MAKING EVOLUTION RELEVANT IN PSYCHIATRY: EVOLUTIONARY DEVELOPMENTAL BIOLOGY AS A FOUNDATION FOR PSYCHIATRY

by

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DEDICATIONS

To my parents Jennifer and Michael Chalmers, for their love and support in all that I do.

To my Sister Amanda Chalmers, my counselor and best friend.

To my Grandmother Noreen, for believing in me always.

To the memory of my Aunt Gladys who encouraged my desire to learn from the very beginning.

Finally I wish to dedicate this thesis to all who suffer from mental illness of any kind.

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ABSTRACT

Evolution has tended to hold minimal significance for psychiatry making evolutionary psychiatry a niche sub discipline. This despite many calls for integrating evolution into psychiatry. One of the main impediments to this integration is that the foundation of much evolutionary psychiatry relies heavily upon a specific narrow conception of evolutionary psychology that is at times incompatible with modern evolutionary biology. Evolutionary psychiatry often produces impoverished evolutionary explanations for mental disorders as a result of relying on this impoverished foundation. I argue that a more relevant evolutionary psychiatry must reflect modern evolutionary biology and in particular the incorporation of developmental biology into evolutionary theory and study. Evolutionary developmental biology provides the adequate scientific foundation that is required for evolutionary psychiatry to provide robust evolutionary explanations and make evolution truly relevant to psychiatry. A better foundation will allow evolutionary psychiatry to expand beyond its current role as a niche sub discipline.

LIST OF ABBREVIATIONS USED

NEP	Narrow Evolutionary Psychology
Evo-Devo	Evolutionary Developmental Biology
DEP	Developmental Evolutionary Psychology
DSM	Diagnostic and Statistical Manual of Mental Disorders
SE	Selected Effect
CR	Causal Role

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CHAPTER 1: INTRODUCTION

1.1 Evolutionary Medicine

Since the publication of On the Origin of Species the science and practice of medicine has been somewhat ambivalent towards evolutionary theory. However, recently a number of theorists have argued that the knowledge that has been gained through the study of evolution can be immensely useful and even essential to medical research and treatment (Nesse et al., 2010). Writing in the Proceedings of the National Academy of Sciences (one of the major research journals in science) Randolph Nesse and colleagues suggest that the time has come to include evolutionary biology in the medical school curriculum alongside other basic sciences such as cell biology and anatomy. These advocates believe that evolutionary theory should be taught prior to and during medical school as a foundational science. Evolutionary theory is argued to be vital to the study and treatment of a variety of maladies including chronic and infectious diseases as well as mental disorders (Stearns, Nesse, Govindaraju, & Ellison, 2010).

It is the potential for an evolutionary account of mental illness, falling under the medical discipline of psychiatry that is the central focus of this thesis. Before going any further however we should equip ourselves with a basic understanding of evolution itself and its relation to the study of psychiatry.

1.2 Evolutionary Theory and Human Psychology

Darwin's greatest contribution to the theory of evolution was his concept of natural selection. Darwin's insights into natural selection can be summarized as follows:

1. Every organism has the potential to produce more offspring than can survive.

> 2. There is always variation among individuals in a population; much of this variation is inherited, so the next generation inherit some of these variable features from their parents.
> 3. Specific variations may make an individual either more or less likely to survive and reproduce than other individuals with different features.
> 4. Those variant traits that enhance survival and reproduction will be passed on to offspring and will be found in an increasing fraction of the population in each succeeding generation (Guttman, 2005, p. 65).

Darwin provided the essential ideas for understanding the process of evolution, but it was not until these ideas were united with the work of Gregor Mendel that the now dominant neo-Darwinian paradigm was formed. The rediscovery of Mendel's work in 1900 helped to establish genetics as a science and geneticists who were mathematically inclined explored the question of how genes operate in a population. Other sciences such as paleontology were also maturing adding new details to evolutionary inquiry. The culmination of this integration was summarized in Julian Huxley's work "Evolution, The Modern Synthesis" in 1942. This synthesis involved the integration of multiple disciplines into the study of evolution and is understood to have remained intact and it is argued that the modern synthesis still forms the foundation of evolutionary thought today (Guttman, 2005, p. 53).

The evolution of human behavior had been discussed since Darwin's time and today is best known in the field of evolutionary psychology. Siegert and Ward provide a common perception of evolutionary psychology (EP):

> EP's strengths include its solid grounding in evolutionary biology, which provides a unifying theory for understanding the behavior of organisms, and also the ability of the resulting theory to generate testable predictions concerning a variety of human behaviors. A basis in evolutionary biology means that EP shares the same scientific foundations as all other disciplines that study living organisms (Siegert & Ward, 2002, p. 235-36).

For the most part clinical psychology has tended to ignore evolution (Siegert & Ward, 2002). However, evolutionary psychiatry does exist as a sub discipline of psychiatry and has generated a substantial amount of literature. In 2008 a group of research physicians at McMaster University published an article calling for the integration of evolutionary theory into psychiatry (Baptista, Aldana, Angeles, & Beaulieu, 2008). The introduction of a new textbook in evolutionary psychiatry has mirrored the progression of evolutionary medicine in general (Brüne, 2008).

In this thesis I will explore evolutionary psychiatry and the commitments to evolutionary psychology upon which it rests. I will dispute the account of evolutionary psychology that currently dominates evolutionary psychiatry and suggest that while the principles of the modern synthesis do still form the basis for modern evolutionary theory, there have been significant changes in how those principles operate and in how they are now interpreted. I will argue that the account of evolutionary psychology that currently dominates evolutionary psychiatry has failed to incorporate these changes and I will offer an alternative approach to evolutionary psychology that promises to improve the future outlook of evolutionary psychiatry.

1.3 Overview of the Thesis

In the next chapter I explore the current state of evolutionary psychiatry. I rely heavily on the work of on philosopher Dominic Murphy who has written a number of articles on evolutionary psychiatry and the potential it holds for providing a causal classification of psychiatric disorders (Murphy & Stich, 2000). Murphy has attempted to capture and taxonomize explanations from the evolutionary psychiatry literature and I examine the ways in which his taxonomy is representative of the evolutionary psychiatry literature. I

find that some explanations fit under Murphy's taxonomy while other evolutionary explanations for psychiatric disorders prove to be difficult to classify under Murphy's taxonomy.

I argue that those authors whose explanations are classifiable under Murphy's taxonomy share a commitment to a specific research program in evolutionary psychology which forms the foundation of their approach to evolutionary psychiatry. This is problematic because this research program (as I will refer to it "narrow evolutionary psychology") does not represent all available biologically informed approaches to the study of the mind. In fact narrow evolutionary psychology has been heavily criticized by biologists for being a poor representative of biology and evolution in general.

I then turn my attention to the assumptions that are held in common between Murphy and other authors who utilize narrow evolutionary psychology to inform their evolutionary psychiatry. I argue that these assumptions are not consistent with modern evolutionary biology and then explain how this problem leads to an inability to account for all of the evolutionary explanations for psychiatric disorders found in the literature. I also show how these assumptions lead to difficulty in providing complete causal explanations of mental disorders. I conclude this chapter in search of a better understanding of evolutionary psychology to undergird evolutionary psychiatry. I suggest that a better account must be one that reflects the changes that have occurred in evolutionary theory since the modern synthesis and in particular the incorporation of developmental biology into evolutionary study.

In the third chapter I explore such an account. I examine the history of evolutionary theory and show that developmental biology has been a missing component

in evolutionary biology for most of the 20th century. The process of integrating development into biology is now being realized through the emerging field of evolutionary developmental biology (often called "evo-devo"). I describe the genesis of this field and provide a brief sketch of its current state. I then look at the potential for a psychological branch of this field sometimes described as "Developmental Evolutionary Psychology" (DEP). I argue that DEP is a better a foundational science for evolutionary psychiatry than the "Narrow Evolutionary Psychology" (NEP) that is currently dominates the field. This is because NEP fails to take development and evolution seriously while DEP takes both seriously and represents a conception of evolutionary psychology compatible with modern evolutionary biology. I conclude this chapter by suggesting how DEP might replace NEP and clarifying the ways in which NEP research might still be valuable.

In the fourth Chapter I argue that in light of the need for a DEP foundation in evolutionary psychiatry we should define mental disorders using causal role function, in contrast to the focus on selected effect function that follows from an approach based on NEP. I argue that using selected effect functions to define mental disorders is untenable while using causal role functions has many benefits beyond compatibility with a DEP foundation. I describe how a DEP based evolutionary psychiatry using causal role functions to describe mental disorders will lead to a different type of nosology than Murphy or Wakefield use. I then outline how a DEP based evolutionary psychiatry would operate and show that it is better situated to account for the evolutionary explanations that did not fit under Murphy's taxonomy. I then consider some objections to my view.

Finally in the fifth chapter I argue that my version of DEP based evolutionary psychiatry should be viewed as good scientific psychiatry and that evolution understood in a DEP context can help psychiatry beyond evolutionary psychiatry's current role as a niche sub discipline. I return to the question of evolution in medicine generally and suggest that a DEP based evolutionary psychiatry provides a good model for how evolution should be incorporated into medicine while the traditional evolutionary psychology based account is a model to avoid both in terms of content and method. I conclude by discussing the importance of teaching evolutionary theory in medical settings, but argue for the necessity of including a biologically realistic version of evolution.

CHAPTER 2: EVOLUTIONARY PSYCHIATRY

2.1 Introduction

In this chapter I will explore the evolutionary psychiatry literature through philosopher Dominic Murphy's attempt to taxonomize evolutionary explanations for mental disorders. I will show that Murphy's taxonomy accounts for some explanations in the evolutionary psychiatry literature; however his taxonomy fails to account for all evolutionary explanations. The inability of the taxonomy to account for some evolutionary explanations is problematic for Murphy, but the explanations that fit under his taxonomy also prove to be problematic. This is due to the assumptions and motivations held in common among the authors who propose these explanations that can be taxonomized by Murphy's proposed taxonomy. I will argue that they utilize narrow evolutionary psychology (NEP) to inform their evolutionary psychiatry and that NEP provides a collection of problematic assumptions that lead to Murphy's idea of the "intentions of nature". These assumptions and the subsequent use of the "intentions of nature" idea are not consistent with the tenets of modern evolutionary biology and they prove to be the keys to understanding why Murphy's taxonomy fails to account for certain evolutionary explanations of mental disorders. I will also argue that the explanations that do fit under Murphy's taxonomy are not capable of providing the complete causal explanations that motivate Murphy's use of evolution in psychiatry. Once again this problem stems back to the use of the "intentions of nature" idea which is not consistent with the tenets of modern evolutionary biology. At the conclusion of this chapter I will suggest that we need to look for a different approach to evolutionary psychology that can provide a better foundation for evolutionary psychiatry. This

account will avoid the problematic assumptions of NEP, be able to account for a wider diversity of evolutionary explanations and also be capable of providing complete causal explanations for mental disorders.

Murphy's extensive focus on psychiatry and particularly evolutionary psychiatry makes him an important figure to consider, as his goal is to provide an overview of evolutionary psychiatry and draw patterns through that overview. His attempt to taxonomize the literature is very informative both in terms of those who his taxonomy captures and those who the taxonomy fails to capture. Murphy is important because he does an excellent job of capturing many of the major trends in the evolutionary psychiatry literature. This is particularly important because some of these trends are problematic. Through Murphy we can gain a better understanding of the evolutionary psychology foundation of the evolutionary psychiatry literature, how it is problematic and what can be done to fix some of the problems. I will first describe Murphy's taxonomy as a way of gaining a broad understanding of the evolutionary psychiatry literature.

2.2 Dominic Murphy and Three Types of Evolutionary Explanations in Psychiatry

Murphy has argued that evolutionary science holds great potential for providing a substantial foundation for psychiatry in terms of classification and explanation (Murphy & Stich, 2000). Recently however, he has adopted a less enthusiastic view where evolution for the most part provides only a secondary role, once removed from the business of classifying and explaining psychiatric disorders (Murphy, 2006). Murphy's theoretical commitments regarding evolutionary explanations have not change so much as have Murphy's convictions about whether or not evolutionary explanations can have a

primary role in explaining and categorizing mental disorders. However, Murphy's taxonomy was designed to reflect the various types of evolutionary explanations found in the literature and so his commitments to the importance of evolutionary explanations in psychiatry are not necessarily tied to his endorsement of the taxonomy itself. Because of this Murphy continues to use his taxonomy but simply with a more skeptical attitude towards its importance in psychiatry (Murphy, 2004; 2005; 2006).

My interest in Murphy lies in what he has captured from the evolutionary psychiatry literature and not so much in Murphy's own position on the potential for evolutionary psychiatry. Therefore when I refer to Murphy hereafter I will be referring to his original convictions and positions, despite the fact that he has changed his mind toward the enthusiasm for evolutionary psychiatry that he once displayed. I will at times discuss his later changed views and I will note when I am doing this.

Murphy's taxonomy consists of dividing evolutionary explanations into three categories of explanatory types. The first explanatory type is a breakdown explanation which represents mental illness as "the failure of some component of the mind/brain to fulfill its evolutionary function" (Murphy, 2005, p. 745). Murphy specifies that he follows "the evolutionary psychology model" of the mind where the mind has specific types of mental structures (Murphy & Stich, 2000). These structures include modules, stores of non-proprietary information, computational devices that are not domain specific, and pathways for information to flow from mechanism to mechanism. These various modules are thought to have the potential to breakdown in various ways. Murphy argues that autism is an example of this type of breakdown in a module or a system of modules (Murphy & Stich, 11, 2000).

Autism involves the loss of the capacity that "normal adults" have to attribute intentional states such as beliefs and desires to other people and then to ultimately use that information to explain their actions. Autistics typically fail false-belief tests designed to test their ability to attribute intentional states. They typically fail these tests more frequently and with lower scores than children with Down's syndrome despite their grasp of general causal cognition exceeding that of children with Down's syndrome (Murphy & Stich, p. 12, 2000). Murphy sees this as evidence that there is a breakdown in the mind/brain with autism; there is a problem with a module or collection of modules and the result is the inability to attribute intentional states.

The second explanatory type is a mismatch explanation, which involves a mechanism that was once adaptive but is no longer adaptive due to a change in the environment (Murphy, 2005, p. 745). Here Murphy uses a theory by Marks and Nesse who consider the possibility that phobias are vestigial adaptations that now seem out of place in the modern environment (Murphy, 2005, p. 747). Fear of public places or fear of being far from home would have been advantageous in dealing with many dangers outside of the home range of early humans. Freezing when faced with heights could also provide advantages. People in the modern environment expressing these outdated adaptations will naturally have difficulty coping, despite the fact that "all of their mental mechanisms are functioning just as natural selection designed them to function" (Murphy, 2005, p. 748). For Murphy they are functioning as designed because "In effect, we are biologically predisposed to find certain stimuli scary on very little basis: 'minimal cues that reflect ancient dangers'" (Murphy, 2005, p. 748). Mismatch explanations

therefore focus on explaining mental disorders in terms of adaptive strategies that are no longer suited to the environment we are currently living in.

The third explanatory type is a persistence explanation, involving disorders that arose as adaptations and remain adaptive in current environments (Murphy, 2005, p. 745). Persistence explanations focus on explaining mental disorders in terms of traits that are problematic for the patient or for society, but nonetheless arose because they were adaptive and continue to be adaptive today. Anti-social personality disorder is an example Murphy offers. This disorder involves patients who are disrespectful of the rights, wishes and feelings of others. Impulsivity, aggressiveness and neglectful behavior are common. In their quest to gain money, sex and power these people can be deceitful and manipulative and often think that everyone is only looking out for themselves (Murphy, 2005, p. 757).

Murphy argues that if we consider the function of a psychological unit to be the effect it has on being passed to subsequent generations, then we can explain some disorders like this one in a historically adaptive manner (Murphy, 2005, p. 758). Traits that lead to aggressively seeking resources and sex were likely to have been adaptive in the evolutionary past. These character traits thus arose as adaptations and are still functioning as they did when they arose. They were adaptive then, and they remain adaptive now if we assume the environment is still similar in relevant ways. These people are therefore functioning according to their naturally selected function and we cannot attribute to them "malfunctioning mental mechanisms" (Murphy, 2005, p. 759). While we might desire to label these people as having disorders, if no evolutionary module is broken then Murphy argues that we cannot use a breakdown explanation to

attempt to understand the mental disorder. If the environment is similar enough to maintain the adaptive status of the trait then we can't be said to be operating in the wrong environment either. In this way persistence explanations are distinguished from breakdown and mismatch explanations.

Murphy originally felt that the distinctions found in his taxonomy are robust and represent important differences between the types of psychiatric disorders and explanations that will be contained in each category. Murphy describes:

> A quite fundamental distinction between those disorders that arise from the malfunction of a component of the mind and those that can be traced to the fact that our minds must now function in environments that are very different from the environments in which they evolved. This mis-match between the current and ancestral environments can, we maintain, give rise to serious mental disorders despite the fact that, in one important sense, there is nothing at all wrong with the people suffering the disorder. Their minds are functioning exactly as Mother Nature intended them to (Murphy & Stich, 2000, p. 62).

Murphy highlights the distinction between the first type of evolutionary explanation involving the loss or break down of an evolved mental mechanism and the second type of evolutionary explanations involving mismatched environments. In the first case the mind is malfunctioning internally and so failing to function "as nature intended", while a mental disorder in a mismatched environment represents a problem that results from continuing to function "as nature intended" internally, but simply using that function in a novel environment.

This distinction marks an important causal difference between what Murphy sees as two different types of mental disorders. Murphy thinks this distinction is important for categorization and explanation despite the fact that this causal difference has traditionally not been recognized by psychiatric professionals. For example, Murphy points out that this distinction is one that would not be recognized by the Diagnostic and Statistical

Manual of Mental Disorders (DSM) which is a publication produced by the American Psychiatric Association (APA) aimed at cataloguing mental disorders. This manual is often described as a "bible for psychiatrists" and has the following aims:

> The purpose of DSM-IV is to provide clear descriptions of diagnostic categories in order to enable clinicians and investigators to diagnose, communicate about, study, and treat people with various mental disorders (American Psychiatric Association, 2000, Introduction, p. 13)

The taxonomy that the DSM produces is expected to be used in the following way:

> The specified diagnostic criteria for each mental disorder are offered as guidelines for making diagnoses, because it has been demonstrated that the use of such criteria enhances agreement among clinicians and investigators. The proper use of these criteria requires specialized clinical training that provides both a body of knowledge and clinical skills (American Psychiatric Association, 2000, Introduction, p. 13)

For Murphy a causally based taxonomy is better than the current symptom based approach utilized in the DSM:

> DSM-IV is a categorical classification that divides mental disorders into types based on criteria sets with defining features.... In DSM-IV, there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder. There is also no assumption that all individuals described as having the same mental disorder are alike in all important ways. The clinician using DSM-IV should therefore consider that individuals sharing a diagnosis are likely to be heterogeneous even in regard to the defining features of the diagnosis and that boundary cases will be difficult to diagnose in any but a probabilistic fashion (American Psychiatric Association, 2000, Introduction, p. 9)

However, the DSM also notes that:

> A categorical approach to classification works best when all members of a diagnostic class are homogeneous, when there are clear boundaries between classes, and when the different classes are mutually exclusive (American Psychiatric Association, 2000, Introduction, p. 9)

The DSM suggests that it does not have the tools for this ideal situation, and Murphy sees his first distinction between breakdown and mismatch explanations as a step toward

filling in this gap. Murphy is therefore motivated to provide distinctions that are more causally based than the DSM and therefore provide a more homogeneous diagnostic procedure. Murphy makes a second equally important distinction aimed at this goal.

He argues for a second causal separation between the persistence category and the other two explanatory categories (breakdown and mismatch). Murphy refers to people who have problems that fall under breakdown or mismatch explanations as having E-M problems (problems that may beset an evolved mind). These problems are distinct from the problems that would be taxonomized in the persistence category of evolutionary explanations:

> If, as McGuire and others have suggested, the mechanisms underlying various sorts of personality disorders are adaptations that evolved in environments which were relevantly similar to the modern environment, then people with these conditions do not have E-M problems, and thus, we maintain, they do not have mental disorders (Murphy & Stich, 2000, p. 90).

Here Murphy argues that some evolutionary explanations for mental disorders give us reason to believe that these behaviors are not mental disorders at all. If these traits are functioning as they were designed to function and in a relevantly similar environment to the one in which they first evolved, then they should not be considered mental disorders. These persistence "disorders" can be explained by their continued adaptive value and so although we desire to call them mental disorders (and currently do) because they are unpleasant for the person afflicted or for society in general, we can't really consider them to be mental disorders at all. This is in contrast to breakdown explanations or mismatch explanations which are to be counted as mental disorders.

Murphy's comments on the importance of these distinctions help to highlight the two goals that motivate Murphy in the creation of his taxonomy. His first goal is to

accurately classify evolutionary explanations for mental disorders from the evolutionary psychiatry literature into different categories of explanation types. His second goal is to have the distinctions between types of evolutionary explanations capture important causal distinctions provided by an evolutionary perspective.

Murphy's first goal of categorizing evolutionary explanations is intended to aid in psychiatry's goal of categorizing mental disorders and distinguishing mental disorders from normal behavior. Murphy's second goal aims at ensuring this psychiatric taxonomy for mental disorders is causally based and avoids the problematic distinctions in the DSM that rely on symptom based criteria. Murphy's second goal therefore aids psychiatry by providing scientifically grounded causal criteria that can provide us with information about the mental disorders themselves such as how they originate and operate. This can theoretically lead to better diagnostic tools and treatment options.

It is important to realize that if Murphy achieves these two goals he is ultimately making two distinct claims. If he achieves his first goal he is claiming that his taxonomy reflects and captures the evolutionary psychiatry literature and that it can therefore help psychiatry to categorize mental disorders. Murphy thinks this is justified if he achieves his second goal of ensuring that the criteria by which his taxonomy operate reflect causal distinctions. Murphy argues that his two goals are met and therefore claims that his taxonomy does indeed reflect the evolutionary psychiatry literature and that the taxonomy uses causal distinctions that can explain mental disorders. I will examine both of these claims in this chapter starting with Murphy's claim that his taxonomy captures the evolutionary psychiatry literature.

2.3 Categorizing Evolutionary Explanations in Psychiatry using Murphy's Taxonomy

I begin by looking at researchers from the literature whose work seems to fit easily into Murphy's taxonomy and later I will examine some work that does not seem to fit very easily into the taxonomy. In Evolutionary Psychiatry: a New Beginning Anthony Stevens and John Price provide evolutionary explanations that fit squarely into Murphy's taxonomy (1996). These authors see the key insight of evolutionary psychiatry to be the fact that ancient adaptive strategies utilized in a modern environment can lead to mental disorders. They argue that we are adapted to a period of time known as the Environment of Evolutionary Adaptedness (EEA) which refers to a proposed aggregate of environments that contained the environmental selection pressures that gave rise to the human species 1 to 2 million years ago (during the Pleistocene Epoch). For example, they state:

> It can be seen, therefore, that the psychiatric states of anxiety and depression are natural and universal experiences which human beings share with all mammalian species. Both are pathological exaggerations of biological conditions which, in the EEA, contributed to survival (Stevens & Price, 1996, p. 51).

Anxiety and depression held an adaptive function during the EEA that has now become pathological due to a change in the environment that has been too rapid for evolution to adapt to.

These explanations are easy to classify because they have the hallmarks of a mismatch explanation. They explain that the trait was adaptive in a past environment, that the current environment does not match that past environment and that the mental disorder arises because of this mismatch. Stevens and Price think that mismatch

explanations are what is unique about evolutionary psychiatry and so focus heavily on these types of explanations (1996).

Jerome Wakefield on the other hand is a prominent defender of evolutionary explanations that fall under the breakdown category of evolutionary explanations. He holds that in order for us to have a mental disorder we must experience what he describes as a "harmful dysfunction" described as follows:

> A disorder is a harmful dysfunction, wherein harmful is a value term based on social norms, and dysfunction is a scientific term referring to the failure of a mental mechanism to perform a natural function for which it was designed by evolution (Wakefield, 1992a, p. 373).

Wakefield is committed to explaining all mental illness not only in terms of evolutionary explanations but specifically in terms of breakdown explanations. A dysfunction is therefore a necessary prerequisite for a mental illness to exist and "a dysfunction exists when a person's internal mechanisms are not able to function in the range of environments for which they were designed" (Wakefield, 1992b, p. 243).

Although Wakefield is committed to using only breakdown explanations Murphy does not require that any one person explain all disorders as mismatch explanations or all disorders as breakdown explanations (Murphy, 2004, p. 329). One person or one research program can provide evolutionary explanations for a variety of mental disorders that fall under all three of the categories in Murphy's taxonomy. Although Stevens and Price focus on mismatch explanations there is nothing to stop them from suggesting that some mental disorders are better explained as breakdown explanations or even persistence explanations. Of course this means that we should also expect a single mental disorder might have multiple explanations that each focus on a different type of evolutionary explanation.

Murphy is well aware that we may find multiple evolutionary explanations for the same mental disorder and his taxonomy can handle Wakefield providing a breakdown explanation for a mental disorder while Stevens and Price provide a competing mismatch explanation for that same disorder. In fact Murphy provides panic disorder as an example of a mental illness that can be explained using competing explanations that fall under each of the three different categories of evolutionary explanations from the taxonomy:

> Depending on how one reads some central ideas of the theory, it can be read as a breakdown-type explanation (if anxiety-producing mechanisms are presumed to be dysfunctional); as a mismatch explanation (if we assume phenotypic variation rather than dysfunction but lack of adaptiveness in the modern environment); or as a persistence explanation (if we assume...continued utility in some environments) (Murphy, 2005, 752).

Therefore three different people can explain panic disorder with three different evolutionary stories that each fit into one of the three different categories of evolutionary explanation. It would then seem to become an empirical question as to which explanation is true. Murphy is simply interested in categorizing the explanations and arguing that they are different. His taxonomy does not provide criteria for determining if one explanation is more likely to be true, although the taxonomy does suggest that *if* the behavior is explained by a persistence explanation then it should not be counted as a mental disorder. If panic disorder turns out to be a breakdown explanation then this suggests a kind of causal similarity with other breakdown explanations that differentiates it from mismatch explanations and the similarities they share. If panic disorder turns out to be a mismatch explanation then the reverse would of course be true.

In this section I have shown examples of evolutionary explanations that are easily categorized by Murphy's taxonomy. I have also described how competing evolutionary

explanations can be taxonomized and coexist until empirical evidence decides which explanation is correct. In the next section I will examine three different evolutionary explanations from the literature for Obsessive Compulsive Disorder (OCD) and suggest that Murphy's taxonomy cannot categorize them as each explanation appears to meet the criteria for more than one of Murphy's categories. I will preface the discussion of the OCD examples with an explanation of why it is problematic if Murphy's taxonomy cannot place evolutionary explanations into a single category.

2.4 Obsessive Compulsive Disorder and Murphy's Taxonomy

In the previous section I discussed dealing with competing evolutionary explanations in Murphy's taxonomy. For example, a breakdown explanation might try to explain the same mental disorder as a mismatch explanation. Murphy implies that empirical evidence can be left with the task of deciding which explanation is true or best supported. Murphy's main objective is to categorize different types of causal explanations regardless of their epistemic status and his success of failure depends on how well he captures the literature and categorizes these explanations according to the causal factors that are at work in each explanation.

In this section I will argue that some evolutionary explanations cannot be taxonomized using Murphy's taxonomy. The problem is not an epistemic one but an ontological one. These explanations cannot be categorized using Murphy's taxonomy because even if we were certain one explanation was true or had better evidence over other explanations we would still be unable to assign any of the explanations to one of Murphy's categories. This is because Murphy's taxonomy rests on the principle of distinguishing and *separating* distinct causal factors. If the taxonomy is to be successful

in this goal then individual evolutionary explanations must be categorized mutually exclusively according to their appropriate causal type. Each explanation must fit into only one of the three categories according to the category of causal factors we are appealing to in the explanation (breakdown, mismatch, persistence). If an explanation appears to meet criteria of more than one category then this threatens to undermine Murphy's claim that his taxonomy can categorize evolutionary explanations for mental disorders into three distinct causal types. I will now present three different evolutionary explanations for OCD that I will argue can be categorized into more than one category from Murphy's taxonomy.

Some have proposed that OCD should be understood as a "dysregulation of evolutionarily conserved behaviors and mental states critical to human survival" (Feygin, Swain, & Leckman, 2006, p. 855). Feygin and colleagues describe studies that have shown a strong continuity between the types of obsessions and compulsions in the population at large and those in people with OCD. Individuals with OCD tend to have an increased number of compulsions that are more severe, longer lasting and more difficult to dismiss than the compulsions experienced by the general population. The obsessions and compulsions also tend to cause a greater negative strain on those with OCD. Despite the difference in severity and impact of symptoms even experts are often unable to note any difference in type between normal and abnormal compulsions (p. 856).

