinds of resources for people; after all, if people live in areas that are afflicted with heavy metal poisoning, then poor health outcomes they experience might imply that they are not living up to what their nature would otherwise allow. In other words, we might want to choose social setups and living conditions that align with one's nature and encourage its ability to flourish. Having some sort of scale that allows for ranking potential outcomes from more to less natural ostensibly offers a way to solve this problem.

Admittedly, my response to this challenge might feel a bit underwhelming. Namely, I do not think this is a biological question, but rather is for ethics and social theory (and, importantly, that the preformationist's solution is effectively disguising some form of social philosophy as biology). That being said, I can gesture at what I think is the proper direction of an answer, but giving a full answer to this question would be the work

of an entirely separate thesis. However, I do have some thoughts on how to approach the matter.

If we accept, as I believe we should, that development is just the process of an outcome-in-transition emerging from constitutive and irreducible interactions within the niche, then there is no innate target to be aimed at. That means, prima facie, all developmental outcomes are to be desired equitably if there is no target to be missed and there are no other considerations that might break the stalemate. My claim above – that this is an ethical/social issue and not a biological one – means that I think there are not specifically biological factors that break this stalemate. However, I do not deny that there are various ethical and social reasons to potentially prefer some outcomes over others. In making this move, I am denying the charge that we have no reason to avoid certain developmental conditions or niches. In other words, I think we have reason to pursue some developmental niches and avoid others, but I think these reasons are properly construed as ethical or social reasoning, rather than strictly biological.

I think there are two threads to my response. First, if there is not an 'inner nature' that ought to be considered (remember that the postgenomic account of nature is just whatever results from the niche), then considerations about what outcomes are preferred is a question about the contingent world. This is a question that society can ask about itself, how individuals fit into it, and what types of things are valued about and within this social collective. This is why I have emphasized that I think this challenge is related more to ethics and social policy than strictly biology, but of course the two can overlap. With such overlap in mind, the second thread of my response is that questions about what outcomes are considered valuable (and what developmental contexts we want to encourage, etc) are questions about who we want to be as a society. These questions can be quite risky. After all, it is easy enough for questions about valuing some developmental outcomes over others to slip into a kind of eugenics, and I can't emphasize strongly enough how much I would like to avoid that. As such, I hope that the question is not merely what society does value, but what it ought to value, especially if we are taking seriously the dynamic character of biology. To use an easy example, we have reason to not want folks to grow up with heavy metal poisoning, not because this would block their

fulfilment of their inner natures, but because few people would consider heavy metal poisoning to be part of their conception of a good life. For similar reasons, we would not want to encourage niches that feature abuse, etc.

Lead poisoning and avoiding abuse are easy examples to appeal to, and I suspect no one would really disagree with them. But in many cases, we're primarily interested in the more socially charged examples. So let's explore that.

One such socially-charged (and recurring) example involves the existence and/or 'normalization' of trans folk. In North America (and possibly abroad, but I'm more familiar with the North American context), there are many avenues this discussion can take, ranging from debates about whether the trans identity is 'natural', to whether children should learn about topics like gender identity at school (and at what age?). As such, I cannot hope to touch on every aspect of this discussion, as doing so would take an entirely separate dissertation. Astute readers, however, can likely guess the direction of my responses to these questions given my previous answers.

In short, I take the existence of trans folk to be every bit as 'natural' as any other identity. In earlier sections of this chapter, I have emphasized that biology doesn't make mistakes, and that we can understand some developmental outcome as natural if it is a product of interactions within the developmental niche. I'm not about to hedge my bets on these matters when the topic shifts to trans folks. Their developmental outcomes are not mistakes, and by virtue of emerging within a developmental niche as a result of interactions within that niche, they are just as natural as every other outcome.

As with my response above, I think folks who are trying to argue that 'being trans is unnatural' are both mistaken about the biology, but also not actually making a biological argument. They are making a social or political argument, dressed up in the trappings of biology. This response only goes so far, however, given I am trying to give a biological answer to what I think is mostly a question of social value. The principal question is not whether certain identities are 'more natural' than others, because I have argued that this is precisely the wrong way to understand development. The principal question is a social one: what do we do about the wide range of ways people can (and

decide to!) express their identity, both in terms of gender as well as other modes of expression? Where do we draw lines around what is tolerated, what is encouraged and what is discouraged? These are, fundamentally, questions about what we value as a society. And as such, giving a full response to these questions would involve specifying what we think of the role of the state, how much we value individual freedom compared to tradition, and the like. These can be tricky questions, and deserve adequate responses. Ultimately, however, these questions are simply beyond the scope of a project that is primarily concerned with development and biology. Because that is, fundamentally, the point: these questions are not biology, nor should they be confused with it.