Neurological evidence is also presented to support the dysregulation theory, purportedly showing that OCD is similar to but more extreme than the usual experiences of compulsion in the general population. For instance, OCD is also argued to share many similarities with romantic love, religious ritual, and parental love. Comparisons between

patients with OCD and people who have recently fallen in love show similar lowered neurochemical binding sites as well as similar activity patterns and behaviors (p. 859). Studies also have linked higher religious tendencies with higher OCD tendencies. Feygin *et al* argue that this suggests that OCD represents the extreme on a continuum of adaptive behaviors that are seen across populations and cultures.

Feygin *et al* further this point by examining studies in ethology. In some non-human animals we see many complex and repetitive tasks designed to shield newborns from harm such as amniotic sack cleaning, bathing newborns and repetitive licking and grooming. It is argued that these adaptive behaviors may degenerate into obsessive behaviors if there are changes in the regulation of this behavior. When certain genes associated with development in mice have been manipulated they have shown the capacity to develop excessive grooming patterns to the point of self harm (Feygin et al, 2006, p. 860).

Understanding OCD as a dysregulation of an adaptive trait seems to suggest categorization under Murphy's breakdown category. The adaptive trait or traits are ones that we use to imagine the future and anticipate danger. We might say that there is a problem with the traits that sense danger themselves or we might want to say that there is only something wrong with the regulation system of these traits. In either case there is reason for thinking that something functional has broken down in people with OCD. A properly functioning threat detection mechanism will allow a person to look once to make sure their baby is sleeping safely, while OCD may cause someone to check the baby repetitively for hours becoming tired and ultimately reducing their effectiveness as a parent.

However, talk of OCD representing an extreme of normal behavior, a difference in intensity but not of kind, does not seem to fit with our understanding of traits being functional or dysfunctional under the breakdown category. We might consider categorization under the mismatch category instead. Perhaps in our evolutionary past the range of threat detection behavior was adaptive, but the environment has changed over time and people on the extremes are now out of place. That range of the spectrum is simply not adaptive any longer and so this explanation could also be classified as a mismatch explanation. It seems as though we have cause to classify this explanation as either a mismatch or a breakdown explanation.

Another theory has suggested that the extremes of OCD should be viewed as a group selection story (Polimeni, Reiss, & Sareen, 2005). OCD is argued to have been beneficial in early hunter-gatherer societies, where a division of labor may have been important to survival. The focus of many obsessional thoughts and behaviors is safety and security and detailed attention to these issues may have been vitally important to the survival of early communities. One person specialized in dealing with threats may work in a group scenario where the other needs of that persons are met by the group (p. 658).

In this case we might view OCD as an adaptation that is currently adaptive. This group selection model suggests that at the very least OCD was adaptive in the past (Polimeni, Reiss, & Sareen, 2005). However the focus of many obsessional thoughts and behaviors is safety and security and these concerns are still very important in the modern day. If OCD was adaptive in the past and our modern social structures are similar in terms of diffusion of responsibility for anticipating danger then OCD may very well be adaptive in some modern environments as it may still improve chances of survival and

reproduction. Of course this would be the case only if the environments are still relevantly similar.

If the environments are not still relevantly similar then OCD may be better classified as a mismatch explanation because there is no longer any adaptive value to the trait. Of course we may face a situation in which living in certain environments make it adaptive for some while living in other environments make it maladaptive for others. In this situation we may not be able to place this explanation into either category as it would be a mismatch explanation for some and a persistence explanation for others.

A third evolutionary explanation is proposed by Martin Brüne who notes that attempts to model OCD using animal models are limited in that they fail to account for the cognitive symptoms of OCD. We can include the cognitive by viewing OCD as an extreme on a continuum of evolved harm-avoidance strategies (Brüne, 2006, p. 317). Brüne argues that a potential causal contributor to OCD involves the imagined consequences of our own thoughts and actions (Meta representations) becoming exaggerated and thus pathological. Therefore OCD is best viewed as a costly by-product of the adaptive ability to anticipate needs or threats in the future (Brüne, 2006, p. 317).

It is not immediately clear where this explanation should fit. We might think it is a breakdown explanation where some people experience dysfunctional behavior as a by-product of the adaptive ability to anticipate needs or threats in others. Perhaps the imagined consequences of our own thoughts and actions are adaptive when they target consequences above a certain probability threshold. However, these might be considered exaggerated and thus pathological beyond that certain threshold. It is only adaptive to imagine consequences that have a high enough probability of occurring to offset the

evolutionary cost involved with putting resources towards considering these outcomes. Spending a great deal of time considering needs or threats that may be possible but are highly unlikely can be considered a breakdown of the original design of the system which can be understood to include considering only probable (beyond a certain threshold) threats and needs. The "intentions of nature" would not allow a system that spent costly evolutionary resources attempting to anticipate threats which were very unlikely or even threats that were only of minimal danger.

However, when viewed in a group selection sense this explanation could become a persistence explanation where individuals experience negative fitness but the group overall develops an important ability. This is the opposite of the group selection explanation offered previously. In that explanation it was the people afflicted with OCD (a small minority) that provided the group with the benefit. Instead this explanation might be construed as one in which the vast majority were provided with a functional threat detection system at the cost of a few people dealing with an overactive detection system. Viewing this trait in the group sense it might be impossible for the "intentions of nature" to be fine tuned enough to specify the threshold of probability that legitimizes a potential need or threat.

It might be the case that in order to develop the best possible threat detection system for the group some members of the group must have an overactive system as a by-product. In order to ensure that no one had OCD natural selection might need to reduce the efficiency of the threat detection system of the majority by 50% which may not be a reasonable trade off. Therefore the reason we see the pattern of OCD behavior we do today is because this pattern was adaptive in a group selection sense in the past and we

understand the people with OCD as part of the process of developing and maintaining the highly adaptive threat detection systems that exist in the majority of the population today.

This explanation could also be described as a mismatch explanation. The threats of the past may have been so dangerous that had we encountered them even once we would have been killed. In terms of a fight of flight mechanism activating numerous unnecessary flight mechanisms is better than failing to activate a necessary one that prevents death. The moral "better safe than sorry" is highly relevant when a single mistake can prevent any genes being passed on to future generations. This is Nesse's "smoke detector principal" (Brüne, 2006, p. 320).

Even if some members of a species are overly attentive to danger, they are still better off or at least were better off in past environments than individuals that were under-attentive to these dangers. The modern environments that we occupy typically have less of a need for fight or flight mechanisms, however, we inherit this trait from our ancestors and our thoughts and behaviors appear inappropriate in the modern environment. Those on the extreme range of the behavioral spectrum therefore become pathological and we consider them to have OCD. Brüne's explanation of OCD as a by-product of an adaptive trait therefore has signs of belonging in all three of Murphy's categories which is highly problematic for Murphy's taxonomy.

The possibility that these explanations actually belong in more than one category is implicitly rejected by Murphy's motivations for the taxonomy in the first place. The purpose behind Murphy's taxonomy is to separate different types of explanations based on causal factors. For Murphy we have seen that a persistence explanation should not be considered a mental disorder while breakdown and mismatch should be considered so.

Persistence explanations arise because of adaptive reasons, and the "intentions of nature" are not violated in these explanations. In mismatch explanations the "intentions of nature" are not violated but expressed in a changed environment. Breakdown explanations arise because of an internal change to the individual and the "intentions of nature" are violated because of that internal change. These distinctions motivate the taxonomy.

Therefore, if we classify an evolutionary explanation as both a breakdown and a persistence or mismatch explanation we are concluding that the persons mind is functioning as it was designed to function and at the same time failing to function as it was designed to function, a clear contradiction. Also categorizing an evolutionary explanation as a persistence explanation as well as a breakdown or mismatch explanation would be claiming that the behavior in question was both normal behavior (and therefore not a mental disorder) and at the same time claiming that it was a mental disorder. Finally claiming that an evolutionary explanation was both a breakdown and a mismatch explanation would be claiming that the cause of this mental disorder was rooted in both the environment and an internal change in the person. This is not a contradiction and certainly seems possible perhaps even probable, but it does undermine the entire purpose of Murphy's taxonomy which is to separate evolutionary explanations based on whether or not they were caused by a change in the environment or a change internal to the person (or no change at all as in persistence explanations). Therefore, for Murphy's taxonomy to remain consistent and useful Murphy's categories must be understood to be mutually exclusive in the sense that one evolutionary explanation cannot be classified into more than one category.

It is then highly problematic that Murphy's taxonomy does not seem capable of classifying any of the evolutionary explanations for OCD that I have presented into single categories. There does not appear to be any way to objectively decide which single category to categorize them into and attempting to classify them into multiple categories leads to a contradiction or renders the entire taxonomy useless. The taxonomy can handle some evolutionary explanations as we saw with Wakefield and Stevens & Price, but it cannot deal with other explanations such as the OCD explanations just described. In order to understand why this is the case we must look at Murphy's idea of functioning "as nature intended" which is at the heart of his taxonomy and much of evolutionary psychiatry. I will argue that this idea arises from Murphy's commitment to a specific conception of evolutionary psychology.

Murphy relies on evolutionary psychology in order to help him determine the nature of normal psychology from which we ascertain abnormal psychology. In the next section I will show that Murphy's taxonomy shares some problematic assumptions with the dominant paradigm of evolutionary psychology known as Narrow Evolutionary Psychology (NEP). I will then argue that these common assumptions lead to the "as nature intended" idea which is at the root of the failure of Murphy's taxonomy to adequately deal with some evolutionary explanations for mental disorders such as the OCD examples we have just seen.

2.5 Narrow Evolutionary Psychology

What is often called "evolutionary psychology" usually represents a specific mainstream approach but does not typically represent the full range of evolutionary approaches to studying the mind. This specific approach has many monikers but following Rauscher

and Scher I will use the term "Narrow Evolutionary Psychology" (NEP) to refer to it (2003).

Adherents to NEP hold that the human mind is composed of specific adaptations to environmental problems that arose in the Pleistocene Epoch (2.5 Million years ago to 10,000 years ago). These adaptations are understood as modules and the period in which they arose is referred to as the Environment of Evolutionary Adaptedness (EEA) (Downes, 2010, p. 244). The EEA does not refer to a specific place or time in history, but represents a statistical composite of selection pressures that existed 1 – 2 million years ago when hominids with large brains originated. The Pleistocene Epoch is therefore the time period believed to contain the EEAs of most human-specific adaptations.

Although hominids with large brains and advanced tool technologies have been extant for one to two million years, it is held by narrow evolutionary psychology that for 99% of this time period humans existed in a nomadic state living in smaller groups subsisting off a hunter-gatherer lifestyle. The other 1% that falls outside the Pleistocene composes the previous 10,000 years of history (the Holocene), a relatively short evolutionary time span during which agriculture has become prominent in human life. No significant genetic change is argued to have occurred in this period, and therefore narrow evolutionary psychologists believe that we are genetically adapted to a hunter-gatherer lifestyle in the Pleistocene. Here I present six essential assumptions of NEP as described by two of its central proponents Cosmides and Tooby:

> 1. Each organ in the body was evolved to serve a purpose, and the brain's purpose is to extract information from the environment. The brain is a computer designed by natural selection that is composed of programs (Modules) which have solved adaptive problems in past environments.

> 2. The behavior of an individual is generated by this computer in responding to information from the internal and external environments. Understanding the modules structures and informational input allows us to understand the behavior of an individual.
>
> 3. All of the modules in the human brain were designed by natural selection in response to selection pressures that we experienced as hunter gatherers. The modules that survive today were better at solving adaptive problems than the alternative programs that arose in our evolutionary past.
>
> 4. The behavior that the modules generate would have been adaptive in our ancestral environments, but it may no longer be adaptive now. Our cognitive modules that exist today were designed for a past environment.
>
> 5. Natural selection has ensured that the brain is made up of many different modules almost all of which are specialized for solving adaptive problems. The evolutionary process has not and will not produce "a predominantly general-purpose, equipotential, domain-general architecture".
>
> 6. These modules invite us to think certain kinds of thoughts; they make certain ideas, feelings and reactions seem reasonable, interesting and memorable. In short, they play a key role in determining the ideas and customs that will easily spread from mind to mind and those that will not. Ultimately, they play a crucial role in shaping human culture" (Cosmides & Tooby, 2005, p. 16-17).

Although all 6 of these assumptions have been questioned, in this section I will focus on points 3 and 5 and argue that they are not legitimate assumptions to hold.

Critics have argued that proponents of NEP assume that significant evolution has not occurred in the last 10,000 years (assumption 3) because they ignore the type of evolution that is likely to have occurred during the Holocene. Niche construction for example studies the way in which organisms construct different parts of their own niches, thus altering their own selective environments. Examples include nest building, dam building and for humans building dwelling structures. Kim Sterelny (2003, as cited in Downes, 2010, p. 249) argues that niche construction in humans also contributes to novel behavioral traits. Niche constructionists are apt to think that the past 10,000 years may

have in fact produced the highest degree of evolutionary change in the human species, obviously rejecting the premise that the human mind is purely a product of the Pleistocene.

If we were to accept the NEP assumptions we would never think to test for the effect of niche construction on human psychology because we assume that no evolution occurred during the Holocene, the time period during which humans stopped a nomadic lifestyle and instead took up agriculture. Of course, evolution involving niche construction is more likely to have occurred when humans had a constant habitat which only occurred after humans gave up a nomadic lifestyle during the Holocene. By assuming that no important evolution occurred in the Holocene we are prevented from even asking about the potential impact that niche construction might have had on the evolution of human brains.

Stephen Downes criticizes NEP's assumption 5 which holds that our mind is composed of modules that are designed by natural selection to solve specific environmental problems. He claims that proponents of NEP are failing to follow standard evolutionary methodology. Downes suggests that we should approach the study of behavioral and mental traits in the same way that we study any other trait or cluster of traits. As an analogy, we are asked to imagine investigating the evolutionary history of the human hand (Downes, 2010, p. 249). We can construct a list of many different tasks that we can do with our hands which our closest primate relatives cannot do. We can sew, make fish hooks and nets, tie knots, play instruments, roll cigarettes and so forth. Part of this investigation must also include reference to the fact that our hands evolved from mammalian forefeet. The structure of mammalian forefeet constrained the ways in

which they could change through selection pressures to ultimately evolve into human hands. Why the hand is the way it is, owes a lot to the fact that it evolved from a specific ancestral structure.

In terms of a physical trait such as the hand, we would not think that it evolved in response to a specific environmental stimulus at a specific point in time. The ability to tie knots is not the only selection pressure we would expect natural selection to respond to. Instead we are apt to think that there are many selection pressures which contributed to the development of the human hand through multiple time periods. The influences on the emergence of the hand include the bipedal nature of humans, niche variation leading to varying food resources and our own niche construction. These all influenced the structure of the human hand so that it can do the various tasks it is capable of doing.

One hypothesis suggests that throwing and clubbing are largely responsible for driving the evolution of the human hand:

> The idea is that throwing and clubbing both pre-date stone tool use and throwing and clubbing grips are present in all humans from Australopithecus onwards, but in no ape lineage (Young as cited in Downes, 2010, p. 249).

Even if this hypothesis is correct, if our hands were actually specifically adapted only for clubbing we might find it difficult to repair fishing nets and to make and use needles and so forth. Nonetheless, a useful course of study is determining the role that differing throwing and clubbing grips had in determining how our lineage diverged from the other great apes. Downes concludes that for any trait or set of traits there are typically many evolutionary time periods which are important to the explanation of those traits. It is thus quite difficult to claim that there was a single set of adaptive problems that the hand faced, and analogously we should not expect a single set of adaptive problems in a single

time period to be predominately responsible for the various functional traits that make up our brain and psychological traits that characterize our behavior (Downes, 2010, p. 250).

Although our brains are more complex than our hands we should expect a similar story about the brain's evolutionary history. The evolution of the human brain was also likely affected by bipedalism, variation in food resources as well as niche construction. We should also consider that the brain's evolution was also likely driven in part by the evolution of the hand. When our hands develop fine-grained motor skills we require increasing monitoring of these skills from the brain.

I have now laid out the foundation of NEP and looked at two criticisms of it. I will next show the influence that NEP holds on many authors in the evolutionary psychiatry literature by looking at problematic assumptions such as those I have just discussed which are imported from NEP into work in evolutionary psychiatry. I will argue that Murphy's taxonomy represents those in the literature who use NEP as a basis for evolutionary psychiatry. Then I will be able to show that Murphy's taxonomy does not work well when the assumptions of NEP are not maintained in evolutionary explanations.

2.6 NEP and Evolutionary Psychiatry

Murphy utilizes what he describes as the "evolutionary psychology model" of the mind (Murphy & Stick, 2000). This is not surprising as others have noted that NEP typically provides the basis for evolutionary psychiatry (Siegert & Ward, 2002). Murphy quotes from Cosmides and Tooby to describe what he understands the "evolutionary psychology model" of the mind to be:

> Our cognitive architecture resembles a confederation of hundreds or thousands of functionally dedicated computers (often called modules) designed to solve adaptive problems endemic to our hunter-gatherer ancestors. Each of these devices has its own agenda and imposes its own exotic organization on different fragments of the world. There are specialized systems for grammar induction, for face recognition, for dead reckoning, for construing objects and for recognizing emotions from the face. There are mechanisms to detect animacy, eye direction, and cheating. There is a "theory of mind" module.... a variety of social inference modules.... and a multitude of other elegant machines. (As quoted in Murphy & Stich, 2000, p. 66)

Although Murphy relies on Cosmides and Tooby for his understanding of how the mind works in an evolutionary context he also states that:

> There is no reason to suppose that all of the mechanisms to be found in the mind are plausibly viewed as modular. In addition to the swarm of modules, the evolutionary psychology model of the mind can accommodate computational devices that are not domain specific, stores of information that are not proprietary, and a variety of other sorts of mechanisms (Murphy & Stich, 2005, p. 5).

So Murphy utilizes the NEP conception of the mind but disagrees with the extent to which Cosmides and Tooby assume modularity in the mind. Murphy allows the methodological adaptationism of NEP as useful and legitimate but he denies that this evolutionary method should be used to generate conclusions about the architecture of the mind (Murphy, 2003).

Another way of connecting NEP, Murphy and evolutionary psychiatry is through the figure of Jerome Wakefield. We have seen that Wakefield's definition of a mental disorder constituting a "harmful dysfunction" fits squarely into Murphy's category of breakdown explanations. Wakefield connects NEP and Murphy because Wakefield's harmful dysfunction analysis is directly endorsed by Cosmides and Tooby. They support the definition of disorder being a harmful dysfunction because it recognizes that "the evolved human architecture consists of a collection of functional mechanisms that may

potentially be impaired and whose impairment may be harmful" (Cosmides & Tooby, 1999, p. 453).

These authors are all connected by a number of assumptions held in common which are derived from NEP. They all think that natural selection designed the mind to solve specific adaptive problems in response to specific ancestral selection pressures of our evolutionary past, specifically the EEA in the Pleistocene epoch. There is therefore a "human nature" that arises out of this collection of modules or mechanisms. There has been insufficient time since this period for any significant evolutionary change to occur and so our brains house a "stone age mind" and the "human nature" that originated in the past still defines us today.

There is some variation among the authors in terms of the form that they each envision evolved functional capacities to take. The authors agree that the functional capacities are solutions to environmental problems, but use different terms for the units that these capacities should be described in.[1] These differences can be important and as we saw with Murphy not everyone takes everything from NEP verbatim but the generic commitments I describe here are typically shared in common and these are the commitments that I shall argue in the next section are captured by Murphy's taxonomy.

[1] Cosmides and Tooby use the term "module" and describe our capacities as programs, while Wakefield often uses the term "mechanism". Murphy uses both of these terms at different times as well as terms like "psychological unit" (2005, p. 758). These terms are all describing solutions to adaptive problems from the past, but there are differences. Murphy follows Cosmides and Tooby in their understanding of the mind, but worries that they may be too modular in their thinking. It is also not clear that Wakefield's mechanism needs to be as modular as Cosmides and Tooby either, although Wakefield does cite Cosmides and Tooby (Wakefield, 2001) and as I have noted Cosmides and Tooby approve of the approach Wakefield takes. The many similarities and cross citations suggest that these differences are fairly minor and have to do with the extent of modularity rather than distinctly different conceptions of these functional capacities.

For now I wish to explore a second set of assumptions that arise from these commonly held NEP assumptions.

Cosmides and Tooby share with Wakefield a common belief that an evolutionary perspective on psychology can "provide an objective basis for analyzing function and dysfunction" (Cosmides & Tooby, 1999, p. 457). We discover these functions objectively in terms of how natural selection has solved various adaptive problems. This is the methodology Murphy uses to frame his taxonomy and he seems to be correct that this kind of thinking is prevalent in at least some areas of evolutionary psychiatry. Murphy uses the NEP understanding of the mind so the outcomes of natural selection operating are best understood as specific adaptive solutions to specific adaptive problems. So when Murphy anthropomorphizes natural selection and talks about the "intentions of nature" he appears to be referring to these solutions to adaptive problems that constitute the modules or mechanisms of NEP. We can therefore gauge whether or not the "intentions of nature" are being fulfilled by ascertaining whether or not the modules are functioning in order to solve the adaptive problems they arose to solve.

These assumptions are also present in the work of Stevens and Price. Their mismatch explanations require that past adaptive solutions are still present today in at least some individuals, some of the time. Anxiety and depression are thought to be universal experiences, easily translatable into a conception of human nature. This helps focus us on the question as to why some people are able to cope with these experiences better than others. This can be captured by the "intentions of nature" being affected by environmental change. Anxiety and depression were once adaptive, and now are not. The "intentions of nature" are being expressed but in a different environment than the one

that made the behavior adaptive originally. Some people on extreme ends of a spectrum of behavior might experience problems because of this different environment.

The term "intentions of nature" is used by Murphy but it also captures an important idea that is shared by Cosmides and Tooby along with proponents of evolutionary psychiatry such as Wakefield. This is the idea that it is essential to think about evolutionary psychology and evolution in general as being extremely goal directed to the point that it is useful to anthropomorphize natural selection as having intentions. There are two methodological assumptions that come out of the "intentions of nature" idea and these are based on the common assumptions derived from NEP discussed earlier. First, there is an assumption that the proper way to study the causal factors in human behavior lies in examining what the various aspects of human behavior were originally designed by naturally selection to do. The second assumption is that we can compare current behavior to the original "intentions of nature" to provide us with objective information about current functions and dysfunctions in humans.

Despite the common set of NEP assumptions as well as the "intentions of nature" idea and these common methodological assumptions, there are differing opinions among the authors I have been discussing on just how evolutionary psychiatry and something like Murphy's taxonomy can actually help psychiatry. Wakefield permits only breakdown explanations to count as mental disorders because he argues that a mental disorder must be a dysfunction relative to the environment it was designed for. Once we have categorized our evolutionary explanations we can then take explanations that fall under the mismatch and persistence categories and deem that as failing to count as a mental disorder. The explanations are legitimate in the sense that they help to explain the

behavior but this behavior should not be classified as a mental illness because nothing in the person is dysfunctional. The remaining explanations in the breakdown category are therefore constitutive of mental illness for Wakefield.

Murphy and on the other hand maintain that mismatch explanations should still be legitimate categories of mental illness as there is something that has gone wrong, but it is simply a matter of locating the failure in another area. The input that the brain is receiving is problematic and this indicates the presence of a mental disorder. Cosmides and Tooby agree with Murphy on this point. This is not surprising considering one of their central assumptions involves the understanding that modules provide behavior that was adaptive in an ancestral environment, but that behavior may no longer be adaptive now (Cosmides & Tooby, 2005, p. 16-17).

Interestingly, it is only Cosmides and Tooby that think persistence explanations might count as legitimate mental illnesses. They suggest that:

> An absence of dysfunction in many normal mechanisms may qualify as a treatable condition if it is capable of being treated and the persons involved wish it to be treated (Cosmides & Tooby, 1999, p. 459).

Cosmides and Tooby argue that determining various evolutionary functions is objective, but that our decisions about treatment should not be based on these functions and therefore that there can be no normative conclusions (e.g. what counts as a mental disorder) made based on evolutionary explanations:

> Although an evolutionary perspective cannot provide any objective basis for validating some values over others, it does provide an objective basis for analyzing function and dysfunction (Cosmides & Tooby, 1999, p. 457).

Wakefield thinks that only breakdown explanations can count as mental disorders. In persistence explanations nothing is "dysfunctional" so they cannot be mental disorders.

Murphy also argues that we should not consider persistence explanations to be mental disorders but he argues that it is because nothing has gone wrong in the mind *or* in the environment. For Murphy either something has to have broken down or the input from the environment has to be faulty for a mental disorder to be present. Murphy and Wakefield draw normative conclusions based on facts about the solutions to environmental problems in our ancestral past which limits which kind of evolutionary explanations can count as mental disorders. Cosmides and Tooby do not think that any normative conclusions about mental disorders can be drawn from their objective understanding of function and dysfunction.

I have now established a connection between NEP and the kind of evolutionary explanations that we saw fit into Murphy's taxonomy. This holds despite some minor differences between how we are to understand the mechanisms that evolved as solutions to environmental problems. I have also shown a shared commitment among these authors to the "intentions of nature" idea which derives from these problematic common assumptions from NEP. The "intentions of nature" idea also provides common methodological assumptions for understanding whether or not the solutions to adaptive problems are still functioning as they were designed to function by natural selection. These commonalities lead to an agreement that Murphy's three categories are appropriate demarcation points for taxonomizing different types of behavior causally, however there is a significant differing of opinions on what we can conclude from this.

Wakefield argues that it is clear that breakdown explanations are objectively the only legitimate category that can be considered a mental disorder. Murphy suggests that breakdown and mismatch explanations should count, while Cosmides and Tooby suggest

that nothing can be concluded about mental disorders based on evolutionary psychology alone. This is despite the fact that we can obtain objective definitions of function and dysfunction from evolutionary psychology for many psychological traits. I will revisit these differences in chapter 4 where I will suggest that the differences are related to differing concepts of "mental disorder" among the authors and divergent understanding of the role that values should be playing in defining mental disorders. For the rest of this chapter and chapter 3 I will focus on the commonalities between the authors and why they are problematic. In the next section I will show that Murphy's taxonomy is strongly biased toward evolutionary explanations using the NEP and "intentions of nature" assumptions as these assumptions are part of the structure of the taxonomy itself. I will also show that using these assumptions as a basis for evolutionary psychiatry actually works to prevent the two goals that Murphy has for creating his taxonomy in the first place.

2.7 Murphy's Taxonomy and Complete Causal Explanations

Despite Murphy's changed views on the importance of evolutionary theory in psychiatry, he continues to believe in the importance of understanding underlying causal mechanisms. He now thinks that more often than not evolution will play a secondary role in discovering these mechanisms (Murphy, 2006). One main reason for this is what Murphy sees as the lack of fit between form and function in evolutionary explanations of mental disorders. Murphy explains that:

> If form is to fit function, all the symptoms of a disorder (or all the manifestations of an aspect of our non-pathological psychology) must be explained. This is a very demanding standard for any theory to meet (Murphy, 2006, p. 762).

Murphy suggests here that evolutionary explanations are typically not complete explanations of mental disorders. They can explain some symptoms of a disorder but not all of them. For Murphy, the resources of evolutionary psychiatry do not seem capable of accounting for a complete causal story of the origin of many mental disorders. I suggest that at least part of the reason Murphy believes this is because of the assumptions he shares with proponents of NEP and the "intentions of nature" idea which is generated from these assumptions.

Murphy's taxonomy aims to separate evolutionary explanations into distinct causal types. Each of his categories represents one of those causal types and he claims that his taxonomy can categorize the evolutionary psychiatry literature. Therefore Murphy is also making a claim that each evolutionary explanation in the literature uses one of three basic causal explanations. Either a mental disorder is caused by an internal dysfunction ("breakdown"), an external change in environment ("mismatch") or a trait that remains functional in a relevantly similar environment to which it evolved and is simply judged to be negatively affecting the person in question ("persistence"). Therefore all mental disorders for which we can provide evolutionary explanations must be explained by an explanation which fits one (and only one) of these three explanatory patterns. The problem I have identified earlier in this chapter is that some evolutionary explanations either do not fit any of these patterns or equally problematic they appear to fit more than one pattern.

For example, one evolutionary explanation for OCD we looked at suggests that OCD is a "dysregulation of evolutionarily conserved behaviors and mental states critical to human survival" (Feygin, Swain, & Leckman, 2006, p. 855). I showed that despite

some evidence suggesting it the dysregulation did not warrant a "breakdown" label as there is a strong continuity between the types of obsessions and compulsions in the population at large and those with OCD. We could apply a "mismatch" label but this seems to minimize the importance of internal changes that may have occurred and there is nothing to suggest that environmental change was the causal factor, only that environmental change could be the causal factor. It seems ambiguous in this explanation which causal pattern is appropriate. This evolutionary explanation doesn't seem to be using any one of the causal types that Murphy suggests we should expect all evolutionary explanations to be using.

Instead this explanation recognizes that threat detection requires a *process* of regulation between an internal system and the environment. The regulatory process involves both internal and external components and so either an internal "breakdown" of some kind or an external environmental change could be the cause of the aberrant behavior. It might be an empirical matter to determine whether or not the dysregulation was caused by an internal breakdown or an external change in environment but in either case this evolutionary explanation would still be correct. The "dysregulation" is simply recognizing that the regulation of threat detection behavior has changed and Murphy might easily concede that empirical evidence is simply needed to clarify which category this explanation belongs to. However the structure of this explanation presents another possibility that is much more problematic for Murphy's taxonomy.

It is possible that the dysregulation involves both an internal change and an external environmental change. OCD may involve an internal change that requires a certain environment to cause the dysregulation that is problematic. If this is the case then

in order to explain the OCD behavior we would require referencing both of these causal factors that have together created a change in the functionality of the threat detection behavior. In other word we need the explanatory resources of the breakdown category and the mismatch category because both internal and external causes are necessary for this behavior to be displayed. Murphy's taxonomy requires that we must eventually recognize each evolutionary explanation as fitting under single causal pattern, but this may be a false dichotomy. This explanation therefore represents a distinctly different causal type that in fact uses two of Murphy's types together. Therefore we should not be surprised that this explanation does not fit under the taxonomy as the taxonomy is motivated by the assumption that evolutionary explanations will be one of Murphy's three types.

Murphy's inability to categorize this explanation stems to his reliance on NEP. NEP assumes that studying a single time period (the Pleistocene) can provide us with virtually everything we need to know about human evolutionary psychology. This ignores changes in a trait may have occurred before or after the Pleistocene and this information may be vital to a full explanation of the trait in question and may be required to explain all of the symptoms of a mental disorder as well. The Holocene epoch is where we are likely to see evidence of niche construction and the possibility of human behavior influencing their environment as well as the environment shaping human behavior.

Since we are investigating a dysregulation that involves a process occurring between the environment and internal factors it seems we must consider how the radical changes in environments and living conditions in the Holocene might have affected threat

detection behavior and also how the radical changes in human behavior shaped human's environments. How else can we discover if OCD is caused due to internal change, environmental change or a combination of both factors working together? Surely we need to examine the period of time in which environmental factors were possible altered by human behavior. It seems that any conclusions reached without considering the Holocene must be considered incomplete at best and completely inadequate at worst.

NEP is also biased against studying evolutionary history in time periods before the Pleistocene and yet the dysregulation example refers to animal studies justified on the basis of conserved mechanisms that date back far beyond the Pleistocene epoch. It seems relevant to study threat detection behavior in our ancestors in order to have some idea about the mechanisms that were modified to create the Pleistocene threat detection behaviors. The tendency for some animal species to experience OCD like symptoms suggests that a lot of explanatory details about OCD may be found by looking earlier in evolutionary history than the Pleistocene.

By considering periods before and after the Pleistocene we are likely to construct explanations that are more complete. Unencumbered by NEP assumptions we might discover that an internal change occurred long before the Pleistocene in several mammalian species and for humans it was only the environment of the Holocene that caused some to experience a dysregulation that we now call OCD. The Pleistocene is not even discussed here and therefore using Murphy's taxonomy we cannot consider this possibility even though it might be suggested by the dysregulation evolutionary explanation for OCD. Using NEP assumptions Murphy therefore risks providing incomplete causal explanations.

In another of the OCD examples Brüne described OCD as a by-product of the adaptive ability to anticipate needs or threats in the future (Brüne, 2006, p. 317). This has shades of all three categories but no way to determine which one is best suited to house this explanation. I have been arguing that Murphy's requirement that each evolutionary explanation needs to fit into a single category is problematic and so if we ignore Murphy's claim we can explore the possibility that OCD may be a result of the process of evolution. This is again a different causal type and yet it might involve all three of Murphy's casual types and in this context they might be better understood as "causal patterns" as I am not assuming that they are the individual distinct types that Murphy does. Murphy forbids combining these causal patterns due to NEP which fails to respect the complexity of the evolutionary process and prevents us from recognizing salient factors that can help to give complete evolutionary explanations.

The NEP understanding of a by-product is limited by adherence to the "intentions of nature" idea. A by-product can only be a product of another adaptive trait which arose to solve a specific adaptive problem. The adaptive trait operating as it was designed to do is the "intentions of nature" and any by-product is a random happenstance brought about because of the adaptive trait. In reality the evolutionary process is not that simple. From Downes' evolution of the hand example we saw that focusing entirely on solutions to environmental problems may prevent us from recognizing that other parts of the body or brain are being modified while evolution is occurring. This moves us beyond a strict adaptive trait and by-product dichotomy. Downes demonstrates the difficulty in determining which part of the hand is responding to which environmental pressures and he argues that this difficulty will extend to the brain as well.

In the OCD example from Brüne we see this theme recapitulated as it is difficult to determine where the adaptive ability to anticipate needs and threats ends and where OCD behavior begins. The adaptive ability to anticipate needs and threats is a complicated trait and intertwined with many other traits so that selection operating on threat detection will be affected by the requirements of the other traits. Once we involve multiple time periods the "intentions of nature" idea becomes even more confusing because traits are adapting to different environments in different time periods and many of the traits originated in mammalian species or even earlier in evolutionary history. We might say that some of the "intentions of nature" are still expressed or that there is a partial expression of the "intentions of nature" but we cannot determine whether or not the "intentions of nature" are being expressed in a binary fashion.

The "intentions of nature" idea helps Murphy to categorize evolutionary explanations according to his taxonomy. We decide whether or not behavior is consistent with the "intentions of nature" and then if those intentions are being expressed in the proper environment. If we cannot answer whether or not the "intentions of nature" have been violated with a distinct yes or no and if we cannot answer whether or not those intentions are operating in the correct environment then Murphy's taxonomy will not be able to taxonomize these explanations. At best there is often no way to tell if the "intentions of nature" are being fulfilled and at worst we might conclude that the entire "intentions of nature" idea is irredeemably confused for any explanations. In either case Murphy's taxonomy fails to categorize evolutionary explanations that offer causal information beyond what the taxonomy is equipped to categorize.

One final way in which the taxonomy restricts evolutionary explanations is by preventing a combination of evolutionary explanations themselves. Each of the three OCD explanations that I have looked at might not be competing but instead might be complimentary and might be able to combine to produce a more complete evolutionary explanation. Different explanations might highlight different causal factors which each might explain different symptoms or might combine together to explain certain symptoms. Viewing OCD as a by-product may be compatible with viewing it as a dysregulation. The dysregulation may be a by-product resulting from an internal change to threat detection mechanisms that was prompted by an environment that no longer exists. The group selection OCD example may be involved if the whole system is beneficial to groups that have a person with the dysregulation. This is highly speculative and it may or may not explain every symptom of OCD but the important point is that it is ruled out *a priori* by Murphy's taxonomy and the NEP and "as nature intended" assumptions. We can't even consider it.

What these examples show is that Murphy's taxonomy is strongly biased towards explanations that share the assumptions of NEP and the "as nature intended" idea. NEP presents a certain view of evolutionary psychology that focuses almost exclusively on a specific time period and on the environmental problems and solutions that were found in that time period. This is coupled by the "as nature intended" assumption that we can determine objective information on the functionality and dysfunctionality of current traits by asking whether or not those traits are still acting as solutions to the ancient environmental problems they arose to solve (in the Pleistocene). If an evolutionary explanation describes that a trait is still acting as a solution to the problem it arose to

solve ("as nature intended") and it is acting this way in a relevantly similarity environment then we categorize that evolutionary explanation as a persistence explanation and for Murphy we can also objectively determine that it is not a mental disorder. If a trait is acting as a solution to the problem it arose to solve in a distinctly different environment then it is a mismatch explanation and is a distinctly different kind of mental disorder than a trait that is shown to fail to act as a solution to the problem it originally arose to solve, the latter being a breakdown explanation.

The structure of the taxonomy requires all evolutionary explanations to share the NEP and "as nature intended" assumptions and use the criteria I have just described to categorize their explanations. Wakefield provides explanations that are explicitly breakdown explanations, while Stevens and Price assume that all evolutionary explanations (or at least most of the evolutionary interesting ones) will be mismatch explanations. They share the NEP and "intentions of nature" assumptions that are the foundation for Murphy's taxonomy and therefore Murphy's taxonomy can easily categorize their evolutionary explanations. On the other hand explanations such as the OCD examples discussed earlier cannot be taxonomized because they do not share these assumptions as I have shown in this chapter. This is problematic as if true these explanations are in fact more likely to provide the kind of complete causal explanations that Murphy is motivated to find using evolutionary resources. The explanations that fit under the taxonomy are likely to be focused on a single time period with a concentration on a single causal factor and tend to ignore the complexity of the evolutionary process.

Therefore Murphy's taxonomy fails to achieve the goals Murphy created it for. Some evolutionary explanations cannot be taxonomized while those that can be

categorized are at risk of providing incomplete causal information, while those excluded often can provide more of the causal information that Murphy is motivated to discover through evolution. Finally those explanations that can be taxonomized are very likely to share many of the assumptions of NEP as well as the "intentions of nature" idea which derives from NEP. Because of the extent of criticism that NEP has received this puts the taxonomy in an even more questionable state and even forces us to question the validity of the distinctions and the causal factors that the taxonomy does recognize.

Since the taxonomy fails to help bring about Murphy's two goals we find that evolution does not appear to provide the benefits Murphy initially hoped for. Since the taxonomy fails to categorize any of the three evolutionary explanations for OCD we risk a failure of identifying OCD as a mental disorder at all, despite the fact that there are a number of evolutionary explanations for it. Alternatively even if we arbitrarily force the three OCD explanations into Murphy's categorize we risk losing out on important causal information about the evolutionary history of OCD. If an explanation has qualities of a breakdown explanation and a mismatch explanation then it behooves us to be aware of both the internal and external changes that have occurred to bring about the behavior we describe as "OCD". It also behooves us to be aware of multiple time periods of the evolutionary history the OCD behavior and the evolutionary process itself and how it may provide us with important causal information. Ignorance of this kind of causal information is likely to lead a loss of important information about the mental disorders themselves.

In conclusion, evolutionary explanations that fit under the taxonomy are likely to be incomplete explanations based on inadequate biology while some explanations that do

not fit into the taxonomy such as the OCD examples actually stand a much better chance of providing complete evolutionary explanations that allow for identification of all mental disorders and the evolutionary causal factors that may have been important in their origin. Murphy's claim that form often does not match function may derive from Murphy's narrow conception of what an evolutionary explanation for a mental disorder should be. His changing attitude towards evolution and psychiatry may have more to do with his idea of evolutionary explanations being constrained by and adherence to NEP and the "as nature intended" idea. These ideas and assumptions lead to evolutionary explanations that are at best incomplete and at worst biologically and scientifically inadequate. In order to solve these problems in evolutionary psychiatry we require a biologically adequate evolutionary psychology that has the potential to provide evolutionary causal explanations for mental disorders that are more complete. Arguing for such a conception of evolutionary psychology will be the focus of Chapter 3 but first I will summarize this chapter.

2.8 Conclusion

In this chapter I have presented Dominic Murphy's taxonomy for evolutionary explanations in psychiatry. His account is aimed at categorizing the types of evolutionary explanations in the literature through a causally motivated categorization of explanation types. Despite a change in attitude towards the potential for a union between evolution and psychiatry, his taxonomy of evolutionary explanations for mental disorders does represent some authors in the evolutionary psychiatry literature. The authors whose explanations fit under this taxonomy typically share several assumptions with Murphy

that lead to his idea of the "intentions of nature". However, some evolutionary explanations in the literature do not seem to fit under his taxonomy.

I have shown that Murphy's taxonomy fails to be able to categorize three evolutionary explanations for Obsessive Compulsive Disorder (OCD). Murphy's motivations for his taxonomy commit him to mischaracterize his categories of evolutionary explanation as mutually exclusive when in fact it appears that many explanations can be categorized under more than one explanation type. I have traced Murphy's problems to the influence of NEP which provide the common problematic assumptions that lead to Murphy's problematic taxonomy. NEP has been heavily criticized and these problems are inherited by Murphy and some others interested in evolutionary psychiatry such as Jerome Wakefield.

The OCD examples do not fit into Murphy's taxonomy because they do not appear to share the same assumptions that lead to the "intentions of nature" idea. Murphy's taxonomy in fact seems biased towards explanations that share these assumptions. Therefore Murphy's taxonomy constrains the kind of evolutionary explanations that we can use and because the taxonomy rests on problematic assumptions of NEP I argue that we should not reject these evolutionary explanations but instead reject Murphy's taxonomy and the assumptions that are interconnected with it. This also casts doubt on Murphy's assumption that the "intentions of nature" idea can help provide the concept of what a mental disorder is especially considering that Murphy and Wakefield do not seem to share the conception of a mental disorder despite agreeing that the "intentions of nature" can help to provide it.

Murphy's taxonomy fails to include all evolutionary explanations and since those explanations that are included have a tendency to be incomplete explanations based on inadequate biology this version of evolutionary psychiatry fails to provide much help in distinguishing different types of mental disorders and also fails to help in determining what should count as a mental disorder in the first place. When the taxonomy does make these distinctions it does so using problematic assumptions which does not provide robust causal information about the mental disorders in question.

In the next chapter I will suggest that we need to provide a better foundation in evolutionary psychology in order to achieve the goals Murphy holds for evolutionary psychiatry. In order to do this we must re-examine the assumptions that lead to the "intentions of nature" idea that seem to be at the heart of the problems now facing evolutionary psychiatry as exemplified through Murphy's taxonomy. I will proceed by examining a long standing debate in the history of evolutionary theory where these assumptions and ideas similar to the "intentions of nature" have been argued about. I examine a promising alternative understanding of evolutionary psychology that follows from evolutionary developmental biology (evo-devo) an important movement in biology over the last 40 years. Through looking at this movement and its history I will argue that it can provide a more biologically adequate version of evolutionary psychology than NEP. With a better foundation we can then develop a version of evolutionary psychiatry that is capable of providing the valuable insights to the discipline of psychiatry that Murphy and others have previously suggested.

CHAPTER 3: DEVELOPMENTAL EVOLUTIONARY PSYCHOLOGY

3.1 Introduction

In this chapter I will examine the history of evolutionary theory and examine a debate that can help illuminate why the NEP assumptions discussed in the previous chapter are so problematic and also how we can avoid the NEP problems by looking to the movement to incorporate developmental biology into evolutionary theory. Development has been a missing component in evolutionary biology for most of the 20th century and only recently has it begun to be fully integrated. I will describe that process which has resulted in the field of evolutionary developmental biology and give a brief outline of what this field currently looks like. I will then look at the psychological branch of this field Developmental evolutionary psychology (DEP) and explore the needs and benefits of integrating development as an alternative to NEP. I will argue that NEP fails to take development seriously and therefore should be replaced by DEP as the foundation for evolutionary psychology research. I first turn to an explanation and history of Evolutionary Developmental Biology (Evo-devo) a field that has emerged in response to the general lack of developmental explanations in biology for much of the 20th century.

3.2 A Brief History of Evolutionary Developmental Biology

Before exploring the benefits of an evo-devo approach to evolutionary psychology and ultimately psychiatry, we need to first examine what this research program is and look at its historical emergence, as evo-devo is a research program with many different roots (Laubichler, 2007, p. 348).

One of the earliest roots of evo-devo can be traced back to the 1820s when Karl Ernst von Baer argued that we should define traits as being "the same" when their embryonic origins are the same. Those who followed von Baer in the study of embryology were typically known as "structuralists" and advocated the need for examining autonomous structural principles of traits prior to elaborating on the functional characteristics of a trait. For example, when explaining why some organisms such as mammals have four limbs the structuralists cite descent from other tetrapods and the fact that tetrapods are all built on a common body plan (or *Bauplan*). The contrasting functionalist position argued that four legs simply work better for mammals, and that is why mammals have four legs (Amundson, 2009).

During the early debates between the functionalists and structuralists a formalized distinction was made by Richard Owen between Analogy and Homology the main principles of similarity for each tradition (Amundson, 2009). Analogy for the functionalists recognized a similarity in body parts between species that is due to a similar adaptive need served by the body parts in question. For example the similarities between the wings of birds and the wings of butterflies are analogies and do not share the same embryonic origins but do share a common function in flight. Homology for the structuralists instead recognizes a similarity because the body parts in question are actually the very same body part under modification, regardless of current function

(Amundson, 2009). The four limbs of mammals in each mammalian species are homologous to each other even though they have taken on new functions in some mammalian species. The similarities between mammals do not necessarily rest on similarity in function, but instead rests upon similarity in embryonic origin and descent patterns.

Toward the end of the 19th century a new research program called evolutionary morphology emerged that was a primitive version of evo-devo (Amundson, 2009). Evolutionary morphology held that phylogenetic reconstruction and embryology were united in the goal of understanding organic form. It recognized that the questions about the origin of the major body plans, how organic form is created during ontogeny and how form is modified through evolution could not be answered separately but instead had to be answered interdependently (Amundson, 2009). Amongst evolutionary morphologists there was a common acceptance of "the core doctrine": to achieve a modification in the adult form, evolution must modify the embryological processes responsible for that form, so that an understanding of evolution requires an understanding of development (Amundson, 2009). This was connected with the project of tracing the tree of life where embryologists aimed to illuminate what common ancestors of different species would have been like (Amundson, 2009).

By the start of the twentieth century evolutionary morphology had died out as the tools needed to understand development were not available (Amundson, 2009). Around 1930, the defunct evolutionary morphology was replaced by a new theory of evolution known as "the modern synthesis" (Amundson, 2009). The rediscovery of Mendel's work in 1900 helped to establish genetics as a science and geneticists who were mathematically

inclined explored the question of how genes operate in a population. Evolution was redefined in terms of gene frequency change in populations over time and was then integrated with research from paleontology, animal breeding and other fields of biology to produce the modern synthesis (Amundson, 2009). This synthesis is understood to have remained intact and today is argued to still form the foundation of evolutionary thought in the early twenty first century (Guttman, 2005, p. 53).

Due to the synthesis interest in homologies, archetypes, and body plans were replaced by the synthesis mechanisms of evolution: mutation, migration, genetic drift, and especially natural selection (quickly accepted to be the dominant cause of evolution). The synthesis simply ignored the structuralist understanding of evolutionary causation involving the modifications of ontogeny (Amundson, 2009). Experimental research programs like genetics and developmental mechanics therefore arose separately in the early part of the twentieth century. Mechanical and physico-chemical causes were sought in terms of explaining development, while evolutionary questions were sidelined for decades (Laubichler, 2007, p. 345).

In the 1970's, proponents of a developmental understanding of evolution reengaged in the debate over structure and function with proponents of the synthesis. Synthesis proponents took a functional stance arguing that adaptation is the essential explanation for traits, arguing that traits should be viewed as consequences or by-products of natural selection (Amundson, 2009). The causal mechanisms of embryological development on the other hand suggested that developmental constraints prevent many potential trait variants from ever existing. Therefore developmental explanations could go a long way in explaining why certain trait variants might exist or

not exist (Amundson, 2009). Natural selection could only contribute to explaining why certain traits continued to exist or not, but it could not provide any help in understanding how or why those traits would come about in the first place and yet the modern synthesis seemed to privilege adaptation by natural selection for most evolutionary explanations.

The mid 1980's brought a consensus that the prevailing adaptationist form of evolutionary theory required a reformation. Twenty five years later a new synthesis has begun and despite differing research agendas among the newly synthesized disciplines Jason Robert suggests that evo-devo now holds a place "at the forefront of contemporary biological inquiry" (Robert, 2008a, p. 291). The scope of this new synthesis includes techniques and research from: molecular biology, anatomy, physiology, functional morphology, cell biology, embryology, developmental genetics, paleontology, comparative genomics, and population genetics among even more fields. This collaborative research is aimed at discovering more about the origins, development and evolution of the diversity of organic form (Robert, 2008a, p. 291).

In the next section I will give an outline of the results that this synthesis has produced and some of the difficulties that still exist from the integration of multiple disciplines into the form of evo-devo. I will then discuss how NEP connects with the research traditions I have discussed in this section and how NEP uses different underlying assumptions than evo-devo, assumptions which I will suggest are outdated and inadequate. This will set up a discussion that compares and contrasts NEP with an evo-devo based evolutionary psychology.

3.3 Evolutionary Developmental Biology

Brain Hall has defines evo-devo[2] as the study of "how development (proximate causation) impinges on evolution (ultimate causation) and how development has itself evolved" (Hall as cited in Griffiths, 2001, p. 2). Evo-devo also compares developmental processes across species in an effort to discover ancestral relationships. These goals are realized through study of how the embryo develops, how new traits are developed through changes in developmental processes, the extent to which developmental plasticity occurs as well as the developmental basis of homology. Homology retains the structuralist definition as the study of traits which share ancestry but not always function. Developmental plasticity refers to the ability of an organism to respond to the environment as an influence in its development as the range of possible outcomes is wider than we might expect if focusing on genetics as the sole determinate of the construction of traits (Griffiths, 2001, p. 1).

We find a classic exemplar of evo-devo in the discovery of *hox* genes which contain a DNA sequence called a "homeobox". These genes were first identified in the

[2] The synthesis I have been describing as "evo-devo" can be described by other names including "devo-evo" and some find the choice of names to be important (Robert, 2008a, p. 291). Jason Robert treats evo-devo and devo-evo as conceptually, epistemically and methodologically equivalent in some situations despite the fact that there are potentially interesting differences between the projects. Robert utilizes evo-devo over devo-evo simply for aesthetic purposes in these instances (Robert, 2008a, p. 292). I follow Robert in this for two reasons. First, the scope of my project does not allow for an in depth account of the differences between evo-devo and devo-evo. Secondly, my main concern is showing the differences between an approach like NEP and various other approaches that take development seriously. The differences between evo-devo and devo-evo are not the same kind of differences that we will see between NEP and either of these or various other developmental approaches. Jason Robert notes that philosophers studying evo-devo have shown dwindling concern over the problems of reconciliation of various approaches to evo-devo. He argues that the diversity of approaches has been accepted and shown to be generally complementary in increasing our knowledge of the diversity of life (Robert, 2008a, p. 297). This is in stark contrast to Robert's claim that NEP fails to take development seriously in any manner (2008b).

fruit fly *Drosophila* and have now been confirmed in many plants and animals. *Hox* genes have a role in regulating cell patterns and activating genes essential to the formation of basic body plans. The discovery of homeobox genes suggests that biodiversity may be less related to differences in protein coding genes and instead might involve changes to gene regulation. Even when developmental genes themselves are highly conserved, the changes in the regulatory mechanisms for these genes can lead to dramatic morphological change (Carroll, 2005).

For example mutations induced in the homeobox regions of *Drosophila* genes have led to extreme morphological change with antennae forming where legs typically grow (mutation in the *Antennapedia* gene) while another mutation produced flies with two sets of wings instead of a set of wings and halters (balancers) (mutation in the *Ultrabithorax* gene). Identifying homeobox genes in the embryos of *Drosophila* has helped our understanding of the developmental biology involved in pattern formation while the evolutionary significance of homeobox genes and mutations is undergoing investigation (Robert, 2008a, p. 294). Ronshaugen *et al* have examined the role of *Ultrabithorax* (*Ubx*) mutation in the transition from many-limbed arthropods to six-legged (hexapod) insects that occurred approximately 400 million years ago. Ronshaugen *et al* propose that the *Ultrabithorax* mutation expressed in the fruit fly thoracic region suppressed 100% of limb development but in brine shrimp only 15% was suppressed. Therefore it may be that changes in *Ultrabithorax* expression caused phenotypic modification (e.g. reduction in limb number) that may have led to the hexapod body plan arising (Robert, 2008a, p. 294).

Examining this "second-order" regulation involving the interactions and timing of activity in gene networks represents a different course of study than simply looking at what individual genes within that network are doing on their own. This provides a significantly different element in evolutionary theory through an examination of the roles of gene duplication and gene regulation in creating morphological diversity. Biologist Sean Carroll posits that changes in some of the regulatory systems of genes is likely more significant for evolution than changes in gene number or protein function of the gene products (Carroll, 2000). This represents a dramatic departure from viewing genes and their protein products as the nexus of evolution. Instead we understand the developmental processes of the organism as the nexus of evolution. In the previous example some of the major evolutionary changes between fruit flies and brine shrimp seem to have occurred due to developmental gene regulation and not protein coding gene alterations.

Due to developments like this evo-devo has forced a reconceptualization of the genotype-phenotype relationship. Thomas Hunt Morgan's understanding of genetic determinism saw genes regulating development. Evo-devo suggests that genes themselves are actually regulated during development – by an expansive network of interactions with other genes and other influences from within the nucleus, the cytoplasm and the extra-cellular environment (Amundson, 2009). So even when we speak of *hox* genes controlling body plans we do not mean individual genes are determining phenotypic traits. Genes are seen as responders rather than controllers, responding to the developmental processes around them. Hox genes are a part of this process directing

body plan segmentation through many other genes, and this process and not individual genes themselves are what are responsible for adult traits (Amundson, 2009).

In evo-devo the core doctrine from evolutionary morphology is vindicated; to understand evolution we must understand *how ontogeny can be changed*. Through the acceptance of this doctrine Ron Amundson see's two major advances coming out of evo-devo. One is the origin of evolutionary novelties. This happens when a new trait arises from an ancestor with no obvious trait that could have been easily modified into the new one. Evo-devo helps to explain these novelties first by finding the ancestral ontogeny in place before the novelty arose through comparison of the ontogenies of related organisms and secondly by showing how it is that modifications could have been made from the ancestral ontogeny to create the novelty. Müller and Wagner 1991 give the example of the vertebrate limb (as cited in Amundson, 2009). The vertebrate limb was shown to involve reduplication of a cluster of Hox genes. The extra cluster of hox genes that arose from the reduplication process was then available to be co-opted to develop the limb. Explanations of this type are quite different from adaptationist explanations as they involve ancestral ontogenies and not ancestral traits while typically paying no mind to questions of populations or fitness.

The second major advance for Amundson involves phylogenetic reconstruction aimed at discerning the commonalities of life. Evo-devo researchers have discovered that all animals that are more highly structured than jellyfish (vertebrates, molluscs, insects, worms, sea squirts etc.) are descended from the *Urbilaterian* ancestor. The ontogeny of *Urbilateria* has been shown to involve a number of regulatory genes such as *Pax-6*, responsible for triggering the development of a proto-eye in the ancestor that was light

sensitive as well as showing known developmental genetic triggers for other body parts such as heart and neural system tissue. It appears that all animals on earth share these basic developmental resources. This opens up the potential for an investigation to determine how basic, shared, embryological processes can be modified to create the variety of life existing today (Amundson, 2009).

In order to achieve these advances suggested by Amundson evo-devo requires examining the conceptions of function that are possible in biology and evolution. Amundson and George V. Lauder contrast three different biological understandings of function. The first they call the selected effect (SE) analysis of function. For this type the function of a trait is described by the history of natural selection that the ancestors of the organism possessing the trait have undergone. The function of a trait is equivalent to the evolutionary purpose it was designed for; the selective history determines the function. This "etiological concept" concept of function is defined as the effect for which that trait was favored by natural selection (Amundson & Lauder, 1994, p. 443).

The second type is non-historical and defines the function of a trait to be its current causal properties determined by those which contribute to organism's current needs, purposes, and goals (Boorse 1976) or on an alternative account those traits that hold evolutionary significance to the organism's current survival and reproduction (Amundson & Lauder, 1994, p. 444). This approach does not bear a particular moniker, but we can call it current survival value (CSV) function.

A third approach associated with Robert Cummins describes function without reference to either evolutionary or contemporary purposes. Amundson & Lauder describe this approach as the causal role (CR) account of function (Amundson & Lauder,

1994, p. 444). They argue that CR function is vital to many research programs in biology. It has become particularly valuable in developmental biology and ultimately to evo-devo. I will focus on comparing the SE and CR conceptions of function as they are the two conceptions of function that can work within an evolutionary framework. CSV explicitly rejects focus on evolution while SE function requires it. CR function on the other hand is the most flexible and can work in both evolutionary and non-evolutionary contexts. SE function and CR function are also indicative of the two research traditions I have described in the previous sections a point I will return to once I have explained the basics of each conception of function.

Amundson and Lauder explain the SE concept through Elliot Sober's definition where the variable Task F is the SE function of some trait X:

> X is an adaptation for task F in population P if and only if X became prevalent in P because there was selection for X, where the selective advantage of X was due to the fact that X helped perform task F. (Sober as cited in Amundson & Lauder, 1994, p. 447).

If a trait is going to be an adaptation it must be historically defined and this is equivalent to saying that the trait has a function (defined by its selected effect). A trait can be an adaptation if and only if it has a function and therefore adaptation and function become interchangeable on the SE account of function (Amundson & Lauder, 1994, p. 447).

CR function as proposed by Robert Cummins suggests that we do not concern ourselves with assumptions about any objective goal of the system in question. Evolutionary history is not necessary for determining a conception of function and Cummins suggests using functional analysis which he described as a distinctive scientific explanatory strategy. Functional analysis involves explaining the capacity of the system by referencing the component parts of that system (Amundson & Lauder, 1994, p. 448).