To round out my response to this challenge, I do have a few passing thoughts on how I would begin thinking about weighing what developmental outcomes and niches to pursue. These are, as I said, merely some thoughts in passing and do not constitute a full response to this question. My own inclination is to use something like a capabilities approach (Sen 1985, Nussbaum 2003, Sen 1993) for this type of question. The capabilities approach centers around two core tenets: 1) one's freedom to achieve well-being is paramount, and 2) freedom should be understood in terms of individual's capabilities, their real opportunities (in a given social arrangement), and the like. This conversation could be informed by getting a sense of what sorts of outcomes would be otherwise preferred by the people who have those outcomes. For instance, it seems unlikely to me that someone with heavy metal poisoning would elect to continue having it if they were offered the choice of some other outcome. Thinking about it this way may also help prevent pernicious forms of ableism or discrimination by outside perspectives undervaluing socially marginalized forms of embodiment. This conversation could of course be further informed by appealing to literature on well-being, disability, etc, as a way of fleshing out a policy-style approach to what sort of developmental niches (and associated outcomes) are to be pursued in terms of hypothetical attempts at social engineering. But as I have said several times throughout this section, sketching out the details of this conversation is the work of a different project. It is beyond the scope of the designs of this project, and so my work here comes to a close.

Afterword

I have spent six chapters outlining my concerns, critiques, and visions about an important issue in biology. I expect that my general stance on matters is clear to the reader, even if folks may have questions about the details. As a result, I am not sure what to say in conclusion that I have not already said in the previous chapters.

Perhaps the best way to begin is to look to how I started this project in the first place. I came to this project in an extremely roundabout fashion. I had interests in neuroscience, which became interests in the way that neuroscience techniques were used to investigate socially-fraught issues, such as the potential for brain-based differences between men and women. There are, to put it lightly, intense disagreements and methodological pitfalls that abound in such discussions; understandable claims of 'neurosexism', sloppy science, and more. But the longer I spent engaged with these conversations, the more I came to think that there was intense disagreement about development and the role of plasticity amidst it all. After all, what we make of differences between individuals depends on whether we think they should be similar in some respect. But if we think that people (and brains) are remarkably plastic, then why would tracking differences necessarily matter, or be especially revelatory? Surely tracking differences matters more if we think there's some intended outcome, or if the outcomes 'ought' to be more similar for some reason.

Ruminating on these topics led me down the rabbit hole of studying development. And, well, we can see how that turned out. The more I started looking at contemporary issues as problems of understanding development, the more I could see the shadow of a Naturalist position in public discourse. Folks are, understandably, fascinated by the notion of a human nature and what it means for who we think we are. By looking to the history of psychology, it was also clear to me that some form of staunch Nurturism had been part of the public discourse as well. But with the public fascination with science, especially neuroscience and gene editing, I saw claims of 'hardwired outcomes' more and more frequently, or judgements about whether some outcome was 'natural' (and tacitly, whether we should value it). Science, the public might think, is right on the cusp of

cracking the mystery of our human nature – with all these advancements, perhaps it already has.

These threads are readily traceable in my introductory chapter, where I mentioned that I am afraid of how socially pernicious the Nature/Nurture framework can be. It is too easily at hand to offer pseudo-justification in public discourse for all sorts of social ills. Attempts at so-called 'scientific racism' are not wholly behind us by any means.

To begin sorting out these issues, we needed a better foundation on which to approach the question of development. This is where my dissertation picks up the metaphorical torch. If we've been locked in a cycle between Nature and Nurture, then one way to break the cycle is to dissolve the categories involved. If we're going to do that, then a different sort of interactionism is needed, since conservative interaction serves to import problematic aspects of the Nature/Nurture framework, thus re-creating the problem all over again. Indeed, the focus on interactionism is both a blessing and a curse. On the one hand, most folks are on board with some form of interactionism already, so selling the notion of interactionism seems relatively easy. On the other hand, folks are already committed to a 'cheap' interactionism, fail to see the cracks in the foundation, and are confused when they hear claims about the need for something better. I think that all (or at least most) of the kinds of interaction that I canvassed in Chapter Two will have some explanatory power in the larger picture of development. But coupled interaction seemed the most promising to me, along with statistical interaction between independent variables, and so I have built my project on that prospect. Doing so also offered a way to fill a useful gap in the postgenomic literature. After all, I am far from the first person to question the validity of the Nature/Nurture constructs. But while some have made good attempts to get out from under this shadow, there hasn’t been a concept of interaction that could be used to help bridge the gap between disparate schools of thought. It may take some work, but my hope is that my contributions can help in that regard.

By offering a way to conceptualize interaction in this more radical way, I have attempted to engage with the evidence in genetics and biology more broadly. My aim was to show that the available evidence is too anomalous to favour the contemporary

paradigm, and that my alternative framework that focuses on irreducible interaction could better accommodate some of these findings. Ultimately, this culminated in an argument that the Nature/Nurture framework is incoherent, since there is no clean divide between phenomena that are traditionally considered part of Nature, and those considered part of Nurture. Instead, we have an overarching developmental network, where developmental conditions are constituted by the interactions from multiple forms of inheritance and local idiosyncrasies that an organism has access to within its ontogenetic niche. Development is complex, but deeply interesting, and I'm looking forward to what developments occur in this field (pun very much intended).

We come, at last, to the (current) end of this project. Thank you, dear reader, for entertaining me thus far. In the immortal words of Porky Pig: "That's all, folks!"

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