Capacities do not need to be goals or purposes of a system. Capacities are first deemed worthy of functional analysis and then we attempt to figure out how it is that the interactions of the various capacities of the component parts lead to the capacities of the system as a whole. We assign functions to each component (or trait) based on the system capacity chosen and the functional explanation that we are giving. Cummins explains that given some functional system S:

> X functions as an F in S (or: the function of X in S is to F) relative to an analytical account A of s's capacity to G just in case X is capable of F-ing in S and A appropriately and adequately accounts for s's capacity to G by, in part, appealing to the capacity of X to F in s. (Cummins as cited in Amundson & Lauder, 1994, p. 448).

CR functions are not constrained by what we have learned about a functional system in the past or by what purpose we think the system exists for. In SE functions we rely heavily on what we have learned about past environments and what purpose we think the trait in question was evolved for. SE functions are highly constrained by these supposedly objective criteria and one concern with CR functions is that there are no constraints on what might count as a functional system.

However Cummins does propose some general constraints. He suggests that we do not want trivial analyses, only valuable ones which add significantly to knowledge of the analyzed trait. One criterion for scientific significance occurs when "the analyzing capacities cited are *simpler* and *different in type* from the analyzed capacities" (Cummins as cited in Amundson & Lauder, 1994, p. 448). Also "an analysis is also of high value when it reveals a high degree of complexity of organization in the system" (Cummins as cited in Amundson & Lauder, 1994, p. 448). Analyzing very simple systems proves to be trivial.

Cummins emphasis on causal parts of a system interacting without specification of an overall goal is shared by functional anatomists such as Walter Bock and Gerd von Wahlert (Amundson & Lauder, 1994, p. 449). Bock & von Wahlert are even further committed to CR function than Cummins by asserting the importance of unused functions which might be possible given the system in question but are not functions the organism actually uses. This alludes to the importance of exaptation (Amundson & Lauder, 1994, p. 450). Exaptation refers to traits which had either a previous purpose or no purpose and then are co-opted for a new purpose by an organism.

The choice of analysis for functional anatomists typically revolves around character complexes with significant biological roles:

> An anatomist might choose to analyze the crushing capacity of the jaw of a particular species. Cummins's s is the jaw, and G the capacity to crush things. In the analysis the anatomist might cite the capacity of a particular muscle (component X) to contract, thereby bringing two bones (other components of s) closer together. If the citation of that capacity of X fits together with other citations of component capacities into an "appropriate and adequate" account of the capacity of the jaw to crush things, then it is proper on Cummins's analysis to say that the function (or a function) of that muscle is to bring those two bones closer together (Amundson & Lauder, 1994, p. 450).

We can evaluate this analysis in terms of Cummins criteria for scientific significance. If the crushing capacity of the jaw is nothing more than two bones act together by brute force using muscle X to force the bones together then the organizational aspect of the system is exceedingly simple. Likewise the force of the muscle is neither simpler nor different in kind from the crushing capacity seen in the jaw and thus we have a functional analysis with little value (Amundson & Lauder, 1994, p. 451).

However, a jaw composed of multiple elements with muscle X exerting a weaker force than the overall crushing capacity of the system because the crushing action results

from rolling and grinding with muscle X moving an attached bones into a position supporting a new direction of motion coordinated with other muscles to occur at a specific time in the cycle provides a candidate for analysis. The muscles function is simpler by far than the capacity of the system and it is also different in kind in terms of one dimensional movement compared with three dimensional in the system and the organization of the smaller capacities proves to be complex and therefore this represents a worthwhile scientifically significant analysis (Amundson & Lauder, 1994, p. 451). Neither the current biological role of the jaw nor its evolutionary history plays any part in the analysis. Certainly we may be motivated by such concerns when *choosing* which system to focus our attention on but these concerns take a back seat during a CR functional analysis. New biological roles that are discovered also do not affect prior analysis although they could provide motivation for examining new capacities in the organisms (Amundson & Lauder, 1994, p. 450).

A CR analysis therefore allows us to avoid some of the pitfalls that occur when focusing only on SE function. For example, there may be many different SE functions that have occurred in the history of a trait (Amundson & Lauder, 1994, p. 460). Present day roles may or may not be able to tell us about the role of the functional traits in the past. There is also difficulty in determining when selection acted on a trait in the past and whether or not other traits were involved as selection on one trait through pleiotropic effects of genes under selection will cause changes in other traits. A lizard's ability to run being placed under selection pressure will affect heart mass, muscle enzyme concentrations, body size, and the number of eggs laid, even if selection was directed

only at endurance in lizard running. This makes it difficult at times to even separate an individual trait under selection (Amundson & Lauder, 1994, p. 461).

Alternatively, functional anatomists can determine the way in which forms have changed over evolutionary time without regard to specific selection pressures acting on an individual trait (Amundson & Lauder, 1994, p. 463). Examining organism structure and function have followed two different approaches, the equilibrium and the transformational approaches (Lauder, Lewontin as cited in Amundson & Lauder, 1994, p. 464). The equilibrium approach is in line with the functionalists looking for adaptations based on current environmental factors. Of course this can tell us relatively little about the past as it assumes some kind of equilibrium between the current selection pressures and the adaptations in question. The transformational approach on the other hand looks at intrinsic factors:

> Functional morphologists also view organismal design as a complex interacting system of structures and functions (Liem and Wake 1985; Wake and Roth 1989). Indeed, the notion of 'functional integration,' which describes the interconnectedness of structures and their CR functions, is central to discussions of organismal design and its evolution. The extent to which individual components of morphology can be altered independently of other elements without changing the (CR) functioning of the whole is one aspect of this current research (Lauder 1991) (Amundson & Lauder, 1994, p. 464-65).

One of the most important alternatives to studying selection pressures and adaptations is looking for homologies. As noted earlier this is a key feature of the evo-devo program and Amundson and Lauder draw a strong connection between CR function and homology. Homologies can have different functions despite having the same morphological ancestry (p. 454). While homologies are now defined in terms of these descent relationships before Darwin they were discerned by examining similar structures and development (p. 455). Regarding differing definitions about homologies Amundson

and Lauder make the following point: "Whatever the favored definition of homology, one feature of the concept is crucial: *the relation of homology does not derive from the common function of homologous organs*"(Amundson & Lauder, 1994, p. 454 emphasis in original). Recognizing homology by structural or developmental similarities is about recognizing a common role within a system that is defined historically but not selectively. Homologies may retain all of their former structural similarity or they may not even perform the same role within a system that they once did. What is important is that they once did share a common role in a system and this is not defined in terms of any selective processes.

Drawing on the discussion from earlier in the chapter we can see connections between the structuralists and homology and causal role function. The structuralists study autonomous structural principles of traits before elaborating on the functional characteristics of those traits. Functionalists were studying what we now call adaptations, and they defined functions in terms of what the organism's overall goals were. The theory of evolution simply showed the functionalists to be talking about adaptations and SE functions while the structuralists were talking about homologies and CR functions. These connections show a long history of divisions in terms of the approaches to studying evolution.

In the next section I will show that NEP utilizes an SE conception of function. I will then contrast NEP with a new understanding of evolutionary psychology based on evo-devo which utilizes CR function at its core but still maintains an ability to incorporate SE functions where appropriate. I will compare some of the assumptions of NEP with some of the assumptions of this psychological branch of evo-devo and argue

that the later approach is a better foundation for evolutionary psychiatry because unlike NEP it takes both evolution and development seriously.

3.4 An Evo-devo alternative to NEP

3.4.1 The Principles of NEP

NEP posits that the brain consists of modules that were designed by natural selection to perform certain functions in response to environmental problems. From the discussion in the previous section it should now be clear that NEP utilizes a strictly SE concept of function. The entire focus is in the functionalist tradition, aiming for explanations about what the trait does. This may be expected in the realm of behavior, as it is not immediately obvious how structural explanations might help. In this section I will explain how structuralist explanations utilizing CR functions and homologies can be important to evolutionary psychology. Evo-devo represents an amalgamation of the CR and SE concepts of function and attempts to place both in their proper contexts. Developmental biology alone or a program like NEP alone does not have this conceptual power. I will compare and contrast NEP and a psychological version of evo-devo, arguing that only the latter takes both evolution and development seriously.

I have shown that the importance of incorporating developmental biology into evolutionary theory has finally been recognized and a "new synthesis" of sorts has been occurring since at least the 1980's, albeit with numerous issues still to be worked out. Paul Griffiths argues that the insights gained from evo-devo that relate to morphological

evolution may be equally productive for evolutionary psychology through the sub discipline of evo-devo for the mind that Griffiths calls "Developmental Evolutionary Psychology" (Griffiths, 2001, p. 4).

I follow Griffiths in using the name DEP, as with evo-devo there are other potential names for an evo-devo approach to psychology. As I showed earlier, for some purposes Robert feels that evo-devo and devo-evo can be considered conceptually, methodologically and epistemically equivalent. For my purposes I take developmental evolutionary psychology to be roughly equivalent to other approaches such as evolutionary developmental psychology and evolutionary developmental psychobiology. [3] The approaches that I draw from all share the commitment that NEP has failed to take development seriously and evolutionary psychology would be better served by utilizing evo-devo as a foundation as opposed to the work and assumptions derived from NEP (Griffiths, 2001; Bjorklund & Pellegrini, 2002; Finlay, 2007; Lickliter, 2008; Moore, 2008; Robert, 2008b). This is the main point to be taken as Jason Robert suggests that the many developmental approaches that exist such as evo-devo, devo-evo and DST are progressively becoming more complimentary over time (Robert, 2008a). This differs from NEP which continues to remain in stark conflict with these developmental approaches as I will show in this section.

I will focus on comparing NEP and DEP by looking at five principles of each approach. This will allow a more detailed understanding of both NEP and DEP as well as provide an understanding of how I see each principle of DEP being incompatible with

[3] It is also the case that evolutionary developmental biology does not necessarily correspond to evolutionary developmental psychology and developmental evolutionary biology does not necessarily corresponds to developmental evolutionary psychology.

one or more of the principles of NEP. Through this comparison I will aim to show why so many authors think that a DEP approach is a much better foundation for evolutionary psychology than an approach predicated on NEP. I derive my principles of DEP from the various authors who have suggested an evo-devo approach as a replacement for NEP (Griffiths, 2001; Bjorklund & Pellegrini, 2002; Finlay, 2007; Lickliter, 2008; Moore, 2008; Robert, 2008b).

I derive the NEP principles from the common NEP assumptions that I suggested were held by Cosmides and Tooby, Wakefield, Murphy and Stevens and Price in chapter two. In short our brain is composed of functional modules which are adaptations evolved to solve adaptive problems we encountered in a hunter gatherer past. This explanation involves several interconnected claims that are easier to deal with if they are broken down into constituent parts so they can be analyzed individually. To that end I have taken apart the assumptions that make up the NEP story and individuated them into what I take to be the principles of NEP which are shared by the authors in the evolutionary psychiatry literature that I discussed in chapter two. They are as follows:

1. Human minds are universal
The "monomorphic mind thesis" describes a 'psychic unity of humankind' (Cosmides & Tooby, 1992, p. 79 as cited in Griffiths, 2006a, p. 17). The differences in behavior between individuals or groups is not due to genetic differences but instead is almost always a result of environmental factors "triggering" various aspects of the same developmental program.

2. Human minds are modular
Often described as the 'massive modularity thesis' or the 'Swiss army knife model', NEP holds that human minds are composed of a variety of separate "modules" that have each been designed by natural selection to solve a specific adaptive problem. We should expect virtually no general-purpose cognitive mechanisms (Griffiths, 2006a, p. 15).

3. Human modules in the mind were created through adaptations

The modules which compose the human brain were created as adaptations through the process of natural selection (from assumption 3 and 4 as cited in Cosmides & Tooby, 2005, p. 16-17).

4. Human modules in the mind were created in response to environmental problems
All human modules were designed by natural selection in response to specific adaptive problems that were experienced by human ancestors in the past (Griffiths, 2006a, p. 15).

5. The environmental problems humans are adapted to existed when we were hunter gatherers
Our modules have been designed to solve adaptive problems that we experienced as hunter gatherers in a specific time period in the Pleistocene described as our EEA (from assumption 3 as cited in Cosmides & Tooby, 2005, p. 16).

I will examine each of the five principles of DEP and show which principles of NEP are incongruent or even incompatible with DEP. I start by describing the fundamentally different conceptions of modularity that exist in NEP and DEP.

3.4.2 Developmental Modularity

A fundamental difference between NEP and DEP is the differing conceptions of modularity apparent in each. In NEP the brain is a computer designed by natural selection and modules are programs almost all of which are designed by natural selection in response to selection pressures that we experienced as hunter gatherers. Surviving modules were better at solving adaptive problems than the alternative programs that arose in our evolutionary past (Cosmides & Tooby, 2005, p. 16-17).

Modules in DEP on the other hand follow an evo-devo understanding in which modules consist of "regions of strong interaction" in what is termed an interaction matrix. For example, as embryos develop they are often modular in that certain spatial regions develop relatively independently of other spatial regions within the embryo. Genetic activity in one segment of a developing arthropod for example, will have little to no affect on the future state of other segments relative to the strong effect this genetic

activity has on the segment in which it is occurring. Developmental modules often show hierarchical organization, with smaller scale physical modules existing within larger physical modules. Despite growing independently of each other, as developmental modules differentiate into specific parts of the embryo, interactions between developmental modules become important. Tissues that are composed of cells of one type often must come into contact with other cells of another type of tissue in order to induce further differentiation between cell types. The effect of the actual interaction between cell types is not what is important; rather what matters is the cascading effect that results from the interaction within the affected module itself (Griffiths, 2001, p. 4).

Paul Griffiths compares these physical developmental modules with what he terms the "virtual mental modules" that are postulated in NEP. The first major difference is that mental modules do not necessarily correspond with any particular region in the brain. That is why Griffiths calls them "virtual modules" as they exist only on a functional cognitive level. Of course, Griffiths point out that these virtual mental modules function *through* the brain, and so must ultimately derive from the physical developmental modules which create the physical brain. Griffiths suggests that since NEP suggests that the virtual mental modules are fine tuned through natural selection this implies that they must also be developmentally dissociable, meaning that changes in the development of one virtual module should not affect other virtual modules' development (Griffiths, 2001).

If they are not functionally dissociable and cannot function independently, then changes in one virtual module will affect performance of the specific tasks in the other virtual modules. So, if the virtual modules can't be instantiated independently, then they

cannot be fine tuned by natural selection independently. Natural selection makes changes to the physical developmental modules which must give rise to the virtual modules. If the physical developmental modules that somehow create the virtual modules are not dissociable, meaning that they interact, then changes in one physical developmental module will lead to changes in other physical developmental modules which are responsible for building the virtual modules (Griffiths, 2001).

When this happens it becomes difficult to accept the claim of NEP that each mental module is molded independently by natural selection for a specific task. NEP must assume that mental modules are dissociable but the development of the brain shows this assumption to be highly suspect as many physical developmental modules are interactive thus it is difficult to see how these changes would not also affect the virtual mental modules that arise from the physical developmental modules (Griffiths, 2001).

Therefore, the principle of modularity proposed by NEP is incompatible with the principle of modularity suggested by DEP. DEP therefore is a better option because it is based on *what is* as opposed to what NEP argues *must be*. The concept of modularity in DEP is based upon physical evidence of the way development *actually occurs* in the body, including the brain. In DEP our explanations are constrained by the actual way we know things to be, namely the interactive nature of developmental processes. In NEP explanations are constrained by the way that natural selection *must have* operated in the past. However, as the hand example from Downes in chapter two suggested, we should not expect evolution by natural selection to operate this way at all. Traits do not typically have one specific function, but instead are often responsible for a variety of functions and we require explanations regarding a variety of selection pressures to understand why it is

different organs and traits evolved the way they did. This is further supported by our understanding of physical developmental modules which further show how integrative the development of traits can be.

While it is possible that some psychological traits have characteristics of virtual mental modules, it is not compatible with modern evolutionary biology to suggest that they must have these characteristics and in fact there is a great deal of evidence to suggest that most will not have these characteristics. The principle of modularity in NEP grounds itself in a conception of evolution by natural selection that is incompatible with the version of evolution by natural selection that is offered by modern evolutionary biology, represented here by DEP.

3.4.3 Homology

A second fundamental difference between NEP and DEP consists in different conceptions of the role of homologous and analogous traits. NEP holds that all of the modules in the human brain were designed by natural selection in response to selection pressures that we experienced as hunter gatherers (Cosmides & Tooby, 2005, p. 16). The NEP focus is on human evolution in the Pleistocene period as the time in which all our mental modules which are responsible for our human behavior originated. These are traits that arose adaptively and they are defined functionally and are compared analogously sometimes only between other humans.

This contrasts with DEP where homologous traits are studied though comparison to a wide variety of species instead of focusing solely on analogous adaptations which arose during a single time period in our own species. By focusing on a single time period we miss important information about the evolutionary history of a trait. Just as the

recognition that our hands developed from mammalian forefeet is an important source of potential insight great insight can also be gleaned by an examination of the evolutionary history of psychological traits.

Like most areas of biology DEP uses both analogous and homologous character types in a comparative methodology. Considering that the comparative method has been described as "perhaps the single most powerful epistemological technique available to biology" (Harvey & Pagel, 1991 as cited in Griffiths, 2001) it is problematic that NEP cannot fully availed itself of these techniques due to a singular focus on one type of character trait in one time period. The comparative methodology proceeds by typically homologizing behavior before postulating adaptive explanations. This determines what is known as character polarity which is discovering "the precursor state from which the current state evolved" (Griffiths, 2001, p. 11). It has been suggested that attempting adaptive explanations before discovering character polarity would be comparable to explaining the American Revolution while not knowing whether it was the United States that seceded from the United Kingdom or vice-versa (O'Hara, as cited in Griffiths, 2001).

Character polarity provides us with a guide that tells us that human hands evolved out of mammalian forefeet and not the other way around. This is an obvious example but in many other situations it proves to be much more difficult to determine character polarity particularly with psychological traits. Homology allows us to assign common structures and functions based on relationships of ancestry. We do not need to know what environmental problem the trait evolved to solve and our efforts to determine this will be more successful if we first understand more about the ancestral precursor trait. In order to do this we need to assign character polarity. Despite this Griffiths argues that

many evolutionary psychologists tend to move in the other direction postulating adaptive function with little effort and seem to hold that adaptive function should be easier to discover than assigning homologous function. For Griffiths this is logically incoherent as discovering homologous relationships before we make inferences about the adaptive value of the traits is not only more likely to be correct than positing adaptive explanations but these homologous relationships can then also dramatically increases the chance that we will make correct inferences about adaptive functions at the appropriate time (Griffiths, 2001, p. 14).

Not only is it important to discover character polarity through homology before attempting adaptive explanations, but it is also epistemically easier to uncover homologous relationships than it is to posit adaptive functions for traits. It is difficult to find evidence about adaptive function from the past but homologous relationships can be studied more directly. Using "adaptive thinking" as a heuristic for understanding complex mental traits becomes problematic when we require a knowledge of those traits in order to understand how they are affecting the adaptive problems that the environment is likely to pose (Griffiths, 2001, p. 14). Instead it is likely that homologies can provide help in determining adaptive functions.

For example Jaak Panksepp and Jules B. Panksepp argue that there is substantial empirical evidence that the brains of humans have been created by evolutionary changes that have occurred to ancient mammalian brains long before the Pleistocene epoch. They suggest that:

> Focusing on these ancestral emotional functions of the brain, to the extent that they are still represented in existing species, provides a unique empirical platform for thinking about the adaptive foundations of the human brain/mind (Panksepp & Panksepp, 2000, p. 110).

They suggest that any "essential character" of human minds was created in ancient emotional and motivational systems which are not unique to humans, but in fact shared with a number of other animal species. They argue that we should look to the ancient sub cortical systems of the brain to find the anatomical, neurochemical and functional homologies that are shared with many other mammalian species. The failure to recognize these homologies can lead us astray when attempting to determine the emergence of uniquely human brain functions. The connection between ancient emotional systems and the operation of uniquely human capacities is of the utmost importance to understanding human behavior (Panksepp & Panksepp, 2000, p. 110).

NEP postulates that modules were created through adaptations (principle 3), in response to environmental problems (4) during the time in which humans existed as Hunter gatherers (5). NEP advocates studying these adaptations in light of postulated selection pressures and environmental problems that must have existed at the time. The principle of homology instead suggests that the functional traits that humans exhibit should first be understood as deriving from the common structures and behaviors of ancestral species. We can discover homology without reference to adaptive function, but postulating adaptive function without reference to homology is more difficult to do and more likely to be prone to error. Once homologous relationships have been established we can examine the change in functions that have occurred since the time of common ancestry between humans and other species. Only then can we and should we postulate environmental problems that were endemic to the Pleistocene and even then only in light of constraints imposed by the realities of homologous ancestral structures.

3.4.4 The Environment as Co-constructor

A third major difference between NEP and DEP relates to their respective understanding of the interaction between the environment and the brain of the organism. This is best explicated in Griffiths and Kim Sterelny's "grain problem" (Griffiths, 2001). They argue that determining whether or not specific features of the environment represent a single problem or multiple problems is dependent on the developmental structure of the brain and not just on the environment itself. If solutions to environmental problems cannot be developmentally dissociated, they become interrelated because they share a common solution mechanism. NEP suggests that we can examine an environment and count various separate evolutionary problems that an organism has to solve through natural selection. Griffiths argues that the way developmental modules work help determine what a separate evolutionary problem for an organism is (Griffiths, 2001).

Griffiths explains this contrast with an example from Cosmides and Tooby and their proposed emotional module of fear (Griffiths, 2001). This module is held to be an evolved response to the danger posed by predators to our hominid ancestors. This is an environmental problem. But we can parse this problem in coarse-grained terms as a general response to danger or in more fine grained terms as the problem of responding to snakes for example. Empirical evidence would suggest that our actual fear response resulted from coarse-grained selection since we respond to all kinds of different dangers in the same way. Moreover the emotional appraisement of fear, the input aspect, responds to snake-like gestalts but also to many other stimuli which require relevant experience in order to provoke a fear response. So, while there are a few "pre

programmed" universalized fear stimuli most input that causes fear results from experience (Griffiths, 2001).

Downes analogy of the hand and brain showed that we should expect the brain to be responding to multiple conflicting problems which pull in different directions. One reason for this is that solutions to environmental problems are constrained by the way in which development occurs. Humans have a single fear response and even if an environmental problem arose in which humans would benefit greatly from a distinct aerial predator fear response and a distinct terrestrial predator fear response (something some mammals have) this response will not evolve if the pre existing fear responses are not developmentally dissociable. Natural selection will not be able to fine tune an aerial or predator response because the human fear response is too developmentally integrated for selection to invent completely separate responses even if it is adaptively beneficial. As we saw in the previous section developmental explanations can often explain why certain traits can or cannot exist while natural selection can only tell us why existing traits are favored or not.

This problem is compounded by the fact that for any environment that exists at a certain time we cannot identify the full range of all niches that are available for colonization. This is because many of the biotic properties of the niche derive from the activities of the life forms that inhabit the niches themselves. So, listing possible niches in an area must include not only those that correspond to biotic features of the environment, but also those that correspond to those niches that could be created with certain types of organisms living in concert with that environment (Griffiths, 2001). An example is certain Eucalypt species that have the ability to colonize patches of dry forest

by facilitating bushfires. This niche exists in large part due to the trees that evolved to fill the niche. The organism adapting to the environment and the creation of that environment are not dissociable. Thus, the environmental problems that the organisms are adapting to are partially determined by the organism itself. Because niches and populations co evolve in this manner it becomes difficult to determine the number of separate adaptive problems that exist in an environment. Each niche in some sense contains indefinitely many overlapping problems and the problems an organism adapts to depend on the organism itself.

In order to help solve these problems Griffiths suggests that we look to work in evo-devo that explores a co-constructing relationship between developmental modules and evolutionary problems posed by the environment. Griffiths argues that this approach could be applied to mental modules as well but we would have to accept that we cannot determine the modular structure of the mind through an independent listing of adaptive problems in any given environment. Empirical investigation of the mind needs to occur with work on development as well as ecological/evolutionary modeling of mental evolution (Griffiths, 2001).

The NEP claim that all human modules were designed by natural selection in response to specific adaptive problems seems incompatible with the DEP understanding of the role of the environment in evolution. If we cannot specify objective adaptive problems to solve and if organisms themselves help determine the problems they are solving then there is no one to one correspondence of problem and solution as claimed by NEP (Principle 4). This also suggests that localizing environmental problems to the Pleistocene is seriously mistaken (principle 5). Niche construction as discussed in

chapter 2 is most likely to have occurred during the Holocene, the period in which Humans established longer lasting niches which they could modify. Therefore, some evolutionary change is likely to have occurred during this period. Viewing the environment as coevolving with the organism we also understand that changes to the environment induced by humans are part of the evolutionary story and not independent of it. Therefore understanding ourselves as housing a "stone aged brain" is problematic. While much of our makeup may remain similar to our ancestors from the Pleistocene, some of it likely has changed and in a co-constructing relationship with the environment.

3.4.5 Plasticity

NEP suggests that environmental inputs "trigger" our various mental modules to stimulate preprogrammed behavioral patterns. DEP instead recognizes the importance of developmental plasticity: the ability of an organism to respond to the environment as an influence in its development. The environment is a co-constructor of traits and so the range of possible traits is wider than we might expect when explaining behavioral traits with NEP modules.

David Buller and Valerie Hardcastle use an analogy of scarring to help explain how plasticity functions in the construction of the brain. Genes can tell us about the microlevel process of the formation of scar tissue but the pattern of scarring requires information about the history of environmental trauma that an individual has sustained. Similarly, genes synthesize the proteins that initially distribute neurons and neural connections but do not control the processing role of neurons. A number of connected factors interact with the genes but then brain activity is guided by environmental factors. The "sculpting" of the final form of the brain has little to do with gene expression and

cannot be explained by it as much of the final form depends on the endogenous innervations of brain cells and interactions between brain and environment (Buller & Hardcastle, 2000, p. 316).

Although Buller and Hardcastle reject the NEP understanding of modularity they do think that modularity could arise from the brain's developmental plasticity by allowing the brain's structures to be determined by environmental tasks that are needed:

> It is a mistake to seek adaptations among the products of brain development – that is, among the relatively special-purpose brain structures that emerge during the course of brain development. Those products are highly plastic responses to environmental inputs. Our cognitive adaptation is, instead, the process that continually generates and modifies special-purpose brain structures. That is, with the possible exception of our sensory transducers, it is not the contingently stable brain structures in an adult's brain that are adaptations; rather, the brain's very developmental plasticity is the adaptation (Deacon, 1997), and the relatively stable structures are by-products of that adaptation's functioning in a particular environment (Buller & Hardcastle, 2000, p. 321).

They compare this process to the immune system which must repel a structurally diverse assortment of antigens. Cells assemble specialized antibodies in response to each antigen they encounter but the specialized structures of these antibodies have little to do with genes. Instead explaining the structural qualities requires reference to the immune system and environment interactions (Buller & Hardcastle, 2000, p. 317).

The process of the immune system responding to a host of antigens is an adaptation in and of itself. The antibodies produced are not adaptations but are instead products of a general purpose adaptation which sometimes creates novel antibodies in response to novel antigens. Similarly mental modules can be created in response to conditions of the environment that may not resemble conditions of the past in any way (Buller & Hardcastle, 2000). Stable structures therefore exist across time due to similar environmental problems and conditions causing developmental systems to produce

similar solutions from a general adaptation which was in turn able to solve many of the diverse problems we faced in our hominid past (Buller & Hardcastle, 2000, p. 316). Changes in structures and behaviors can sometimes occur due to a general adaptation of plasticity coming up with a unique solution based on a process that was selected for in the evolutionary past.

Part of the developmental flexibility that Buller and Hardcastle argue for lies in their distinction between an NEP conception of domain specificity and their conception of domain dominance:

> Our brain systems are not *domain specific*; rather, they are *domain dominant*. One sort of processing in a brain region may be more prominent than others, but other processing is still occurring. Our "modules" are not so specialized that they deal only with restricted domains. Instead, they deal mostly with particular domains, and do so only contingently; the dedication of a brain system to a particular task domain is subject to change as the inputs to that brain system change (Buller & Hardcastle, 2000, p. 313).

Even if humans have some degree of functional modularity we cannot assume that is a result of past adaptation or even strict compartmentalization. The tasks that various modules accomplish can change based on environmental parameters despite the possibility that certain brain areas might have a bias towards certain functions.

Because of this flexibility Paul Griffiths extends the importance of plasticity to an examination of plasticity as a potential source of evolutionary novelty (Griffiths, 2011). Griffiths argues that a biologically plausible evolutionary psychology recognizes that humans develop in a social context, and phenotypes can result from a combination of genetic input as a product of evolution as well as input derived from socialization. The phenotypic plasticity that results can benefit from evolutionary explanation. We find

phenotypes outside the ranges in which they operated in the evolutionary past and so plastic organisms are capable of developing new functional phenotypes.

Plasticity directly challenges all five of the NEP principles as our brains are not universal (1) but instead depend greatly on environmental factors during development, NEP modules focus on domain specificity (2) when domain dominance is more accurate, our modules are sometimes created by a domain general adaptive process and not by specific adaptation (3) responding to environmental problems (4) from the distant past (5).

3.4.6 Variation

NEP is associated with Cosmides and Tooby's "psychic unity of humankind" concept that suggests we all share a "human nature". Variation is either to narrow to be significant or explained by different experiences "activating" different aspects of universal human psychological modules. These assumptions help lead to the "intentions of nature" idea suggesting there is a way to objectively determine question of functionality based on whether or not people's traits are functioning as they were designed to function by natural selection in past environments. Behavioral traits are species typical if they fall within the functional range of operating as they were intended to by natural selection. Variation outside of this range should arguably not be called "variation" and instead should be referred to simply as instances of dysfunctional behavior.

DEP instead recognizes Paul Griffiths' point that traits in general do not necessarily tend towards species typicality. In terms of variation amongst humans we see all human beings have lost the ability to synthesize vitamin C while some people can

metabolize lactose as adults while others are lactose intolerant and cannot metabolize lactose. When ecological factors are stable across time and space natural selection tends to favor low variation in traits if a clear winning evolutionary strategy exists. However frequency dependent selection can favor variation in traits. This occurs when it is only advantageous for a certain number of organisms in a species to have a certain trait. When ecological factors are unstable selection may also tend to favor variation in traits allowing organisms to hedge bets or use phenotypic plasticity as a strategy to generate more variation (Griffiths, 2011). When selection pressures and weak or non-existent we should expect higher amounts of variation in general as mutations are less likely to be weeded out. Different traits at different times experience differing amounts of variation in general and it is once again not clear why behavioral traits should be any different.

An NEP understanding sometimes views variation as a mask which hides the "psychic unity" of humans as NEP understands most variation resulting from different environmental inputs "triggering" different aspects of a universal program. DEP holds that what NEP describes as a "program" is itself composed of both elements of the environment and elements of genetic heredity. For DEP "human nature" is not about human universals but is instead about interpreting and making sense of human diversity (Griffiths, 2011). A DEP research program is in fact likely to avoid using terms like "triggering", "program" or "human nature". DEP instead studies how a variety of developmental resources stably reproduces phenotypic traits across generations and the pattern of diversity that is produced by this process.

Griffiths therefore argues that DEP needs a new principle of classifying diversity that embodies two insights from evo-devo. The first insight is that vastly different traits

can be classified together by common origins and the second is that evolved developmental mechanisms are capable of generating truly novel traits (Griffiths, 2011). Griffiths argues that the method of classification that embodies these insights is already available and it is one that I have already discussed: homology. Classification by homology has been neglected as a way of studying cultural variation amongst humans and yet homology has precisely the features needed for a better conception of what human diversity entails (Griffiths, 2011).

Homology operates as a principle of identity through difference. For example, human expressions of facial emotion are homologous to certain facial expressions in chimpanzees despite substantial differences in form and function. Chimps and humans operate in different environments and yet homology can show correspondence between these two systems. Therefore we can use homologies as a model for investigating systems comparatively even within our own species living in different environments. Comparing diverse phenotypes in different cultural contexts is therefore best done via homology and not via overt similarity that is argued to correlate to NEP's universal programs. Human diversity can instead be studied via common origins (Griffiths, 2011).

Similar behavior in different cultures can mean dramatically different things while quite different behavior can often be shown to have a common purpose. Comparing traits by overt similarity is akin to cataloguing traits by analogous functions but this does not tell us much about the evolutionary history or status of those traits. Homology can tell us something about the evolutionary history because it is "the same" trait in a historical sense and this puts us into a better position to determine adaptive issues and questions of distribution and universality.

Evo-devo has also lent the insight that identifying homology at one level of biological organization is independent of identifying it at a lower level of organization. Trait A can be homologous with trait B and this will have no effect of breaking down trait A into sub components and identifying their homologous relationships. Griffiths therefore suggests that instead of trying to ask "is this the same trait" across different cultures, a more meaningful question would be "is this homologous, and at what level of analysis?" (Griffiths, 2011). This moves us away from superficial similarity colored by our own cultural experiences and towards a deeper similarity based on correspondence, ancestry and common origins and developmental systems.

Instead of the search for universality apparent in NEP thinking (principle 1), DEP emphasizes studying the pattern of similarity and difference in human behavior. Ironically for NEP the truly specialized systems that exist in our human brains are likely to be ancient sub-cortical systems that share homologous structure and function with many other animal species. The uniquely human traits on the other hand are more likely to be those that are shaped by individual experiences that affect our highly plastic neocortex (Panksepp & Panksepp, 2000, p. 110). This makes the concept of "human nature" fraught with confusion, especially when a universal "human nature" is proposed. We are better off examining variation in light of homologies, common developmental pathways and the role the environment plays in shaping our many plastic traits.

I have now discussed five principles of DEP and contrasted them with five principles of NEP. I will now link some of these themes together in a discussion of the different understandings of function in DEP and NEP.

3.4.7 Evolutionary Psychology and Function

Functionalists typically use SE functional descriptions to compares traits analogously looking for similarity in function in terms of organisms solving the same type of environmental problem. Structuralists typically use CR function to compare traits homologously in terms of ancestral or developmental correspondence despite possible functional and structural modification. We have seen in this chapter that the modern synthesis focuses exclusively on selected effect (SE) function in the functionalist tradition while developmental biologists and anatomists instead have used causal role (CR) function in the structuralist tradition.

We have also seen that NEP focuses exclusively on SE function following in the tradition of the modern synthesis. It may be tempting to credit DEP with a focus on CR function in the structuralist tradition as the historical disagreement between structuralists and functionalists has traditionally been about whether analogy or homology was the proper way to compare traits. While it is true that DEP focuses on CR function one of the great insights of evo-devo is that both concepts of function are important and required for use in the comparative method. DEP therefore uses both conceptions of function but holds that SE function is best used after foundational questions of development and homology are answered through the use of CR function. Therefore the issue for DEP is not about which conception of function is correct or best but instead which conception of function constrains the other.

NEP suggests that their adaptationist approach to discovering mental modules should be the focal point of investigating human evolutionary psychology. We can figure out the environmental problems of the past (namely the Pleistocene) and with this

knowledge explain what *must have been* the modular solutions that evolution invented. Unfortunately the knowledge about *what is* for physical developmental modules shows that these developmental modules in fact constrain the possible modular solutions that evolution could have possible invented.

In fact Griffiths reminds us that how developmental modules work help to determine what is a separate evolutionary problem for an organism (Griffiths, 2001). We can't use SE function here because it requires a predefined definition of a separate environmental problem and of course that is exactly what we are trying to determine with this kind of research. CR function allows us to work with homologies so that we can see the internal evolutionary change that has taken place without concerning ourselves with the selection pressures that caused the change. Once we have that foundational knowledge then we can ask meaningful questions about selection pressures while understanding the environment as a co-constructor providing resources that are essential to the development of an organism. The environment has the ability to affect different plastic systems and create a wide array of different and at times completely novel traits. The end result of this evolutionary process should be seen as generating patterns of similarity and diversity instead of assuming a "human nature" residing in common modules or "programs" and we can best study this variation by using CR function and homologies. Homologies allow us to compare by correspondence and not overt similarity which provides a better description of the patterns of diversity and similarity that exist in human behavior.

Therefore DEP holds that the principles of developmental modules, homologies, environmental problems and developmental plasticity all constrain the NEP principles of

modules created through adaptations in response to environmental problems in the Pleistocene. Claims about universality are also constrained by understanding the patterns of similarity and diversity in terms of the DEP principles which are grounded more on *what is* than what *must be*. Therefore CR functions must be determined prior to SE functions and can be considered foundational in this sense. This is not to suggest that SE functional ascriptions are not vitally important. It may even be possible that in many cases proper SE functional ascriptions that are constrained properly by the DEP principles may yield more vital information than the CR functional ascriptions they are based on. However DEP also suggests that CR functional ascriptions that study developmental modules, homologies, environmental co-construction, plasticity and variational patterns can provide great insights in their own rights. DEP does not suggest that one conception of function is better than another, only that there is a relationship between the functional ascriptions that should be adhered to.

In this chapter so far I have outlined the historical biological debates about function and the connected them to the origin of evo-devo. I have then suggested how an evo-devo based evolutionary psychology (DEP) would be superior to the currently dominant NEP. One of the main points I have used to argue this is that DEP takes development seriously while NEP does not. However, some proponents of NEP suggest that they do take development seriously. In order to address this issue and also elucidate the extent of incompatibility between NEP and DEP I will rely on Imre Lakatos and his work on scientific change and progress. This will help show the extent to which a potential merger of NEP and DEP are possible.

3.5 The Future of Evolutionary Psychology

3.5.1 NEP and Developmental Biology

I have set up DEP and NEP as competing research programs because NEP fails to incorporate development although some evolutionary psychologists claim they do incorporate development:

> In fact, cultural variation constitutes natural developmental manipulations; hence, cross-cultural tests are one of the key kinds of developmental experiments open to evolutionary psychologists. This is why evolutionary psychologists have placed so much emphasis on cross-cultural research (Tooby, Cosmides & Barrett, 2003, p. 861).

This claim is partially what Griffiths is responding to when discussing universal emotions in human cultures. Griffiths points out that NEP methodology explores aspects of similarity by using analogous comparisons that focus on functional similarity (Griffiths, 2011). This is indicative of how NEP studies development. Griffiths of course suggests that it would be more practical and meaningful to discover homologous elements of the emotional expressions between cultures and to elucidate the level of organization at which homology does or does not exist.

NEP researchers claim to be testing development by studying whether or not humans solve problems in an analogous way but this fails to determine whether or not the humans are using "the same" resources to solve these problems in a historical sense. Different cultures could very well solve the same environmental problems using the same methods or exhibit the same behavior but this course of study does not tell us whether or not they are doing so because of the same trait. In fact, Griffiths suggests that the question "is this the same behavior?" is not a very good question at all. A better question to ask is "are these people using homologous resources?" (Griffiths, 2011). Taking

development seriously requires using a principle of correspondence before attempting to discern adaptive similarities.

There is often ample opportunity for NEP to integrate previous homologous findings into their adaptive research but NEP often fails to do this:

> The exploration of emotional systems is a major challenge that is recognized by many evolutionary theorists (Nesse1990; Buss1999; Cosmides/Tooby 2000), but, rarely is existing brain evidence incorporated into such discussions. For instance, the adaptive 'fear' module postulated by Cosmides/Tooby (2000) does not adequately recognize one 'fear system' that has already been characterized in the brains of other mammals (Panksepp 1982, 1990a; Graeff 1994; Rosen/Schulkin 1998). The fear module envisioned by Cosmides and Tooby appears to be a master module that coordinates the activity of the many smaller modules dedicated to cognition and Evolution and Cognition and autonomic regulation. In fact, the type of fear 'module' that has been revealed by animal brain research appears to have come into existence long before any sophisticated cognitive capacities existed (Panksepp 1990a, 1998a). From the way Cosmides and Tooby appear to envision matters, the modularization of fear came after the existence of rather sophisticated cognitive capacities. Their fear module's main purpose is to simultaneously recruit and coordinate cognitive activities during fearful situations, so that the likelihood of an adaptive behavioral response is maximized (Panksepp & Panksepp, 2000, p. 110-111).

This type of NEP research appears to contradict evolutionary developmental research. This is not surprising considering the differences we have seen between the principles of NEP and DEP. In order to clarify the relationship between NEP and DEP it is worthwhile using theoretical tools from philosophy of science to better understand the ways in which these research programs are contradictory and also investigate any potential that exists for the survival of some parts of NEP.

Timothy Ketelaar provides an account of the relationship between NEP and its competitors that is useful here. Ketelaar uses Imre Lakatos and his theory on scientific change and progress to suggest that NEP shares essentially all of what Lakatos describes as the "core" of other competing evolutionary psychology research programs. I agree the

Lakatos is useful for this project but disagree with Ketelaar's conclusions, however the way in which Ketelaar is wrong is important because it helps elucidate the way in which NEP thinks it takes development seriously. Once this is apparent we can clearly see why NEP cannot be understood to take development seriously and what ramifications this has for utilizing NEP and DEP research together.

3.5.2 Methodology of Scientific Research Programs

Imre Lakatos utilizes a theory of scientific change called the methodology of scientific research programs (MSRP). He argues that the appropriate unit of appraisal is not an individual proposition or even an individual theory but instead a sequence of related historical theories (Lavor, 1998, p. 50). We should thus describe science as a collection of these "research programs". For example, Newtonian science does not consist solely of four conjectures; laws of mechanics and gravitation. Instead, the laws are only what Lakatos describes as the "hard core" of the Newtonian program (Lakatos, 1974/1998, p. 23). In addition to this hard core we have a large "protective belt" of auxiliary hypotheses that helps protect the core from refutation. Finally we have the "heuristic", techniques that can help us to take recalcitrant problems and turn them into positive evidence for the theory. When faced with anomalies we examine all different kinds of conjectures in our research program but the essential aim of a scientific research program is the prediction of novel facts (Lakatos, 1974/1998, p. 24).

The hallmark of a good scientific research program is that it can predict novel facts that no one thought would be true or that no one could have even imagined before. All research programs continue to operate in a sea of anomalies but only successful scientific programs are able to predict novel facts. Scientists have "thick skins" and are

willing to make explanations for anomalies that should be regarded as recalcitrant instances and not as refutations (Lakatos, 1974/1998, p. 23).

Scientific revolutions are accounted for by differentiating between progressive and degenerative research programs (Lakatos, 1974/1998, p. 25). How a research program changes can determine its status as progressive or degenerative. Progressive programs change according to their own inner logic as opposed to the degenerating ones which change according to external criticism. Newton developed his program for a planetary system piece by piece according to the heuristics of the program, with the core never being questioned and the auxiliary belt being adjusted accordingly. He begins with a fixed point-like sun and one planet and derives the inverse square law from this and although this was forbidden by Newton's third law of dynamics a simple change to the model in which sun and planet revolved around a common centre of gravity solved this problem. Following this more planets were added without interplanetary forces, acting as if the sun and planets were mass-balls and not mass points. After these "puzzles" were solved spinning balls and their wobbles, interplanetary forces and finally perturbations were added sequentially.

For Lakatos this is a perfect example of a research program, a sequence of theories that are stages in developing a central idea. Instead of focusing on facts, Newton focused on continually working within the program to solve problems confident that he would find a model that could account for and also explain the evidence. The heuristics of the program were eventually able to do just that. So a progressive research program should develop its central idea with refinements made by the heuristics. Anomalies are tolerated and expected and we hope to sort them out and eventually turn them into

positive supporting evidence. However, if the heuristics cannot solve the problems that are emerging within the program then ad hoc efforts will begin that attempt to protect the hard core with techniques external to the program. Successive changes of the protective belt need to be "in the spirit of the heuristics" to make "heuristic progress" (Lavor, 1998, p. 54).

Heuristic progress is necessary, but not sufficient for a progressive research program. While ignoring anomalies, it must also make empirical progress. This requires new but also unexpected predictions to be confirmed, and it is particularly good if these newly discovered facts contradict rival research programs. Scientists have a tendency to join the progressive programs that are predicting novel facts, although sometimes degenerative research programs take a long time to become progressive, and scientists making an effort to stick with a program should not be seen as irrational. However, for many research programs there eventually comes a time when the program becomes degenerative and scientists will abandon it. For Lakatos progress and change in science can be described as a sequence of progressive research programs replacing degenerative ones (Lakatos, 1974/1998, p. 26).

3.5.3 Evolutionary Psychology and the MSRP

Ketelaar utilizes Lakatos' MSRP in an effort to explain the relationship between NEP and other competing evolutionary psychology research programs. Ketelaar argues that the general evolutionary principles from the modern synthesis are shared by those who prescribe an NEP outlook and those who offer alternative approaches. Ketelaar argues that the divisions in differing evolutionary approaches to psychology occur in the

protective belts of the various research programs and not the hard core of the research programs (Ketelaar, 2003, p. 36).

I suggest instead that the conflicting aspects of NEP do in fact lie in the hard core of the theory. We might describe the hard core as consisting of the six assumptions of NEP from chapter 2 or perhaps as the five principles I described in this chapter. These represent something akin to Newton's four conjectures; the laws of mechanics and gravitation. They represent the essential aspects of the NEP theory. These are assumptions that are not tested and are all interconnected with each other such that falsifying one proves problematic for others. These appear to be the principles that NEP refuses to give up and yet these are the principles that DEP as an alternative approach is directly inconsistent with.

Now there are certainly some consistent assumptions that are shared by NEP and DEP. They both hold that life evolved and that human cognitive abilities evolved and that we can study these abilities. They both likely do share tenets of the modern synthesis but in DEP the need to reinterpret and qualify those principles is much more apparent. Evo-devo descended from the modern synthesis but has required modification. One of the most important changes has been the incorporation of the core doctrine of evolutionary morphology. The necessity of understanding how ontogeny can be changed was obviously not part of the modern synthesis but it is now part of modern evolutionary biology. The result is that evolutionary biology now accepts the need for developmental explanations in evolutionary studies. This has also been reflected in evolutionary psychology with the recognition of the need for a new evolutionary psychology to be

based on evo-devo instead of NEP (Griffiths, 2001; Bjorklund & Pellegrini, 2002; Finlay, 2007; Lickliter, 2008; Moore, 2008; Robert, 2008b).

NEP and DEP may both be compatible with the modern synthesis and the modern synthesis may be the foundation for modern evolutionary biology, however without the insights of developmental biology a theory is unlikely to be consistent with the theory and practice of modern evolutionary biology. DEP's principles of modularity, homology environmental interactions, plasticity and variation must constrain NEP's principles of adaptive modules arising from specific environmental problems in the Pleistocene. CR function is the fundamental unit of function which constrains the ascriptions of SE function. Lakatos' MSRP may be the correct characterization for NEP and alternative research programs but NEP's "hard core" is not consistent with the "hard core" of approaches like DEP. NEP has managed to remain successful because it continues to make predictions and operate according to its own internal logic. However, with the rise of DEP the many cracks that are already evident in NEP will continue to grow. The multitude of criticism will also continue especially as DEP emerges as a discipline. As DEP grows it is likely to outstrip NEP in terms of predictive ability.

That being said there remains a possibility that a new progressive research program DEP might still make use of some research in NEP. NEP is such a dominant approach that it has infiltrated virtually every sphere of evolutionary psychology, being called "narrow" despite the fact that it is dominant in the field (Ketelaar, 2003, p. 31). Much research in evolutionary psychology has some relation to NEP, but may not be fully committed to all of its tenets. Murphy's taxonomy seems to be related to NEP in

various ways despite his call for development as a focal point in evolutionary psychological research (Murphy, 2003).

It might be suggested that all research of NEP is infected and must be disregarded but of course there are indeed many common assumptions regarding evolutionary theory that are shared between NEP and DEP. As DEP emerges there may still be a place for some of the work of NEP if it is willing to give up some of its core principles that are so problematic. Some of the belt level theoretical insights and some of the heuristic level experimentation of NEP may be salvageable if interpreted with proper core assumptions of DEP. Those using NEP methodologies and those influenced by the NEP paradigm may still have much to contribute, depending on NEP's ability and willingness to adjust to the realities of modern evolutionary biology exemplified by DEP. The extent to which NEP research can be utilized in DEP will be at least partially an empirical matter and will likely have to be decided case by case.

3.6 Conclusion

I began this chapter by discussing the history of Evo-devo, as evo-devo as a discipline has arisen in a large part as a reaction against the exclusion of development from evolutionary explanations in much of the early and mid twentieth century. From this history I developed and explained some of the new tenets, methodology and progress that has come through the emergence of evo-devo over the last forty or so years. I then described some of the many calls for an evolutionary psychology to be based on evo-devo instead of NEP. I contrasted the differences between NEP and DEP and described what a psychological sub discipline of evo-devo is likely to look like.

I have also argued that there is a substantial difference between NEP and DEP using Imre Lakatos' MSRP. I have suggested that the hard cores of NEP and DEP contain different principles that are incompatible and incongruent in various ways. The MSRP does provide a good method of understand the differences between NEP and DEP but contrary to Ketelaar NEP and DEP are not compatible in the core assumptions of their respective theories. Instead it is likely to be the belt level theories and the heuristic progress of NEP that holds potential to be reinterpreted in light of the core assumptions and methodologies of DEP. NEP and its methodologies might still have much to offer evolutionary psychology but it must follow suit with the rest of biology and incorporate development. This will require changes to some of the main theoretical tenets that NEP holds.

DEP represents a conception of evolutionary psychology that is compatible with modern evolutionary biology while NEP is not. This makes DEP a much better foundational candidate for evolutionary psychiatry based on this fact alone. In the next chapter I will present an outline of how evolutionary psychiatry might differ with DEP as a foundation compared with much of current evolutionary psychiatry that uses an NEP foundation. I will also examine how DEP can provide a better grounding for evolutionary psychiatry by showing how this approach can solve some of the problems of evolutionary psychiatry that I discussed in the second chapter.

CHAPTER 4: DEP AND EVOLUTIONARY PSYCHIATRY

4.1 Introduction

In chapter 3 I argued that DEP provides a better biological foundation for evolutionary psychiatry than NEP does. This foundation is consistent with modern evolutionary biology in a way that NEP is not. One of the most fundamental differences between these approaches is the importance of assigning CR function (particularly in the form of homology) before assigning SE function. DEP does not abandon SE function but simply relegates it to a more appropriate role. In some sense DEP protects SE function from misuse. In this chapter I will argue for the use of CR function as the better functional type for ascribing mental disorders. This chapter will focus on how evolutionary psychiatry might look when utilizing DEP as a foundation as opposed to NEP.

First I will look at Wakefield's arguments for the necessity of SE function in mental disorders. I will then argue that SE functions cannot provide the objective causal explanations that both Murphy and Wakefield seek in order to separate different kinds of

mental disorders and also that Wakefield's harmful dysfunctional account does not match many different people's concept of mental disorders. I will then look more generally at the history of the literature in distinguishing health from illness and argue for the importance of retaining functional talk when dealing with mental disorders. Then I will describe how we can use CR function and dysfunction to describe mental disorders.

I will then suggest that there are different motivations among Murphy, Wakefield and the DSM that suggest different goals for psychiatry. I then examine the role the natural kinds plays in psychiatric taxonomy and argue that a conception of non-traditional kinds replace the problematic traditional account. I then describe a DEP CR functional account of mental disorders and how it can help explain the evolutionary explanations for OCD that proved problematic for Murphy's taxonomy. I then conclude by considering some objections to my view.

4.2 Function in Psychiatry

4.2.1 Psychiatry and Traits

In chapter 2 I laid out three assumptions that I argued Murphy's taxonomy rested on. The first was an NEP picture of the brain. The second assumption was the "intentions of nature" idea where the "intentions of nature" are fulfilled when the NEP modules are functioning to solve the adaptive problems they arose to solve. The third assumption was that the two previous assumptions together could help tell us what a mental disorder is and how to group these disorders.

We can describe these three assumptions with the help of the background in chapter three as answers to three questions. The question how do we study behavior and divide it into "traits" is answered by Murphy's use of the NEP assumptions which

provides the NEP adaptationist methodology to divide traits into SE functional modules. The question how to divide traits into dysfunctional units is answered by the "intentions of nature" idea by comparing whether or not traits are functioning as there were intended to function in the environments they arose in. Finally the third question what are mental disorders is answered differently depending on the author but both Wakefield and Murphy think that mental disorders must be minimally SE dysfunctional modules.

Chapter 3 was largely about challenging the NEP answer to the first question. I laid out the history and current status of evo-devo and argued that DEP was far superior to NEP because it was compatible with modern evolutionary biology and therefore a better scientific foundation for evolutionary psychiatry. The "intentions of nature" idea is based on the NEP assumptions and these two ideas together were used to argue for a specific conception of mental disorders. Using the new foundation of evolutionary psychology I argued for in chapter 3 I will now examine the evolutionary psychiatry literature and argue for a different understanding of dysfunction and ultimately a different concept of mental disorders.

I will look at the arguments Jerome Wakefield offers for Murphy's two assumptions as his evolutionary explanations fit well into Murphy's taxonomy and he shares the "intentions of nature" idea. He also shares the commitment of using the "intentions of nature" idea to determine what a mental disorder is as both Murphy and Wakefield seem to hold that it is an objective matter to determine what a mental disorder is once you have the equally objective analysis of the "intentions of nature". The fact that they seem to have different intuitions about the concept of a mental disorder is part of what I will use to undermine this argument but first I will discuss Murphy's second

assumption about the "intentions of nature" idea and its potential for describing dysfunctions in evolutionary psychology and ultimately psychiatry.

4.2.2 Expressing dysfunctional traits as SE functions

Wakefield's position is based on the assumption that we can atomize traits and ascribe a specific function to each individual trait. As we saw in chapter 2 this is connected to an NEP understanding of evolution in which the mind consists of modules or mechanisms that have arisen in a hunter-gatherer evolutionary past in response to specific environmental problems. In chapter 3 I looked at the modules of NEP which are the "atoms" in the atomization of traits that Wakefield's "mechanisms" are based on. This discussion showed that if these modules even exist they are heavily constrained by physical developmental modules which help determine what environmental problem is being solved and at what level the solution will occur. The environment is a co constructor of traits not the triggering mechanism that NEP envisions it to be. Downes hand example is once again useful in suggesting that we cannot in fact atomize traits in the way Wakefield claims is important. I concluded by arguing that CR function should be the fundamental unit of function which constrains the ascriptions of SE function. I now look to extend that to argue that CR function should also be the fundamental unit of function when ascribing dysfunctions and ultimately mental disorders. I am not the first to criticize SE dysfunction for mental disorders and suggest CR function instead.

Richard J. McNally has argued for CR function to be used as the unit of psychological dysfunction that we use to classify mental disorders. McNally argues that confirming a current causal role was naturally selected does not add anything to our understanding and determining historical adaptive value or even current adaptive value is

quite difficult relative to determining CR functions. He suggests that evolution cannot provide much of substantive value to psychiatry (McNally, 2001, p. 312). Therefore Wakefield's account makes things more difficult than need be and by doing so does not add anything to the value of the analysis.

Wakefield responds to McNally's claim that evolution does not add anything to an account of CR function:

> Only the functional-design account has the power to explain how it can be that some dysfunctions are valued or at least not disvalued, and some functions are disvalued or at least not valued, as illustrated above. If one is to explain disorder attributions, one cannot replace the evolutionary standard with a value standard; both standards must be involved (2001, p. 353).

Wakefield is here reiterating his claim from chapter two only a SE dysfunction can be a candidate for a mental disorder as CR function does not have the explanatory/historical information about design that is necessary for distinguishing dysfunctions from undesirable outcomes. For Wakefield there is a distinction between normal grief and depression that is pathological. Normal unhappiness is not an adjustment disorder. Wakefield argues that we implicitly use a "failure-of-designed-function" criterion when we are making these judgments (Wakefield, 2001, p. 362). These are all disvalued and harmful conditions but only abnormal conditions that are disvalued and harmful should count as mental disorders.

The first thing of note here is that McNally uses the term "Current Causal Role" (CCR) function instead of simply CR function and this appears to be a way of emphasizing his argument that an evolutionary analysis adds nothing to psychiatry. McNally and I agree that CR functions are easier to assign than SE function but we obviously disagree about the importance of evolution to psychiatry. He appears to be

equating "evolutionary analysis" with "adaptive analysis" which is potentially due to the fact that most evolutionary psychiatry is heavily based on NEP although as discussed in chapter three a great deal of evolutionary analysis can be done without reference to SE function at all. Of course when SE function and adaptationist hypotheses are properly informed and constrained by a foundation of DEP we can also discover a great deal about psychological traits and also about potential mental disorders that can arise. I maintain that evolution can add a great deal to psychiatry both by using non-adaptive evolutionary methodology as well as using adaptive methodology in the right way. I will argue further for this claim later in the chapter but for now I remain focused on analyzing this debate and clarifying the differences between McNally and myself in our use of CR function and the different ways that we disagree with Wakefield on the subject.

I suggest that McNally's CCR account of function can be compared to the tradition of the majority of the structuralists during much of the 20th century. They both focus on a pure CR functional account that ignores evolution. On the other hand Wakefield's SE account of function is comparable to the functionalist tradition with a focus on adaptive traits and little concern for competing conceptions of function. These analogies are not apt in every way as NEP is not synonymous with SE function and in fact uses SE function in a specific and arguably extreme way by postulating modules that require natural selection to be an extremely powerful evolutionary force. The functionalist tradition was not always as strongly dismissive of evolution as McNally is either as shown by the successes of evolutionary morphology.

None the less these comparisons are helpful because they help to clarify my position as I seek to reject and keep aspects of both McNally and Wakefield's positions

by following the evo-devo tradition of recognizing the importance of developmental explanations from the structuralist tradition while merging equally important evolutionary explanations traditionally offered by the functionalist tradition. Therefore we need to recognize the importance of CR function from McNally while recognizing that this does not prevent us from exploring the vitally important evolutionary perspective which Wakefield argues is important. Of course my evolutionary perspective differs greatly from Wakefield's, one of the most obvious ways being in the use of CR function.

CR can be used in evolutionary contexts and using it does not exclude the possibility of adding SE functional analyses and in fact if anything it increases the likelihood that those analyses are going to be of a high quality. So I suggest using a DEP conception of CR function (which includes the importance of adding SE functional analyses where appropriate) instead of a CCR function or an NEP conception of SE function. I will now argue that this DEP understanding of CR function presents a better way of answering our second question about defining dysfunctions than does the NEP understanding of SE function and the "intentions of nature" that Wakefield relies on. I will first suggest that even if we use SE function we cannot properly explain dysfunction in terms of whether or not the "intentions of nature" are being expressed. I will then explain how CR function can offer an alternative that has a better chance at success.

The NEP modules that Wakefield endorses refer to the environment of the past as a reference to evaluate the functionality of traits. This "environment" is of course the EEA and represents a correlation of many different environments. Traits evolved in different environments but are capable of promoting survival in a variety of these. Organisms that survive are often those that are more plastic as they are likely going to be

better at solving short term problems that arise and they are also likely to be better at solving long term problems that occur at different "grains".

If even some of our traits arise due to plasticity and environmental interaction as opposed to "triggering" then we need a way to account for this even if we accept an NEP style SE functional account of modules. But how can Wakefield's "as nature intended" idea capture this plasticity? If a trait is valuable because it is plastic and because it can create new functional capacities in humans how can we tell if it is working properly? Let's say there is a general developmental trait A which creates novel function A1, A2 and A3 in a modern environment. Is this trait functioning properly, as it was historically? In one sense it is. Trait A is adaptive and evolved historically by helping the organism survive. However, the new environmentally created functions A1, A2 and A3 are not functioning in the historical sense as they should be. However, A1, A2 and A3 are the result of adaptive processes so we should be able to describe them as SE functions. The problem is that we cannot because these functions never existed historically there is no way to tell if this trait is functioning as it should be and yet it is clearly adaptive.

Even cases of simple variation may be problematic. Wakefield argues that there is a normal range of behavior and an abnormal range of behavior both of which can be traced to design elements. Natural selection requires variation so adaptive processes can only emerge where variation is present. Because variation is essential for evolution we might argue that even variation outside the "normal range" is as much "intended" by evolution as the variation inside the "normal range". Of course there are some traits that are fixed within a species, but contrary to NEP these traits are more likely to be far deeper in our evolutionary history. Many aspects of our brains are shared not only with

all other humans, but with all other mammals. We have a better chance at describing many of these traits in terms of whether or not they are functioning as they should be. Many of our uniquely human traits will vary and some will diverge even further due to plastic changes that occur during an individual's lifetime.

Even if we look for mammalian wide traits they are likely to be discovered in terms of homology and therefore more likely to be identified with CR function than SE function. We are more likely to discover universal functions among more ancient traits. The most recent adaptations and changes in a developmental pathway are likely to be the ones which are most likely subject to change through co construction via the environment. If we attempt to describe the functioning of certain aspects of the brain and mind in terms of adaptations from the Pleistocene only then we fail to account for the homologous structures upon which these adaptations were built. If humans used some kind of prey tracking mechanism in the Pleistocene it was almost certainly not developed *de novo* and was more likely developed directly from a structure for prey tracking in the ancestral species of many other mammals. This also means that any new adaptations from the Pleistocene are likely to be functionally integrated as well as developmentally integrated.

Griffiths notes that evo-devo has produced sophisticated work linking the concepts of homology, modularity and character allowing such a division of characters to take place (Griffiths, 2006b). Marc Ereshefsky extends this to the realm of understanding psychological categories as homologies. He supports the examination of ethologist theories about the phylogeny and ontogeny of behavioral homologies. Operational methods for testing behavioral homologies and theoretical insights from ethology can be

applied to the study of psychological homologies using both adaptationist and functionalist approaches (Ereshefsky, 2007).

One of the insights from evo-devo is that HOW things evolve can be as illuminating, if not more illuminating than the adaptationist question of WHY they evolved. It can also be epistemically easier to discover facts about how things evolved than why they evolved. We can discover the internal developmental and genetic changes that led to diversification without knowing the environmental problems that an organism was facing. This knowledge will always be on a more solid foundation than knowledge about adaptive reasoning. This is not to say we should abandon adaptive reasoning, but simply that we should not start with it as is common methodology in NEP, Murphy's taxonomy and Wakefield's harmful dysfunction analysis.

Therefore a biologically realistic account of evolution shows that we should not expect to find atomized "intentions of nature" which can be used to distinguish mental disorders from behavior that is simply negatively valued. This is not to discount the importance of natural selection but instead to suggest that the way that NEP expects to be able to discover and describe traits is biologically unrealistic.

In this section the NEP influenced assumption that each mental disorder can be described as a dysfunction of a SE function that was selected to solve a specific adaptive problem in our evolutionary past has been shown to be unrealistic and based on inadequate biology. I have offered a DEP version of CR function as an alternative as it is a more biologically adequate conception of function to base an account of dysfunction on. Later in this chapter I will show how CR dysfunctions can help to better define mental disorders but first I must show that Wakefield's claim that a historically based

account of functionality and design reflects people's concept of what a mental disorder is does not always hold true.

4.2.3 The concept of a mental disorder

In chapter 2 I showed that Wakefield argues for a "harmful dysfunction" account of mental disorders. Wakefield argues that all theories of mental disorder are in their essence theories of dysfunction accompanied by value judgments which transcend the sphere of functional ascriptions (Wakefield, 1992a, p. 385). Therefore he suggests that:

> The concept of disorder thus places two constraints on any theory of mental disorder. The value criterion implies that any successful theory of disorder must link up in the right way with the commonsense concept of harm. The explanatory criterion implies that any successful theory must offer an account specifically of dysfunctions (Wakefield, 1992a, p. 385)

Wakefield suggests that any method of meeting these criteria must essentially involve SE functional ascriptions:

> Because natural selection is the only known means by which an effect can explain a naturally occurring mechanism that provides it, evolutionary explanations presumably underlie all correct ascriptions of natural functions. Consequently, an evolutionary approach to personality and mental functioning (Buss, 1984,1991; Wakefield, 1989a) is central to an understanding of psychopathology (Wakefield, 1992a, p. 383)

This is why Wakefield only counts evolutionary explanation in Murphy's breakdown category as mental disorders. Mismatch explanations cannot count because there is no "dysfunction" according to Murphy's definition or in other words the "intentions of nature" are being fulfilled.

Murphy would agree that the "intentions of nature" are being fulfilled but for Murphy the fact that these intentions are expressed in a relevantly different environment is enough to count the behavior these explanations were describing as mental disorders.

Stevens and Price obviously agree with Murphy and in fact their entire book is based around the theme that what is perhaps unique about a union between evolution and psychiatry is the ability to identify mismatch explanations (Stevens & Price, 1996). Murphy, Wakefield and Stevens and Price all share a NEP understanding of the brain and all believe that evolution and the "intentions of nature" are part of defining mental disorders and yet there is dissent on their conception of what a mental disorder is. Not to mention the fact that Cosmides and Tooby hold that while evolution can provide objective definitions of function and dysfunction these definitions do not suggest anything about mental disorders which for them lies in the realm of values (Cosmides & Tooby, 1999).

Although there is dissent among Wakefield, Murphy and Stevens and Price about exactly what the conception of a mental disorder is they all think that a mental disorder is a comparison against some kind of natural functional state. Murphy and Wakefield agree that persistence explanations should not be counted as mental disorders for example because there is no deviation from what we might call "normal function" as the "intentions of nature" operating correctly in the wrong environment is not functioning normally.

Ron Amundson is a strong critic of the concept of normal function. In his aptly titled article "Against Normal Function" he argues that an examination of current biology fails to show a clear distinction between normal and abnormal function (Amundson, 2000). He makes an important distinction between the level of performance of a function and the mode of its performance (as cited in Silvers, 1998, p. 101).

> Functional mode is the manner in which a functional outcome or performance is achieved. Performance level is the quantitative degree of the functional

> performance, such as the speed or the strength of a motion. In addition, we can identify functions at different organizational levels of the biological hierarchy (Wachbroit, 1994b, p. 237). Functions can be seen as occurring at genetic and physiological levels of the hierarchy, at the level of limb movements, and even in ecological interactions (e.g. 'obtain food'). Whatever the hierarchical level, functional determinism states that functions take place in a uniform mode at a relatively uniform performance level by a statistically distinctive portion of the members of a species. These are the normal's (Amundson, 2000, p. 36).

Amundson refers to some of the same factors as me when describing the problem with "normal function": "Variation is ubiquitous. It is always 'different, not abnormal' simply because there is no Darwinian interpretation of abnormality" (Amundson, 2000, p. 36). He notes genetic diversity (p. 38) as well as plasticity (p. 39) help to refute a Darwinian concept of normality. In terms of deviation from typicality Amundson suggests that:

> A non-typical but viable phenotype is not broken by its failure to comply with some imagined blueprint for its species. It will function anyhow, in spite of its atypicality. It will owe its function to the same developmental processes of integration and adaptation responsible for the function of typical organisms of its species (Amundson, 2000, p. 39).

Amundson cites Hydrocephaly as an example of a "disorder" that can cause both physical and mental problems. Cerebrospinal fluid backs up and causes brain ventricles to greatly increase in size. This results in pressure which can enlarge the cranium and/or reduce the amount of brain tissue that develops in a person. Cases where 95% of the cranium is filled can cause severe disability but half of these cases show people with IQs over 100 (Amundson, 2000, p. 40). Amundson describes "The mode of function of these persons is statistically 'abnormal', even though their level of performance is statistically average" (Amundson, 2000, p. 41).

Amundson addresses the concept of normality in anatomical and medical sciences by describing and endorsing Jir'ı' Va'cha's (1982) basic position: "He considers the

common use of 'normality' to be typological and idealistic in that it assumes that 'the frequent [is] the [normal] and, besides that, the healthy' "(Amundson, 2000, p. 43). Instead of a dichotomous health/illness state Amundson agrees with Va'cha that health is a multi dimensional spectrum of health and normality. Sometimes within individuals some extreme variables are compensated for by other variables. Va'cha follows the lead of L.R. Grote and argues for individual normality or "responsive" take over the concept of species normality that currently dominates (Amundson, 2000, p. 44). This conception focuses on the relation between individual performance and whether the needs of said individual are met. What is interesting is that the concept of normality in physiology and anatomy seems to be based more upon statistical rubrics than it is based upon historical design specifications.

In terms of psychiatry Norman Doidge criticizes the NEP claim that adult human brains have remained unchanged since the Pleistocene as neuroplasticity is a part of our genetic heritage and the brain has the ability to change its own structure and function through thought and activity (2007). He cites the fact that the occipital lobe which normally processes vision can also process sound and touch when blindfolded. Doidge's influential book The Brain that Changes Itself (2007) collects stories from brain researchers who have induced enormous change in plastic brains of patients. Stroke victims have been told that they will never walk again and yet have gained varying degrees of mobility back through therapies that take advantage of neuroplasticity using to create new neural connections. Sometimes after injury healthy areas of the brain take on tasks that normally are done by the damaged areas (Doidge, 2007). In psychiatry neuroplasticity has helped many with depression and OCD to "rewire" their brains to

produce different neural connections and create different functional capacities (Doidge, 2007).

Amundson also discuses a psychiatry example where people with autism sometimes use "abnormal" behavior to achieve a "normal" functional end:

> Many people with autism engage in 'stimming', small repetitive self-stimulations like rocking while sitting, tapping one's face, or flipping fingers in front of one's eyes. These visibly abnormal behaviors are strongly discouraged by most educators. But it turns out that stimming is often functionally beneficial to people with autism. It reduces the chaos they experience, chaos created by their heightened sensory sensitivity in 'normal' environments, and allows them to concentrate on particular features of the environment (e.g. the voice they are listening to) (Amundson, 2000, p. 49).

Like the hydrocephaly case these examples show various modes of function that are statistically "abnormal" although they produce levels of performance that are "statistically average" or at least much closer to average than they would be using the "normal" functional mode. The abnormal mode produces results that are closer to "normal". There is great difficulty in objectively defining normal function and an appeal to SE function makes the problem worse. I have described philosophers, scientists and psychiatrists that describe disability, dysfunction and disorder in terms that do not seem to be expressing implicit historical design concepts and so SE function does not appear to be the universal concept of disorder that is suggested by Wakefield. There does not seem to be a universal concept of normal function that we can use as a comparison in order to obtain the objective dysfunctions Wakefield requires.

I suggest that once again CR function is a better option than SE function in an attempt to describe deviations from normal functioning that can be conceptualized as mental disorders. CR function avoids the assumptions about statistical normality and historical selective functions that are clearly not apparent in everyone's understanding of

normal function. DEP concepts such using as homology to explore variation in terms of patterns of difference and similarity is more in line with Amundson's views of variation in function compared with SE function which requires *a priori* assumptions about normality. Doidge references the need to incorporate an understanding of plasticity into psychiatry and again a component of DEP is plasticity which can use CR function to explore the goals of new variants of traits that have come into existence in response to current environmental factors. CR function through a DEP lens therefore avoids some of the criticisms that SE function through an NEP lens has garnered although I have not yet made the positive case for a DEP CR dysfunctional account of psychiatry. This will come throughout the rest of the chapter but first I will summarize this section.

In this section I have suggested that Wakefield's concept of a mental disorder is not biologically plausible as we cannot assign SE functions to the specificity that is required for Wakefield to define mental disorders as SE dysfunctions. I have also rejected Wakefield's claim that the concept of mental disorder is universally held to relate to a historical dysfunction as even those who share all of Wakefield's assumptions do not share his conclusions about the concept of a mental disorder. I have also showed that the entire concept of normal function that is required for Wakefield's concept of a mental disorder is not biologically plausible or universally held either. Therefore between chapter 3 and this section I have now shown that all three of Murphy's assumptions that he uses to build his taxonomy in chapter 2 fail to answer the implicit questions they are required to.

I have also shown that a DEP based CR functional account solves many of the criticisms and problems facing the NEP based SE functional account by using different

assumptions. First instead of NEP behavioral traits should be studied by DEP methodology using DEP ontology. Second instead of referring to the "intentions of nature" dysfunctional traits should be defined in terms of dysfunctional CR functions. Third dysfunctional CR ascriptions can avoid the problematic assumptions of limiting mental disorders to only SE dysfunctional ascriptions. CR dysfunctional ascriptions can also express SE dysfunctional ascriptions as mental disorders if and when it is appropriate to do so. Therefore a DEP CR functional account of mental disorders can solve some of the problems of NEP although I have not yet answered the questions of how this account can deal with the incompleteness problem that beset NEP evolutionary psychiatry as described in chapter 2.

In order to accomplish these tasks I need to examine what the goals of psychiatry are. Murphy assumed that evolution could help psychiatry through the first goal of differentiating different kinds of mental illnesses from one another while differentiating mental illness from normal behavior. The second goal Murphy aimed to help with was providing information about the mental illnesses through the robust causal distinctions of the taxonomy. I will now return to the question of motivation in psychiatry by examining the literature on health and disease in order to determine what a DEP based evolutionary psychiatry is positioned to help to provide psychiatry.

4.3 Psychiatry and Health

4.3.1 Health and Disease

Marc Ereshefsky in a recent article on health and disease nicely describes the three main positions in the literature:

> Naturalists (Kendell 1975; Boorse 1976, 1977, 1997; Scadding 1990) desire definitions based on scientific theory. Their definitions attempt to highlight what is biologically natural and normal for humans. Normativists (Margolis 1976, Goosens 1980, Sedgewick 1982, Engelhardt 1986) believe that our uses of 'health' and 'disease' reflect value judgments. Healthy states are those states we desire, and diseased states are those states we want to avoid. Hybrid theorists (Reznek 1987, Caplan 1992, Wakefield 1992) define 'health' and 'disease' by combining aspects of naturalism and normativism. Their aim is to provide an account of health and disease that captures the virtues but not the vices of naturalism and normativism (Ereshefsky, 2009a, p. 221)

Ereshefsky cites problems common to all of them. Naturalism fails at finding definitions of 'health' and 'disease' that are objective as we have seen with Wakefield previously. Wakefield claims that there is an objective definition of healthy behavior and diseased behavior but his supposed objective concept is rejected by many others who argue that objective criteria for normal behavior cannot be found. Normativism on the other hand is unsuccessful at capturing the intuitions of lay people and medical practitioners who feel that disease must be more than value judgments about whether or not a condition causes some kind of negatively valued experience for the person in question. The hybrid approach shares the problems of naturalism by failing in the task of describing objective criteria for health and disease. The systematic problem identified by Ereshefsky is that naturalists and normativists each fail to recognize at least one of the two salient factors in disease: the physiological or psychological states of patients and the values we attach to those states (Ereshefsky, 2009a).

Although hybrid theorists consider both aspects, diseases are restricted to states that are both dysfunctional and disvalued. For Ereshefsky this prevents us from adequately dealing with the controversial cases that are at the heart of the debate. Instead of continuing the unfruitful quest of differentiating health and disease Ereshefsky recommends that we directly focus on what is at stake in health and disease ascriptions:

state descriptions (descriptions of physiological or psychological states) and normative claims (claims about what states we value or disvalue) (Ereshefsky, 2009a).

Ereshefsky gives some example of state descriptions. We can measure the amount of calcium in a patient's tissues or we can describe a patient whose red blood cells are rupturing. Ereshefsky also argues that we can describe how a patient feels or provide technical details for a description of a psychological state in a patient. State descriptions avoid terms such as "natural" and "normal" even if it is impossible to eliminate all normative talk. Ereshefsky notes that normative assumptions typically go hand in hand with functional claims in medicine and therefore we are better off using state descriptions to avoid this (Ereshefsky, 2009a).

Ereshefsky defines normative claims as explicit value judgments regarding the relative worth of a physiological or psychological state. We are making value judgments when we promote, avoid or seek certain states. We do not value the state of having rupturing blood cells, while we value having legs that can walk, while being indifferent about whether or not people are gourmets. Normative claims are made when value judgments become explicit (Ereshefsky, 2009a).

Ereshefsky argues that this can help us in controversial cases such as whether or not deafness is a disease. Using his approach we can see that what is at stake is a state (deafness) which is agreed upon, while whether or not we value it is what is in conflict. We avoid the debate about whether or not deafness is a "disease". Some people value deafness and think it has benefits over being able to hear (Ereshefsky, 2009a). What is at stake in this debate is therefore clarified by Ereshefsky's method.

State descriptions also avoid talk of normality and so avoid the problems of the hybrid and naturalist theories. Ereshefsky thinks that state descriptions avoid functional talk and this is also a benefit and cites Wachbroit (1994b) and Cooper (2002) who suggest that when we lack empirical or theoretical evidence for ascribing function in a biological or psychological system we then assign function normatively. Therefore naturalism and hybrid theories risk normative functional ascriptions being discretely equated with state descriptions because the concept of 'normal function' plays a central role in their definitions (Ereshefsky, 2009a).

Ereshefsky argues that his theory avoids a problem in normativism where we attempt to capture the intuitions of how we use the term "disease". If disease is simply an evaluative concept based on what we disvalue then why is there debate about whether or not alcoholism is a disease? There appears to be an intuitive distinction between disvalued states and disvalued states that are also diseases (Ereshefsky, 2009a). Instead of attempting to capture this distinction Ereshefsky instead simply avoids this problem by breaking down the problematic cases into state descriptions and normative claims.

Ereshefsky argues that his theory has three main benefits. First, it clarifies the discussion surrounding controversial cases. Second, 'health' and 'disease' become superfluous as we cut to the heart of the matter in medical discussions, particularly the controversial cases. Third, the problems facing the major theories of 'health' and 'disease' are avoided (Ereshefsky, 2009a).

Ereshefsky clarifies that he is not arguing for the elimination of the terms "health" and "disease", simply that in controversial cases where this distinction is not agreed upon, we should revert to talk about state and value descriptions. Ereshefsky cites the

similarity of the situations with the terms 'species' and 'gene' (Ereshefsky, 2009a). Scientists disagree about how to define species. As a result the meaning behind the term "species" is typically clarified when used in professional publications and conferences, yet "species" is still used in general textbooks and by the general public in common discourse. Similarly we could avoid use of the terms "health" and "disease" in technical discussions and instead focus on state descriptions and values. Ereshefsky still expects "health" and "disease" to be discussed in clear cut cases and to continue to be used by laypeople (Ereshefsky, 2009a).

Ereshefsky criticizes normativism for failing to capture the intuition that there is a distinction between disvalued states that are medical conditions and disvalued states that are not. He accepts that his theory cannot account for this either, but it doesn't matter because he is not attempting to give an account of health and disease but rather an account that eliminates these terms. Nevertheless, Ereshefsky picks up on a sociological distinction suggested by Rachel Cooper that he feels might be useful: after deciding that a state is not valuable that state can be defined as a medical disease if it is treated by health care workers, otherwise it is not a disease (Cooper, 2002 as cited by Ereshefsky, 2009a, p. 226).

A final concern that Ereshefsky addresses is whether or not his distinctions between state descriptions and values is a false dichotomy considering the common claim that science, and especially the human sciences are laden with normative values. For example, Ereshefsky cites Amundson and his claim that purely medical accounts of disability in fact use social values when defining quality of life issues (Amundson 2005

as cited in Ereshefsky, 2009a, p. 226). Terms like 'abled' and 'disabled' beg the question toward the abled suggesting that they have higher quality lives.

Ereshefsky understands state descriptions as containing no explicit normative parts and by keeping the distinction between state descriptions and normative claims we can illuminate implicit normative assumptions:

> Once values are seen as entering a discussion, any talk of values is highlighted as a 'normative claim'; that way, discussions concerning values will be explicitly normative. State descriptions will never be completely value-neutral, but we can do our best to label value judgments as such when they are identified (Ereshefsky, 2009a, p. 227).

Using a distinction between state descriptions and normative statements makes explicit the presence of values. That is an improvement over the naturalists and hybrid theories that use concepts like normal and natural, which are typically value-laden, although taken to be descriptive. Ereshefsky argues that his theory can avoid disguising these normative concepts as descriptive concepts. Naturalist and hybrid definitions of 'health' and 'disease' fail to make values explicit, while Ereshefsky's theory accomplishes this to a far greater degree (Ereshefsky, 2009a).

Ereshefsky concludes by stating that he disagrees with the commonly held belief that 'health' and 'disease' are real categories in nature and therefore they are natural kind terms. Biological theory does not distinguish natural states from unnatural states and therefore the naturalists and hybrid claims that we can objectively determine normal and abnormal states fails. The best evidence for natural kinds such and 'health' and 'disease' would be scientific evidence but our best scientific theories fail to provide this evidence and we should therefore doubt that health and disease are natural kinds (Ereshefsky, 2009a, p. 26).

Ereshefsky provides one last benefit of his theory as aiding in distinguishing the current state of the world from future states that we want to promote or diminish. When we talk about a disease we can be referring to current states we are in and/or future states we desire to be in and this may differ between speakers. State descriptions make this distinction more explicit than talk about "health" and "disease" (Ereshefsky, 2009a, p. 26).

Ereshefsky's analysis highlights a great deal and I agree with much of his analysis. His description of the failure of the three major theories of health and disease is concise and accurate. He also echoes some arguments I have presented earlier regarding the difficulty of deriving concepts such as "normal" or "natural" from biological theory. I also concur with his next step of attempting to clearly articulate what it is that we are interested in with medical discussions. Ereshefsky is dealing with health in general, although he expects this to cover mental health as well as physical health. My interest here is in mental health although nothing I say would prevent an extension to physical health.

Where I disagree with Ereshefsky is his dismissal of functional talk. I agree with Ereshefsky that functional talk can involve values but as he admits so can state descriptions. Functional talk can import assumptions about normality as NEP's use of SE function is an explicit part of the theory. CR functioning however does not have a concept of normality inherent in it. This is not to suggest that CR function is value free in fact in one sense the opposite is true. When we use CR function we must first *explicitly* choose a set of interests which sets the level of analysis as well as the goals and the boundaries of the system. This provides a better way of making the values explicit

and focusing on what it is that we are actually valuing. Both of these goals of Ereshefsky's are simply better met by CR function than SE function and even state descriptions.

For example in the debate about deafness we could list two different state descriptions as "hearing enabled" and "deaf" and then state that we value "hearing enabled" over the state of being "deaf". This is problematic though because we fail to realize *why* we value this state over the other. We might assume that we value the "hearing enabled" state at least in part because it allows us to communicate and we disvalue being deaf because it prevents us from this function. In reality though someone who is deaf may simply function atypically with an atypical mode of communication but still achieve a high degree of successful communication.

State descriptions can reinforce the idea that some states are intrinsically valuable because those who function abnormally do so because of a problem internal to the person. Of course states are often only valuable in relation to other social and environmental parameters. If someone who is deaf can function equally as well as someone who can hear then it becomes unclear why we disvalue that state. We must make it explicit whether or not it is the ability to communicate or being "hearing enabled" that we value.

A CR analysis can take these facts into account as we are forced to explicitly choose goals and boundaries for the system that we are dealing with. If the boundaries of the system is the individual person and the goals of the system is simply being "hearing enabled" then the deaf person is dysfunctional because they are deaf and unable to perform the function of hearing. However an equally valid CR functional analysis could

set the boundaries of the problem as the physical or social environment and set the goals of the system to be "communication" instead of "hearing" and under this analysis the person is not dysfunctional when certain social or environmental parameters are met. In essence if the person cannot communicate the dysfunction is shifted from the person to the environment. In the first analysis there is something "wrong" with the person but in the second analysis there is something "wrong" with the environment.

Agreeing on an anatomical description of deafness and showing that some think it is a valued state and others think it is a disvalued state really gets us no further in clarifying why there is disagreement. The deeper values at play in the disagreement are only illuminated when we talk about the *functional* abilities of someone who is deaf and examine various goals of the various different systems we could analyze. Therefore in order to make our values truly explicit we require CR functions to be the unit of dysfunctional behavior. Only CR functions are required to choose ends before analyzing a system.

For example if we chose to analyze the goal of listening to music then this is something that cannot be overcome by environmental accommodation. This highlights an intrinsic reason why deafness would be disvalued. Of course some might still value deafness for intrinsic reasons such as heightening other senses or possibly even allowing a higher degree of communication using a different mode such as sign language. Examining different possible goals of a system force these sometimes hidden and possibly implicit values to be elucidated. State descriptions only ask *if* we value a behavior and we risk losing the insights gained from asking *why* we value a behavior.

For mental disorders we can also give state descriptions such as the description that depression is a biochemical imbalance in the brain. However engaging in a discussion about this state of affairs would mean a lot more if we knew that some people also described it in terms of negative cognitions or negative thinking patters. How we value depression and the discussion around depression is likely going to be influenced by whether or not it is described as a biochemical or a cognitive problem. Agreeing about how to describe the state may be precisely what is at stake.

Choosing a CR functional description requires values to set the goals and boundaries of the system as well as setting the level and type of analysis. Ereshefsky is concerned that when we lack evidence for ascribing function in a psychological system we assign function normatively but using CR function forces us to make this explicit while state descriptions can be provided without offering any account of their potential normative import. We could compare state descriptions and attempt to describe potential normative import but there is no impetus to in Ereshefsky's theory and in fact the separation of objective state descriptions from normative factors is the driving force of the model. Even if we wanted to compare state descriptions at different levels of explanation it is difficult to find any common frame of reference.

CR function on the other hand allows us to compare the different descriptions by comparing the goals of the different systems. The goals that are being thwarted in depression can be stated in biochemical terms or cognitive terms and we can compare the goals of two different systems at two different levels studying the same problem. In terms of depression it has been suggested that biochemical and cognitive change is intertwined and represents different aspects of the same phenomena. Therefore CR

function can study these two systems and how the goals of each system respond to changes in the other system. This not only provides better science but also allows us to cut to the heart of the debate and expose values.

For example people may disvalue depression when it's couched in biochemical terms but feel completely different about it when expressed as a cognitive problem. These value judgments are completely obscured when we simply describe depression as either a biochemical problem or a cognitive problem alone. Stating that someone has a low level of some neurotransmitter is not as powerful as a functional description that describes how the low neurotransmitter level affects goals at other levels of analysis such as the cognitive.

Therefore, to sum up, I agree with Ereshefsky that avoiding definitions of "health" and "disease" provide a good way of moving the debate around controversial cases of "health" and "disease" forward and avoiding some of the problems of the other major theories of health and disease. Therefore I am not interested in providing definitions of health or disease and I do not concern myself with distinctions between disability, disease or disorder either. My focus remains on arguing for a DEP concept of "mental disorder" and how we should define individual mental disorders using CR function.

Like Wakefield Ereshefsky still assumes that there is some way we can isolate scientific or factual matters and then subject them to value analysis. However avoiding discussion of functions only serves to further obscure values and lower the range of scientific resources available to solve problems. The important intuition that mental disorders involve both values and facts (typically scientific facts) is one that I agree with

but values and facts are intertwined together at all levels of analysis including when choosing a description of the mental disorder we want to study. Therefore CR functions and dysfunctions provide a better way of describing mental disorders than state descriptions because they force our values to become explicit and they better help us focus on important issues which remain hidden on many analyses of state descriptions. I will now provide a general account of how we can describe mental disorders using CR function and dysfunction.

4.3.2 CR Function and Dysfunction for Mental Disorders

There are three different questions we need to ask before attempting a CR functional analysis of a mental disorder. First we need to determine what level or type of analysis is appropriate, second we need to define the system we are analyzing (or the boundaries of the system) and third we need to determine the goals of the system we are analyzing. The level of analysis helps to determine what our choices are for the system we are analyzing and the boundaries of the system we choose help us in choosing the goals of the system.

In order to get some idea of possible levels of analysis I look to the philosopher Jason Robert who is an advocate of using multiple levels of analysis in psychiatric classification and suggests the following approach:

> An integrative, systems approach to the brain recommends an alternative approach to psychiatric classification, one that reflects input from all relevant sources, incorporating multidirectional feedback between genes and larger phenotypic and environmental elements. This integrative approach to nosology requires that the nature of psychiatric classifications be decided by interlevel, interdisciplinary negotiation rather than reductionistic revelation. To be sure, the point of the foregoing is not to deny the importance of biological aspects of psychiatric disease; it is instead to begin to understand such disease at an appropriate level of complexity. But clinical phenomenology is of absolute necessity (Robert, 2007, p. 213).

Robert is reacting to a reductionist ideology that understands "true disease" existing at the level of genes and brains. Some of the levels of complexity we can use are biochemical/physiological, the cognitive and the social/environmental. At all of these levels we can assign CR functions and dysfunctions. When we assign CR functions and dysfunctions at multiple levels and compare the results we are in the best position to obtain maximum causal information about a disorder including the interactions between CR function goals at different levels of analysis. We are also in the best position to simultaneously expose the values that are involved in assigning mental disorders. We need to consider both scientific and value questions at the same time because there is no way to objectively describe potential mental disorders without importing value judgments.

Sometimes when defining disorders the information about the disorder can help guide us toward the proper level of analysis. For example social anxiety disorder occurs when people experience extreme anxiety when placed in social situations. By defining this disorder in terms of biochemistry alone we are very likely to miss important salient facts about this disorder. Adding an analysis at the cognitive level might help but the social/environmental level appears to be able to provide the most important information.

Sometimes it is not clear what level of analysis is best. An analysis for depression might focus on a biochemical level that suggests that certain people are dysfunctional because of an insufficient level of the neurotransmitter serotonin. Instead we could perform a cognitive analysis in which we might determine that people's negative thinking is responsible for the depression. An environmental analysis might yield a different story in which poor social relationships lead to negative thinking which in turn is

correlated with biochemical changes. This latter analysis is valuable because it connects multiple types or levels of analysis, systems and goals and can be understood to be operating at multiple levels.

A biochemical analysis might define depression as a dysfunction in system of the brain with a failure of that system to meet the goal of keeping serotonin above a certain level. A cognitive analysis might focus on the mind with a failure to maintain a certain level of positive and realistic thought patterns. An environmental analysis might show that social relationship goals have failed to be met. Studying the connections between these types of analyses is the kind of interlevel investigation that Jason Robert suggests is important in psychiatry. It also helps to really elucidate the different values that are at work.

For example, Amundson notes that some disability theorists claim we should always look for a "dysfunction" in terms of social/environmental levels when there is a failure to accommodate different levels of function and different modes of functioning. This elucidates the point that many mental disorders might disappear if different social structures existed while other mental disorders might be more focused on emotional and cognitive problems that people want eliminated and there may be only a limited amount of accommodation that society can offer. These are all salient facts and values that must be open and on the table when defining mental disorders. Of course as Ereshefsky, Wakefield and others have pointed out values do not seem sufficient to describe mental disorders. Scientific reasons can be just as important in prompting us to choose one level of analysis over another or one set of goals over another.

The use of homologies common to all mammalian species may present a way of categorizing some mental disorders. A dysfunction can be explained by referencing a certain brain structure based on one of the core brain processes that exist in all mammalian brains (Panksepp, 2006, p. 777). These can often be connected by strong neural and genetic underpinnings. Homologies are central to H. Stefan Bracha`s argument for the use of the "Neuroevolutionary Time-depth Principle" of innate fears. He argues this can be useful for providing a more neuroevolution-based taxonomy for stress-triggered and fear-circuitry disorders in the upcoming DSM-V. This proposal suggests dividing up fear circuits according to the evolutionary time in which they likely originated. Four clusters are suggested: 1) Mesozoic (mammalian-wide); 2) Cenozoic (simian-wide) 3) mid Paleolithic and upper Paleolithic (Homo sapiens-specific); 4Neolithic circuits (arguably mostly related to stabilizing selection driven by gene–culture co-evolution) (2006).

These types of explanations can help us to evaluate the validity of certain CR functions and dysfunctions we might suggest for mental disorders. Even for something like social anxiety disorder that is based around human interaction homology may still be important. We saw with the OCD examples in chapter two that many current disorders may have evolutionary roots in mammalian wide brain structures. Finding homologous behavior in other mammals can help us evaluate the patterns of function that we see in humans. If social anxiety disorder showed evidence of being related to homologous behavior in other mammals like the explanation for OCD suggests then we have a way of comparing function across many species which can help provide scientific evidence for

certain CR functional explanations over others and can possibly lead to better explanations for how it arose and how it might be treated.

Another article suggests that characterizing delusions as false beliefs produced by incorrect inference about external reality as the DSM does is problematic because of the difficulty in using this explanation at the level of neurobiology and neuroanatomy. A neurocomputational theory is suggested using evolutionary considerations focused on the role of the prefrontal cortex in regulating offline cognition. This represents a "bottom-up" account which attempts to combine neural network theory of the role of dopamine in delusion with the insights of the more cognitive accounts. It is suggested in the article that this approach should be seen as a general model for an evolutionary psychiatry which aims at avoiding problems inherited from evolutionary psychology (more specifically NEP) (Gerrans, 2007). This explanation recognizes the importance of cognitive and neurobiological levels of analysis and we can examine the two conceptions of function and evaluate them both in terms of scientific validity and values.

Jaak Panksepp argues for the need to link basic emotional processes that are typically impaired in psychiatric disorders to neuroanatomical, neurochemical, neurophysiology, and molecular genetic levels of analysis. This is to be done through the use of animal behavioral-genetic and gene expression, microarray analyses. This method can offer what Panksepp describes as a more "coherent" infrastructure for a psychiatric taxonomy than NEP can offer. Further work is also suggested in terms of neurobiological dimensions of psychiatric disorders focusing on "psychosocial and environmental stress vectors that converge to create imbalanced emotional and motivational brain activities of psychiatric significance" (Panksepp, 2006, p. 774).

These examples all show the type of evolutionary psychiatry research that would take place with a DEP CR functional account of psychiatry. They use comparative biology and focus on homology, developmental systems and environmental co construction. They encourage the use of CR function which does not include an intrinsic sense of normal function and assumes that psychiatry arises out of a study of the patterns of similarity and difference.

The power of CR function lies in its flexibility that allows us to compare and contrast something like a CR functional account of homologous structures in the brain with a social CR functional account of the same disorder. It is even possible that we can produce CR functional descriptions that make reference to the biochemical, cognitive and the social level. This is a better CR system simply in virtue of the fact that it is likely to reveal a higher degree of complexity in this system than one that contains only a single level of analysis. Examining the connections between the three levels of analysis in depression might lead us to construct a definition that can refer to all three goals.

The key to the account that I have presented is the ability to bring this kind of scientific evidence together and consider it alongside our values in order to determine our definitions of various mental disorders. As shown earlier Jason Robert has argued that theorists have shown a tendency to overlook patients and clinicians experiences of mental disorders as legitimate factors in psychiatric nosologies. For example when attempting to deal with social stigmatization of mental disorders it may not be useful to talk about homologous regions and regulator genes. This suggests the possibility that we may be better of using different definitions of mental disorders for different purposes.

In the next section I consider this possibility further and examine the goals of psychiatry through Murphy, Wakefield and the DSM and suggest that there are different goals in psychiatry which can cause conflict when attempting to categorize and define mental disorders and I suggest this must be taken into account when defining a concept of mental disorder. I will then provide an argument that we might require different definitions and categorizations for the same mental disorder in psychiatry depending on the psychiatric goal that is being pursued.

4.3.3 Motivations and Goals in Psychiatry

In this section I am going to examine the positions of Murphy, Wakefield and the DSM in an attempt to tease out the different motivations in psychiatry. I will then use these motivations as guidance in my argument for how we should go about defining and categorizing mental disorders using CR function. The different goals of psychiatry are apparent in Murphy, Wakefield and the DSM but in each case there is a different primary focus that is motivating the creation of a psychiatric taxonomy.

Murphy shows interest in evolution because he thinks it helps us distinguish causal factors that the DSM has failed to distinguish. In this process of examining etiology (causal origins) we also discover what behavior can be classified as a mental disorder and what behavior should not be. Murphy suggests we should generate nosology (psychiatric classification) according to etiology and therefore one motivating force in psychiatry is to classify mental disorders according to causal factors.

Wakefield on the other hand is interested in evolution because it distinguishes what behavior should be classified as a mental disorder and what behavior should not be. A nosology needs to capture the concept of a mental disorder and we can tell if a

behavior counts as a mental disorder by examining the etiology of a potential mental disorder. Therefore another motivating force in psychiatry is distinguishing between people who have mental disorder and people who do not.

Finally the DSM has several motivations for a taxonomy:

> The purpose of DSM-IV is to provide clear descriptions of diagnostic categories in order to enable clinicians and investigators to diagnose, communicate about, study, and treat people with various mental disorders (American Psychiatric Association, 2000, Introduction, p. 13)

The DSM seems to have a hierarchy of motivations. There is the aim to diagnose so that mental disorders can be talked about and studied and then ultimately treated. The concerns of Murphy's etiology are not addressed nor are Wakefield's concerns about distinguishing mental disorders. Murphy in fact criticizes the DSM because it is lax on etiological concerns while Wakefield criticizes the DSM because it includes mental disorders which Wakefield suggests should not be included. The introduction of the DSM clearly states that a taxonomy based on etiology would be ideal this is not a realistic expectation. If we can't be sure of etiological concerns then we might end up diagnosing people who don't have a mental disorder according to Wakefield's criteria. The DSM seems concerned with categorizing people in a way that leads to better research and communication that is aimed at treating people.

These three motivational factors each represent a different purpose or goal in psychiatry. Murphy's motivational focuses is on taxonomizing by etiology in order to determine how psychiatric disorders arose. Wakefield's motivational focus is on determining the difference between someone who has a mental disorder and someone who does not with an implicit goal of determining appropriate assignment of responsibility and accommodation possibly including who should be treated. DSM is

motivated to taxonomize according to treatment outcomes in order to achieve the goal of changing or curing someone who has a psychiatric disorder.

Ideally Murphy's taxonomy should map onto Wakefield's taxonomy and both of these should map onto the DSM taxonomy. This taxonomy should help divide up people into treatment categories where people in category A are more likely to respond to a treatment than people in category B. Ideally the DSM, Murphy and Wakefield want a single taxonomy that is based on the etiology of mental disorders, uses a common concept of mental disorder to distinguish normal behavior and also helps to guide treatment by showing that people in the same categories respond to treatment in a similar fashion.

Wakefield and Murphy both suggested that evolution could help with this ultimate ideal goal. In the next section I will suggest that this goal is indeed unrealistic because it requires mental disorders to be natural kinds. Wakefield and Murphy and most of evolutionary psychiatry seem to be hoping that we can find SE functional modules that are natural kinds and therefore common deviations from the normal functioning of these modules will be natural kinds as well. In the next section I will argue that we should not expect mental disorders to be natural kinds or at least not traditional natural kinds. I will instead argue that mental disorders are at best non-traditional natural kinds and I will show how a DEP CR functional account can accommodate this.

4.3.4 Natural Kinds in Psychiatry

Traditional natural kinds are taken to be real categories in nature and biologists often use these categories to predict and explain biological science (Ereshefsky, 2009b). Only members of a kind share a common essence and this common essence is shared by all

individuals in the kind. This essence is constitutive of a set of properties or a single property that all individuals in the kind must have and the essence of the kind is causally responsible for additional properties associated with that kind. For example Gold's essence is a certain atomic structure shared only by other samples of gold and this essence causes the other contingent properties we associate with gold. With this knowledge we can predict that a certain piece of gold will conduct electricity because we can explain why it is that gold is conductive in the first place (Ereshefsky, 2009b).

NEP holds that we share a collection of universal modules and therefore we share a human nature. When these modules become dysfunctional we should respond the same way to the same type of dysfunction. Of course NEP notes that differences in behavior can also be due to variations in the input to the different universal "programs" so there may be some variation but for the most part common modular dysfunctions will give rise to common dysfunctional behaviors which we can diagnose as a mental disorder. This categorizes abnormal behavior according to etiology that explains the behavior and that can then predict various properties of the behavior related to appropriate assignment of responsibility and successive treatment regimes.

However DEP holds that "human nature" is a problematic term and Paul Griffiths suggests that whatever is meant by "human nature" is better captured by a study of patterns of similarity and diversity. We have common developmental modules for example but how development plays out is dependent on a bevy of individual environmental factors that we encounter. Our homologous structures and behaviors have significant similarities with other mammals and yet our neocortex is different than other

animals and its plastic nature means that our behaviors are also going to change and develop over the course of a lifetime.

If we don't have a universal human nature then the NEP SE functional modules cannot be natural kinds and therefore their dysfunctions cannot exist as natural kinds either. If we do not expect dysfunctions to occur in a universal pattern then we should not expect psychiatric disorders to be natural kinds. They ways in which we function are diverse and so the ways we dysfunction are going to also be diverse but that is not to say that there are not significant patterns to these dysfunctions that are worth investigating. We simply require a different understanding of natural kinds that can operate with CR function and DEP. I will use Marc Ereshefsky's argument that a non-traditional conception of natural kinds is required to deal with the concept of "species" as an analogy to dealing with the term "mental disorder". This argument also has some similarity to his argument for elimination of the terms "health" and "disease".

Ereshefsky describes three major approaches to species classification offered by biologists. First there is the biological species concept where reproductive isolation separates species from one another. A species is then defined as the most extensive interbreeding group that exists (Ereshefsky, 1992, p. 672). Others believe in the environmental approach and argue that each species occupies a different area or niche and has a different set of evolutionary forces working on them. Finally in the phylogenetic approach species are categorized according to descent. Species groups contain only the descendents of a common ancestor (Ereshefsky, 1992, p. 673).

Despite the differences the three major species concepts share an assumption that species are lineages, a descendent-ancestor sequence or group of sequences sharing a

common origin (Ereshefsky, 1992, p. 674). They are incompatible because different concepts can classify the same organism into different lineages either by organisms being grouped into two different lineages where one lineage is completed contained within another (Ereshefsky, 1992, p. 675) or alternatively by an organism belonging to two separate lineages (Ereshefsky, 1992, p. 676). Each lineage presents different explanatory interests and predictive powers and we lack a fourth parameter that would encompass all of the other three as each lineage type generally does not correlate with the other types (Ereshefsky, 1992, p. 678). Therefore we have more than one legitimate ontological species category (Ereshefsky, 1992, p. 679). Ereshefsky concludes that the term "species" has no meaning and should be eliminated and replaced by three distinct species concepts: "biospecies" for lineages picked out by the interbreeding species concept, "ecospecies" for lineages picked out by the ecological approach and "phylospecies" for lineages picked out by the phylogenetic approach (Ereshefsky, 1992, p. 680).

Analogously there are at least three different criteria by which a psychiatric taxonomy might be created. We are interested in grouping people in terms of how disorders arose, appropriate assignment of responsibility and accommodation and in effectiveness of treatment regimes. Murphy and Wakefield both suggest the "intentions of nature" might be a natural kind essence that could serve as a fourth parameter to taxonomize psychiatric disorders for all these purposes. However the "intentions of nature" idea is irredeemably confused and lacking a fourth parameter I suggest that we need to consider multiple taxonomies to account for multiple incompatible causal factors in psychiatry analogously to how Ereshefsky required multiple taxonomies for the

different causal factors present in the different conceptions of "species". Different causal factors are being taxonomized according to the different goals in psychiatry.

For example we might discover a clear etiology for both depression and OCD and decide to group people based on the factors that caused their mental disorder. We tend to assume that there should be a direct relationship between the causal origins of a mental disorder and how we should accommodate and treat that mental disorder. People with depression should be accommodated and treated in a similar way to other people with depression and likewise for OCD. However it may be the case that treatment success cannot be predicted based on the same causal factors that allowed us to establish what caused the mental disorder in the first place.

It is difficult to imagine this because we are so used to understanding "depression" as a category which presents an etiology which gives rise to other features such as the need for a certain type of accommodation and the need for a certain type of treatment. I suggest that it is worthwhile questioning this assumption and exploring the possibility of a pluralistic taxonomy that attempts to isolate causal factors for the three separate criteria I have discussed. I will now suggest how this might work using CR function and also describe how values are incorporated.

4.4 DEP as a Foundation for Evolutionary Psychiatry

4.4.1 Psychiatric Goals and CR Function

I previously described how comparing different types of analyses of different systems and goals is necessary to assign a CR dysfunction as a mental disorder. I will now explore the possibility that certain analyses at different levels might be better than analyses at other levels for specific purposes in psychiatry. I will also entertain the

possibility that even the same type of analysis (e.g. a biochemical one) might yield a different taxonomic distribution when directed toward a different purpose in psychiatry.

Perhaps when examining social anxiety disorder (SAD) we do a CR analysis with an etiological purpose at the level of the biochemical defining the system of interest to be the brain and the goal of the system to be keeping a certain chemical above a certain level. We might be satisfied that the disorder arises because of a chemical imbalance and therefore we have established a category of people who we label as having social anxiety disorder. However when we do a CR functional analysis that aims to investigate questions of responsibility and accommodation we find that half of the people who we have labeled as having "social anxiety disorder" respond to accommodating techniques and half do not. Imagine then that we have another group who through a biochemical analysis are grouped together as having OCD but when we do an analysis with the aim of accommodating these people using the same techniques that were tried with the social anxiety group. We again discover that half of the OCD group responds to these techniques and half don't. Traditionally we respond to this by saying that some people with social anxiety disorder respond to the same accommodation techniques as some people with OCD. Instead I am suggesting that we use a different taxonomy for the purposes of accommodating people than we did for determining the purpose of etiology because it better tracks that causal factors involved in each purpose.

What this suggests is that why the disease arose might have no bearing (or little bearing) on how we might accommodate it. This rejects the traditional natural kind's picture that suggests that we can find some kind of essence which can then predict other contingent properties. Perhaps the accommodation techniques involve providing anyone

with an anxiety disorder with first choice for seating when out at a theatre, restaurant or public gathering. This may be analogous to providing first choice in seating to people who have poor eyesight or poor hearing. People with SAD may benefit by choosing certain locations which are out of the way where they are less likely to be the center of attention. This accommodation technique does not depend on whether or not the disorder arose because of a low level of chemical A (the Social anxiety disorder chemical) or a low level of chemical B (the OCD chemical). However it may still be that in each case half of the people with social anxiety disorder and half of the people with OCD are able to maintain their respective chemical level that is required to avoid the anxiety. The accommodation appears to have the same affect on each chemical but in each case it only helps half the people suggesting that there are other causal factors at work that overdetermine the role of the chemical involved.

We might discover that in terms of treatment 75 % of people with OCD respond to drug X while 25 % of people with SAD respond to it. Alternatively 25 % of people with OCD respond to drug Y while 50 % of people with SAD do. This drug may alter a different chemical than either of the chemicals we identify as the "cause" of either disease but it may be involved in a chemical pathway that interacts with these chemicals at some point.

In order to investigate these patterns I suggest that we use a different taxonomy for each purpose. A CR functional analysis can group together people based on etiology in terms of chemical differences and one group is labeled as having social anxiety disorder and one group as having OCD. A second CR analysis aimed at accommodation can merge the 50 % of OCD patients and the 50 % of SAD patients who experience

lessened anxiety when accommodated by first choice in seating. Finally two categories can be created based on those who experience relief from each of the respective medications available. These three taxonomies are incompatible with each other as the categories are not mutually exclusive but the taxonomies represent incompatible causal differences just like a category of "biospecies" recognizes causal factors that a category of "phylospecies" fails to recognize.

Using these different taxonomies can help us find out information about the various CR dysfunctions we are experiencing. For example we might want to discover what allows some people to experience relief from being accommodated by being given first choice in seating. It may be that for some people as long as they are not sitting next to a stranger they do not experience the negative cognitions that lead to an increased anxiety level however for others this is not a factor in whether or not they experience anxiety. This division might not occur down the lines of biochemical divides. Therefore doing CR functional analyses at multiple levels can help to show how various causal factors come apart depending on the psychiatric goal that we are aiming at. All of this information provides with a better understanding of all three psychiatric goals. While it would be ideal if there were a natural kind essence that we could use to predict other properties biology suggests that this is not the case. Therefore we must use a plurality of taxonomies with different types and levels of analyses that focus on different systems with different goals if we are to be successful at gaining a full understanding of mental disorders.

By focusing on separate goals we keep already complicated causal concerns separate. This makes it easier to incorporate values when only one goal is put forth. For

Wakefield accommodation, responsibility and who should be treated end up being dictated by whether or not people have an objective mental disorder. However by focusing on multiple taxonomies the question of who can be treated and who deserves to be treated come apart. Of course it may be morally relevant to know whether or not people can be treated in discussions of who deserves to be treated. The same might be true of accommodation and responsibility. Scientific evidence about whether or not people can be accommodated and what reasons we have to believe people should or should not be held responsible for their behavior are important when determining if people should be accommodated or held responsible for their behavior. The more we separate out causal factors the more specific information we can obtain about mental disorders and the more apparent our values become about specific situations.

Now that I have provided an alternative to an NEP SE functional account of evolutionary psychiatry I can show how the alternative DEP CR functional account of evolutionary psychiatry can help to solve the problems Murphy's taxonomy faced in chapter two.

4.4.2 DEP and OCD

In chapter two I showed that Murphy's taxonomy was unable to account for some evolutionary explanations such as those for OCD and that these explanations were likely to provide the causal explanations Murphy required while the NEP influenced evolutionary explanations that did fit into his taxonomy were likely to be based on inadequate biology and unable to provide complete causal explanations. I will now explain how using DEP as a foundation we can understand a wider variety of

evolutionary explanations and also explain a wider variety of causal factors involved in those explanations.

One evolution explanation we examined in chapter two involved understanding OCD as a "dysregulation of evolutionarily conserved behaviors and mental states critical to human survival" (Feygin, Swain, & Leckman, 2006, p. 855). Because this explanation utilizes conserved behaviors and mental states we cannot understand this explanation in terms of adaptations that arose in the Pleistocene. First of all the conserved behaviors and mechanisms under discussion are homologous and require a CR functional analysis and not a SE functional analysis that focuses on adaptations. Second the comparative animal research requires studying multiple time periods most of which predate the Pleistocene. These concepts are foreign to the NEP version of evolutionary psychiatry I discussed in chapter 2 but of course they are exactly the concepts and techniques that DEP utilizes.

DEP can also study the "dysregulation" which involves an examination of the relationship between environmental factors and the organism itself. SE functional modules either function "as nature intended" or not. The regulation system is unlikely to be "dysfunctional" because we saw that there is a range of behavior in OCD that seems to be on a continuum with normal behavior. Therefore people with OCD are not dysfunctional in terms of the regulation of threat detection but instead are simply functioning at what Amundson would a different level of performance.

Without an objective way of determining the category of OCD we need to identify precisely the goals of the system that cannot be met by people with OCD. The DSM states that:

> The essential features of Obsessive-Compulsive Disorder are recurrent obsessions or compulsions that are severe enough to be time consuming (i.e., they take more than 1 hour a day) or cause marked distress or significant impairment (American Psychiatric Association, 2000, Introduction, p. 1)

We can therefore construct a CR analysis that describes the goals of a system to be the functional ability to perform various tasks without experiencing obsessions or performing compulsions longer than 1 hour a day and without feeling distress. When obsessions or compulsions occur for lengthy periods of time or if they occur with distress then we recognize a CR dysfunction has occurred that we can now call a mental disorder. We are dysfunctional with respect to a specific contextual goal and not with respect to some adaptive mechanism.

Of course evolutionary analysis can help our understanding by using a CR functional ascription of the conserved mechanism that is thought to underlie this condition. This can provide a different level of analysis that is possibly genetic, biochemical or at the level of developmental pathways. We can then compare the previous CR analysis which was at the cognitive level examining thoughts and emotions with this one to see how these conserved mechanisms might be related to our cognitive level goals of avoiding an overactive threat detection mechanism. We can then introduce questions of psychiatric goals and see if these analyses work better in terms of one goal rather than other. We can also look at treatment results in OCD and explore the possibility of a need for a pluralistic taxonomy.

These CR functional ascriptions abandon the search for explanations that fit a breakdown or a mismatch category but the insight of the breakdown category can be preserved by letting go of NEP assumptions and recognizing the idea that internal change in the brain over evolutionary time can change our behavior. Alternatively the mismatch

category lends us the insight that the environment that we live in today is likely different from the one we lived in during the evolutionary past and therefore the functionality of traits might be different today depending on how they react to the modern environment which includes the potential for new variants derived from plasticity. Murphy's taxonomy assumed that internal functional change and external environmental change could be studied separately but both operate interdependently and this insight must be understood in order to account for the dysregulation evolutionary explanation for OCD.

The second OCD explanation is the group selection model explanation (Polimeni, Reiss, & Sareen, 2005). It suggested that OCD evolved to increase group fitness by increasing the safety of a group that had a person or people dedicated to concerns about safety. Murphy's taxonomy had difficulty with this explanation because in terms of the individual it appeared to be a mismatch explanation but in terms of the group it might still be an adaptive mechanism. In other words a potential function at one level causes a dysfunction at another level and determining the "intentions of nature" is extraordinarily complicated. On the other hand I have provided an array of examples to show that CR function can make sense of comparing multiple goals at different levels of analysis. Nothing is strange about this analysis for DEP because it recognizes that the process of evolution is working on multiple different trajectories at once.

We can take from this explanation the insight of the persistence category that even some traits that have adaptive value can be described in terms of a CR functional analysis that chooses goals so that the behavior in question is dysfunctional. Murphy's example of anti-social personality disorder showed that in order to gain money, sex and power these people can be deceitful and manipulative. In terms of an evolutionary

strategy to gain sex and power this is functional but this behavior becomes dysfunctional if the goals of a system we are analyzing are centered around living a well adjusted life and respecting the feelings of others in society. Therefore even adaptive evolutionary strategies can be considered as dysfunctions in the context of a different level of analysis and a different system with different goals.

The third OCD explanation suggested that OCD was as an extreme on a continuum of evolved harm-avoidance strategies where OCD is a costly by-product of the adaptive ability to anticipate needs or threats in the future (Brüne, 2006, p. 317). This example suggests that OCD is a result of the process of evolution and it is difficult to fit into any of Murphy's categories. Like the dysregulation example it suggests that OCD is continuous with normal behavior but simply represents extremes on a spectrum. The dysregulation example suggests that the extremes of behavior are in terms of conserved mechanisms for threat detection that are dysregulated while this explanation suggests that the imagined consequences of our own thoughts and actions (meta representations) are exaggerated and thus pathological. In both cases it is difficult to tell where "normal function" ends and pathological activity begins without the use of values. This of course is a problem for Murphy's taxonomy but not for my account of DEP CR function that explicitly uses values and science together.

Not only can DEP CR function account for each of these explanations individually but by comparing these three explanations we can also recognize some themes. First of all two of these explanations suggests that OCD behavior is continuous with behavior in the general population and yet the behavior also seems extremely bizarre and difficult to understand for people who don't have OCD. Because there is no natural

kind distinction to be made between the behaviors we define our own goals and we decide using both values and science where it becomes acceptable to say that the behavior has crossed a point to where it is pathological. Second of all we recognize that OCD is described as a dysfunction but what we actually get are extra functions or excess function. Even though the regulation mechanism is functioning at a lower level we end up with certain behavior happening at an increased level whether we describe it in terms of cognitions or conserved behavioral mechanisms.

We can study these themes because we have the different levels of explanation to compare and this information can help us to determine which CR analyses are good and which should be abandoned in light of both the facts and the values that are elucidated by these comparisons. Therefore a DEP based Evolutionary psychiatry can not only account for these OCD explanations and the disorders they represent but it can also combine them in such a way as to produce an extensive set of causal hypotheses about the evolution of OCD which can help to provide more complete causal information about OCD itself. Therefore a DEP based evolutionary psychiatry better achieves the psychiatric goals that Murphy hoped evolution could assist with. I will now consider some possible objections to the view I have presented.

4.4.3 Objections

Murphy argues that according to Wakefield's account vestigial organs cannot be diseased because they have no SE function. Wakefield responds that this is the wrong level to look for dysfunction. Inflammation in the appendix is a dysfunction at the level of the tissues and not the level of the organ (Wakefield, 2000, p. 256). In other words the tissues have an SE function which can be analyzed. He suggests that an infection in the

nose that does not inhibit the nose's functions of breathing and smelling is still dysfunctional just not at the level of the nose. Therefore this suggests that any lower level dysfunction means that a higher level change of function will "imply dysfunction". Therefore Wakefield might accuse me of failing to respect the various levels that SE functional analyses can occur at.

For example some of the OCD examples may simply represent a problem in SE functions older than the Pleistocene. A dysregulation is a SE dysfunction at the level of the regulation of an ancient conserved mechanism. Looking for SE functions at different levels of organizations will not help Wakefield though. He still wants to use the "intentions of nature" idea to compare the functionality of traits today to their functionality when they arose. Since this trait is highly conserved in most mammals is likely arose in a common ancestor of all mammals in the distant evolutionary past in a potentially radically different environment long before humans even existed. The traits function has been changed and altered in each mammalian species over the vast evolutionary time since it arose. Of course we can compare this traits functionality in terms of the commonalities of the conserved mechanisms but of course this is comparing homologous functions and not adaptive functions.

In terms of adaptive functions the trait is functioning in distinctly different environments in distinctly different species now as compared to when it first arose. Without doing the homologous research we have no grounds on which to consider adaptive questions and propose a reasonable SE functional ascription. Therefore I agree with Wakefield's move to examine traits at lower levels of organization unfortunately this does not help an SE functional account of evolutionary psychiatry it in fact only

further supports the need for a CR functional account that can use homologies to compare conserved mechanisms. This is especially true considering how easily traits can acquire new adaptive SE functions while homologous functions often maintain homology at one level while functional changes occur at a different level of homology.

The second potential objection is similar to Wakefield's objection to McNally that a CR analysis will simply yield the failure of various causal chains to bring about desired outcomes without distinguishing between which failures are pathological and which are not. For example negative side effects of normal functioning such as pain in childbirth, normal variation valued negatively such as shortness, as well as socially negative conditions such as illiteracy will be classified as disorders because there is "the failure of certain possible causal processes that would prevent them" (Wakefield, 2001, p. 362). Wakefield suggests that a CR functional psychiatry would study all effects of internal mechanisms that are of interest to humans without discriminating between medical and non-medical domains as they currently operate as medically relevant effects are distinguished with an implicit appeal to questions of what the organism is designed to do (Wakefield, 2001, p. 363).

This criticism that is directed at McNally would no doubt apply to my DEP CR functional account of mental disorders as I am replacing Wakefield's "objective" conception of a dysfunction in the concept of mental disorders with a plurality of concepts of mental disorders. For Wakefield I therefore have no objective component in my account of mental disorders and so I cannot provide a way to distinguish them from normal behavior. This however assumes that Wakefield's concept of dysfunction in his concept of mental disorder is value free.

Wakefield argues that the concept of a mental disorder requires that only an objective SE "dysfunction" can be called a mental disorder. Even if we assume that we can objectively determine SE functions and dysfunctions the claim that only these "dysfunctions" can be legitimate mental disorders is explicitly normative. If everyone holds this concept of a mental disorder it simply means that everyone holds the same normative judgment. Therefore the universal aspect of mental disorder derives from a shared concept and not from inclusion in a natural kind category such as the category of "dysfunctions". Therefore Wakefield's concept of a mental disorder is explicitly normative and different values would construct a different concept of a mental disorder.

Wakefield might argue that "dysfunction" must be part of any concept of mental disorder and since "dysfunction" can only mean an SE dysfunction therefore any concept of mental disorder must at least minimally conform to an SE dysfunction regardless of values. Wakefield would then be treating "dysfunction" as a natural kind term in the same way that Saul Kripke suggests that "gold" is used by a community of speakers to refer to a certain kind of thing. There is a relationship between the speakers and this kind of thing with identifying marks we can use to pick this kind of thing out (Kripke, 1980, p. 118). For example the essence of gold is having an atomic number of 79 (Kripke, 1980, p. 123). Kripke suggests that at some point in the past we designated this kind of thing as "gold" and each time that name is used it links us with that name and carries it forward from person to person into the future creating a "rigid designator" (Kripke, 1980, p. 91-97). If something has identifying marks of gold but does not have atomic number 79 then it is a case of counterfeit gold (Kripke, 1980, p. 124 - 125).

Kripke suggests that people can therefore be referring to gold even if they don't know all of the properties associated with gold or even if they describe gold by a different name. Analogously Wakefield seems to be arguing that even though it appears that people might have different concepts of "dysfunction" they are all appealing to the same natural kind term what I have been calling SE dysfunctions or the failure of an organism to do what it was designed to do. There are several problems with this.

First of all even if we assume for the moment that everyone is referring to historical design when discussing functions it is not clear that SE dysfunctions are even the best way to determine what the organism "was designed to do". The variable nature of SE function and dysfunction make it difficult to compare the "intentions of nature" from one period to another. If we were committed to a concept of dysfunction that studied what the organism was designed to do we would likely be better off studying homologies which could provide us with categories that might be closer to be "natural kinds" in the sense that we are much more likely to be able to identify a "mammalian nature" than a "human nature". Many basic systems were "designed" millions of years ago serving different purposes than they now do. Many modern functions might have to be considered SE dysfunctions in this light. Wakefield's concept actually seems to be referring to dysfunction as "a trait failing to function as it did during the Pleistocene" which has little to do with the historical origin of the trait which typically originate before the human species even existed.

Of course it's not clear that people are even thinking of natural kinds when using the term "dysfunction" even if they are using it to refer to evolutionary design failure. Instead there could simply be a general appeal to the evolutionary process being able to

explain why modern traits function as they do now. Wakefield simply ignores competing definitions of "dysfunction" and it is likely to make a difference to how people use the term if all concepts of dysfunctions were available and their normative components emphasized. Therefore "dysfunction" cannot work like "gold" does for Kripke as Wakefield assumes that when anyone uses the term "dysfunction" they really mean "SE dysfunction".

Of course Wakefield is likely correct that our concept of mental disorder refers to a "dysfunction" but of course the I have shown that there are many people who disagree with a concept of dysfunction that relies upon a conception of "normal function". So the general concept of dysfunction is much broader than Wakefield allows. Murphy in fact argues that by-products or "spandrels" provide a problem for Wakefield's harmful dysfunction analysis because they can become dysfunctional. Wakefield responds that "In fact, failed spandrels in and of themselves, when they do not imply any failure of designed function, do not imply disorder" (Wakefield, 2000, p. 255). Reading is a spandrel but being unable to read is only a disorder if an internal SE function has failed. Murphy has a different conception of "dysfunction" than Wakefield has but of course we already know that Murphy has a different understanding of mental disorder so this is not surprising. For Murphy "dysfunction" is not synonymous with SE dysfunction as mismatch explanations can be mental disorders without SE dysfunctions.

However the OCD examples suggest an even wider divergent concept of "dysfunction" as differences in the magnitude of the behavior was classified as a difference in kind. Behavior below a certain threshold is normal but above a certain threshold is a mental disorder. The point at which behavior on that continuum becomes

pathological depends on the goals of the system which of course can depend on values. This represents a radical departure from Wakefield's concept of a "dysfunction" that leads to a mental disorder. Therefore it is not clear that Wakefield's concept of "dysfunction" is objective and even if it is an objective natural kind limiting mental disorders to only consist of these "dysfunctions" is an explicitly normative claim which does not appear to be universally held.

So in what way is CR dysfunction not objective? CR dysfunctional analysis of course does not offer a way of objectively distinguishing mental disorders from normal behavior but I have argued that Ereshefsky and Wakefield fail to do so either. A CR dysfunctional analysis contains normative aspects and objective aspects like other theories of mental disorders but nothing is excluded *a priori* from being a mental disorder as there is with Wakefield's conception of mental disorders as this exclusion is normative. Ereshefsky's state descriptions are also normative in what they include and what they might obscure. CR dysfunctional analysis also at least makes it clear where the values are coming into play. Wakefield appeals to the objectivity of science when he is using SE dysfunction but this simultaneously prevents him from exploring other important avenues of research such as homologies.

Objectively CR functional analyses must still be accurate. If we analyze the goals of a system and suggest that the constituent parts function in order to achieve those goals then there is an objective way to determine whether or not this is true. Using CR dysfunctions also permits us the flexibility to use the widest variety of scientific resources available to help us achieve the widest range of psychiatric goals. Simultaneously it exposes values to the highest degree and forces them to be as explicitly

stated as possible. It makes clear those distinctions between normal functioning and mental disorders are combinations of facts and values from the beginning instead of attempting to present a value laden definition as objective. Like Ereshefsky's state description account my account does not attempt to be objective the way Wakefield does because my account assumes this is not possible. Instead it focuses on making scientific facts and value judgments as explicit as possible as Ereshefsky suggests. I have argued that a DEP CR account is the best theory for accomplishing these goals.

As far as the claim that my account does not capture medical distinctions, it captures the clearly stated need that psychiatry has for a variety of concepts of mental disorders:

> Moreover, although this manual provides a classification of mental disorders, it must be admitted that no definition adequately specifies precise boundaries for the concept of "mental disorder." The concept of mental disorder, like many other concepts in medicine and science, lacks a consistent operational definition that covers all situations. All medical conditions are defined on various levels of abstraction for example, structural pathology (e.g., ulcerative colitis), symptom presentation (e.g., migraine), deviance from a physiological norm (e.g., hypertension), and etiology (e.g., pneumococcal pneumonia). Mental disorders have also been defined by a variety of concepts (e.g., distress, dysfunction, dyscontrol, disadvantage, disability, inflexibility, irrationality, syndromal pattern, etiology, and statistical deviation). Each is a useful indicator for a mental disorder, but none is equivalent to the concept, and different situations call for different definitions (American Psychiatric Association, 2000, Introduction, p. 8)

Wakefield claims that CR fails to capture a medical distinction but he does not agree with the DSM which is arguably a prime source for understanding how medical concepts are actually used by the practitioners Wakefield often appeals to.

Therefore I suggest that science and values together determine what a mental disorder is based on psychiatric goals using whatever concept of mental disorder is appropriate. This is no less objective than Wakefield's account and in fact makes the

values in play more explicit than he does while simultaneously increasing the scientific tools available for achieving psychiatric goals while also accounting for the DSM's need for multiple concepts of mental disorders.

The third objection I will consider is that my account of evolutionary psychiatry contains too many categories and the variables involved are too complex to form a coherent taxonomy for psychiatry. I will reply to this potential critique by arguing that first the complexity is required both by the realities of nature and the various goals of psychiatry and second that while some cases may become very complex some, possibly even the majority of cases will be much simpler.

In the previous section I quoted from the DSM to show that they acknowledge the need for multiple definitions or concepts of mental disorders. I have also suggested that we require multiple definitions of specific mental disorders themselves in order to capture the causal factors involved in different psychiatric goals as well as causal factors that are captured with different levels of analysis. The reason for this pluralism is partially empirical as we may not be able to locate the common causal factors we are interested in but it can also be ontological. We should not expect mental disorders to be natural kinds or if we do they are more likely to be non-traditional natural kinds that require a taxonomic pluralism that might divide the same mental disorder into incompatible categories. Therefore the pluralism is supported by pragmatic aims that best support the goals of psychiatry as well as a foundational assumption that mental disorders and the dysfunctions they describe are not traditional natural kinds like our conception of "gold".

Beyond the DSM others have recognized the need for a plurality of categories of mental disorders:

> The primary claim of this paper, then, is that psychiatric diagnoses differ in the sorts of categories that best capture them. Several more or less distinct kinds of category are evident in the psychiatric domain, and theoretical approaches that privilege one kind may correctly represent some disorders but misrepresent others. To generalize an approach beyond its sphere of appropriateness may mislead us about how some disorders are best described, classified, explained, assessed, and theorized. Conversely, determining which of a plurality of models is appropriate for a particular condition may improve our chances of apprehending it properly. I argue that making determinations of this sort is important, and is at least in part an empirical matter (Haslam, 2002, p. 204)

Nick Haslam describes five structures that he suggests compose a conceptual taxonomy of psychiatric kinds. The five structures form an ordered set and the categories meeting more requirements are progressively more defined by internal structural criteria and less by artifactual or pragmatic considerations. Haslam's five "structures" or types of mental disorders is provisional and open to modification by other theorists (Haslam, 2002, p. 204). This makes it ideal for integration with my account of evolutionary psychiatry.

Haslam's first category is called "non-kinds" which focus on a dimensional continuum where the placing of a binary distinction is arbitrary and the discontinuity is artifactual (Haslam, 2002, p. 204). The second category is called "practical kinds" and once again there is no underlying discontinuity but in this category practical considerations suggest a certain placement of boundaries. For example certain boundaries may better reflect those who require treatment. The distinction between non-kinds and practical kinds may be open to debate and depend on the degree of practical benefit that might be perceived in identifying the need for clinical attention without pathologizing normal behavior (Haslam, 2002, p. 206). The third category is fuzzy kinds featuring objective discontinuity that does not rely on essentialism or dichotomies. Instead of cutting nature at its "joints" this represents a bend which is not arbitrary but

also not a sharp distinction and therefore this category might have three classes: unambiguous category members, unambiguous nonmembers and intermediate ambiguous members (Haslam, 2002, p. 208).

The fourth category is that of discrete kinds which represent an objectively discoverable dichotomy that is not based on discovering essential properties. These might be caused by nonlinear interactions of multiple factors producing the same variant or a continuum where individuals exceed a threshold might yield discrete kinds as well and both of these require multifactorial accounts of etiology and not essentialist identification (Haslam, 2002, p. 210). Finally the fifth category is natural kinds that as we have seen must share a single, invariable feature in common, and this shared feature gives rise to direct and necessary observable characteristics. Haslam discusses the possibility of natural kinds in psychiatry but points out the intense criticism of the idea and the criticisms in general of natural kinds in biology (Haslam, 2002, p. 213).

Haslam echoes many of the reasons I have provided for multiple concepts of mental disorders. Even some of Haslam's categories may be too strict though as the category of practical kinds and fuzzy kinds may not always be discrete as Haslam himself hints towards. I agree that often there are often practical reasons when choosing where to place disunity on a continuum but there might also be pragmatic reasons (related to values of course) which are operating *at the same time*. Therefore we might suggest that the reasons we choose to place the threshold at one particular point on the continuum requires both science and value judgments. If there is one disagreement I have with Haslam it is the common theme that science and values can come apart when defining mental disorders or even kinds of mental disorders. Haslam thinks that we can grade

different concepts of mental disorders according to the degree of objectivity but values and objective principles do not come apart quite this easily.

What is important is that there is support for Mental Disorders to vary depending on the psychiatric purpose, the level and type of analysis, the system and goals that we choose and even the individual Mental Disorder itself. The complexity is required by the goals of psychiatry and the realities of human behavior that do not easily break down into natural kinds. Therefore it appears that we need all of this complexity in order to better define mental disorders and achieve the goals of psychiatry.

The second response to the complexity criticism is that simply because we have an enormous array of conceptual tools this doesn't mean they will be required every time we want to define a mental disorder. Ereshefsky suggests that it is only the controversial cases that we need to eliminate the terms "health" and "disease". Likewise we may find that some mental disorders require only a single taxonomy. We identify it according to a collection of symptoms; discover an etiology which gives rise to appropriate guidelines for accommodation and treatment that make sense to our intuitions and values. It is also likely that many CR functions will map onto SE functions of some kind and we will often be analyzing things according to what they were designed to do in the adaptationist sense. What is important is that a DEP based CR functional account would make sure that when we are doing this it is appropriate and discover the controversial cases and deal with them according to the vast array of conceptual and scientific machinery available.

Wakefield and Murphy both suggest that the DSM would be much better off if it would base things causally using a SE dysfunctional natural kind account. However as the DSM is currently set up it makes much more sense and is much better theoretically

understood using CR functions. The DSM recognizes that we require different kinds of mental disorders in psychiatry as does a DEP CR functional analysis.

This can also help explain why the DSM needs to diagnose people according to symptoms and not causal factors. There can be different causal factors at play depending on which goal of psychiatry is being pursued not to mention differences in level of analysis, goals of the system and so forth. Therefore we can understand the DSM as an amalgamation of purposes although it does privileging treatment it must be able to respond to the needs of researchers and even the law courts attempting to determine issues of responsibility and accommodation. The DSM categorizes people according to symptoms which can then be taxonomized into smaller categories depending on the variety of factors I have discussed.

Therefore we can still have people identify with themselves based on symptoms while still acknowledging that there are other causal factors and purposes that might be needed that group these people differently. Although there are different conceptions of species we still retain a general concept of "species" and Ereshefsky suggests that we should do this for health too. Locally there can be little change for the DSM and the way it is structured allowing things to remain as they are without the need to overhaul the diagnostic category we can still gain valuable insight from a DEP CR functional account.

First the DSM needs to let go of the ideal of natural kinds and treating mental illness as if they are a unity category despite the fact that the DSM clearly states this is not the case. Second the DSM needs to emphasize the importance of incorporating the widest range of values and scientific information as possible into consideration. Third this needs to be done in reference to the goals of the DSM and not an external conception

of mental disorders. The DSM has made efforts at achieving these goals but the criticisms from people like Murphy and Wakefield are not helpful and they offer and unrealistic alternative that ends up being no more objective or simpler than the DSM already is. This alternative is also based on problematic science and a set of implicit hidden values which in fact hinders the goals of psychiatry.

4.5 Conclusion

In this chapter I have argued that the SE conception of function cannot provide the objective causal explanations that both Murphy and Wakefield seek in order to separate different kinds of mental disorders as well as distinguish mental disorders from normal behavior. I have also argued that Wakefield's harmful dysfunctional account does not match many different people's concept of mental disorders. I have examined the literature on the health and illness debate as well as the different motivations among Murphy, Wakefield and the DSM that suggest different goals for psychiatry. I have suggested that psychiatry in general might benefit from a kind of eliminative pluralism with regard to mental health and disease where we focus on defining mental disorders in terms of the purpose of the taxonomy. Different taxonomies can classify the same behavior in different ways depending on the criteria involved. These taxonomies can be incompatible so that a group of people who may be grouped together in terms of etiology may not be grouped together in terms of treatment regime. Therefore we have a conception of health and disease only in terms of specific criteria.

When defining mental disorders within these criteria we must recognize the importance of values. Instead of understanding values as helping us choose from a list of potential mental disorders I have argued that values must be present during the entire

process of defining mental disorders using CR functions. CR functions force us to specify the goals of a system and make salient the values that are involved. We choose the CR systems based on a combination of values and science which are inseparable.

A DEP based psychiatry is highly flexible and I have implicitly suggested that it may be a good general approach to psychiatry. At the same time I have suggested specific evolutionary principles that are already in use in the literature which are compatible with DEP. In these approaches we see homology replacing SE function and we see a growing scientific literature that can help provide scientific grounding to our decisions about appropriate levels of analysis. This includes appropriate understandings of variation, plasticity, developmental modularity as well as the impact of the environment on mental disorders. DEP suggests an abandonment of the old traditional natural kind account of psychiatric disorders. Instead I have presented a pluralistic account that integrates values and science through CR function and a biologically realistic account of evolution.

CHAPTER 5: CONCLUSION

5.1 Introduction

In this chapter I will provide some examples of how a DEP based evolutionary psychiatry might affect psychiatry itself beyond its role as a niche sub discipline. First by developing a stronger account of how we might prevent mental disorders and why this is an important but overlooked aspect of psychiatry. I also suggest that DEP provides the impetus for developing a different understanding of responsibility in psychiatry. Then I suggest that some debates about treatment could benefit greatly from a DEP analysis that could highlight the important connections between both sides. I then summarized the thesis, and discuss some of the themes and make some concluding remarks about the role of evolution in medicine generally.

5.2 A New Evolutionary Psychiatry

5.2.1 Prevention

The first general goal I discussed for psychiatry in the previous chapter was that of discovering etiologies. We have an interest is discovering how mental disorders come into existence. What is interesting is that little attention has been paid to the idea of preventing mental disorders when compared with the attention focused on determining whether or not people with mental disorders can be held responsible for their behavior or whether or not someone can be said to have a mental disorder or even the issue of how to treat people with mental disorders. There might be good reasons for this. Until we are sure what a mental disorder is it might not make sense to try and figure out how to prevent them. We might also argue that there is a general bias in medicine toward palliative or curative medicine compared to preventive medicine. It is also difficult to see how we can prevent the malfunction of a module for NEP.

However, a DEP based psychiatry focuses on the importance that environmental factors have in co-constructing traits. Therefore the environmental inputs are part of what causes mental disorders and can also be part of what can help prevent them. Remembering Amundson's insight about disability we recognize that different social situations can also create mental disorders. If a mental illness is defined as the inability to do x, y and z then the functional goals of that system can be understood to be x, y and z. There might be a social system in place that prevents the achievement of x, y and z. If this is true then the mental disorder only exists when the proper social supports are not in place. Therefore a DEP based CR functional account of psychiatry can help to shift some focus to a potentially overlooked psychiatric goal.

5.3.2 Accommodation and Responsibility

Most discussions of responsibility in psychiatry focus on determining when a person who has a mental disorder should be held responsible for their behavior and what behavior this person should be allowed accommodation for and the manner this accommodation should take. The study of neuroplasticity has facilitated new discoveries about the brain suggesting that a traditional notion of responsibility may be insufficient for psychiatric conceptions of responsibility and blame. We now know that the brain has an enormous capacity to change its structure and function through a lifelong developmental process. Many aspects of someone`s extended environment can contribute to these ongoing changes and one of the most fundamental sources of change involves the behavior of other people and social and political organizations. These findings suggest that there is a different sense of responsibility that must be examined namely who is responsible for the creation, accommodation and treatment of mental disorders if these all depend greatly on social and environmental parameters. DEP has provided us with the impetus to look for a different conception of responsibility.

I suggest that Iris Young's social connection theory may help us envision a new conception of responsibility and accommodation. She argues that "People often stand in dense relationships with others prior to, apart from, or outside political communities" (Young, 2006, p. 105). Young uses an example of a sweatshop worker and suggests that somehow consumers, retailers and sweatshop owners all bear responsibility for how the clothing is produced. Young feels that blame or liability models will not be sufficient to solve these problems and a different notion of responsibility must be used to deal with the harsh conditions of sweatshops (Young, 2006, p. 107). Young argues that "The structure

of the global apparel industry diffuses responsibility for sweatshop conditions" (p. 110). A causal chain exists between the producers of the clothing and the ultimate consumers of that clothing. Sub contractors and companies do what they feel they must at their link in the chain which includes paying employees working at the bottom very little. The workers also suffer domination and coercion. This system is an example of structural injustice.

Responsibility for structural injustice is not directed at individuals because it is not perpetrated by individuals alone. It is a result of social processes that structurally create and manage a system of domination and deprivation. The social relationships that exist mean that some are unable to exercise their capacities at the expense of others having an extended range of ability to exercise their capacities (Young, 2006, p. 114). If injustice is caused by structural means then Young argues everyone who is a part of those structures bears some responsibility to correct those injustices. Traditional injustice is based on liability or blame in a direct relationship between two parties, and while this type of injustice may still occur, Young wishes to develop a new kind of responsibility in which we can account for structural injustice (Young, 2006, p. 117). When it is the structural system itself that is originating the injustice, those who are benefiting from that structure might not be blame worthy in the traditional sense, however, Young argues it is inappropriate to argue that they have nothing to do with the plight of those who suffer in other parts of the chain of actors and thus we need to develop a new kind of responsibility, namely the social connection model.

While Young`s social connection model clearly was intended for a different purpose I suggest that it provides an important alternative account of responsibility for

people with mental disorders as well. DEP suggests that mental disorders can be the result of a confluence of social and environmental influences. They are not limited to a dysfunctional module or piece of the brain. Whether or not social programs are available can determine whether or not people develop mental disorders in the first place. Young argues that the structure of social relationships can lead to important moral claims about responsibility. DEP suggests that mental disorders can also arise due to unjust social structures. Young argues that attempting to determine accommodation and responsibility without considering social structures is unjust. I am suggesting that determining issues of accommodation and responsibility for people with mental disorders must also consider the same issues.

Young's argument is based on a moral claim about the impact that social structures can have on people's lives. In DEP the social environment coupled with neuroplasticity suggests that social structures can influence people so deeply that it can actually change the structure and function of their brains. DEP therefore provides scientific evidence for the claims that Young makes, while the social connection model provides us with the moral machinery to make sense of this type of evidence that challenges traditional moral theories.

My DEP account of psychiatry defines mental disorders using an inseparable relationship between science and values and when determining issues of responsibility and accommodation for mental disorders we require examining the influence that the social environment has on the creation, maintenance and treatment of mental disorders. A theory such as Young`s allows us to recognize Amundson's important concerns about how we define normal and abnormal functioning.

Young's focus on positive responsibility suggests that even those who are in vulnerable situations have responsibilities to have their voices heard and do what they can based on their situation. The concept of responsibility for people with mental disorders usually consists in listing the ways in which they are excused from certain responsibilities or accommodated somehow for atypical behavior. There is rarely a focus on accommodating different levels and modes of functioning and then assigning responsibilities that are appropriate. Young's theory provides psychiatry with a new way of analyzing the issue of responsibility and accommodation for people with mental disorders.

5.2.3 Treatment

I have discussed the fact that depression can be described as a biochemical imbalance in the brain. The biochemical hypothesis finds support in studies of rats in which anti-depressant drugs cure symptoms in rats that are argued to be similar to symptoms of depression found in humans. Similar biochemical pathways and neurological circuits have been found in rats and humans which further supports this research. On the other hand, other theories focus entirely on problematic cognitive processes and there has been great success in utilizing treatments such as cognitive behavioral therapy which concentrates on correcting patient's distorted cognitions. This type of therapy postulates that depression results from making false judgments about the world and about oneself. When these "distorted thoughts" are analyzed and corrected, patients often report relief from the symptoms of depression. Both drug therapies and cognitive therapies have had successes, and several studies have found that applying both treatment regimes together

produces the maximal benefit for patients. Despite this congruence though, there is often animosity between different research practices. Not surprisingly there is strong debate about what level of analysis is correct and this has clear and immediate consequences for the choice of treatment that might be available.

Supporters of the biochemical hypothesis often use rats as a test subjects for drugs intended for humans due in large part to the fact that we share many homologies in our brain structures. One of the complaints leveled against the use of animals for human depression experiments is that they fail to take into account the cognitive aspects of depression which are not present in rats. We might share a lot with rats, but we assume that they do not have many of the cognitive faculties that we have and we don't think that we experience depression in the same way as they do. After all we don't know what it's like to be a rat. But with homology, differences in genetics, developmental pathways and brain structures between rats and humans can give us clues to the cognitive aspects of depression.

DEP suggests that studying only one aspect will not give us a complete understanding of depression. We should expect that the biochemical aspects of our brain can affect the cognitive aspects and likewise that the cognitive will affect the biochemical. Neuroplasticity research has already helped patients with OCD. Patients can undergo cognitive behavioral therapy in which they change in a variety of ways how they respond to their obsessive thoughts and actions. The end result is argued to be a change in the actual structuring and functioning of the brain as the patients develop new behavior patterns with evidence of resulting functional changes in the brain (Doidge, 2007). A DEP approach changes the way that we treatment techniques by moving

toward the recognition that multi level analysis can help to show which treatment regimes work well together and why.

5.3 Summary, Conclusions and Future Work

In this thesis I have explored the current state of evolutionary psychiatry through the work of Dominic Murphy and Jerome Wakefield among other authors. Murphy provides a taxonomy for evolutionary explanations of mental disorders and some explanations fit under Murphy's taxonomy while other evolutionary explanations were unclassifiable. The authors whose explanations were classifiable under Murphy's taxonomy proved to share a commitment to a specific research program in evolutionary psychology which forms the foundation of their approach to evolutionary psychiatry. This adherence to NEP proved problematic and I discussed many criticisms that show NEP to be biologically inadequate. I argued that Murphy's taxonomy failed to taxonomize many evolutionary explanations for mental disorders found in the literature because of the adherence to the NEP understanding of the brain.

I then described the "intentions of nature" idea which derives from NEP. I argued that a scientifically acceptable and useful evolutionary psychiatry cannot be based on NEP and that we require a more biologically adequate evolutionary psychology. This account also needed to be able to account for the diversity of explanations in the evolutionary psychiatry literature as well as be robust enough to account for the many different causal factors that seemed to be present in some evolutionary explanations. The account also required an ability to explain the variety of concepts of mental disorders and the role they play in defining and categorizing mental disorders.

In the third chapter I examined the history of evolutionary biology and showed that developmental biology has been a missing component in evolutionary biology for most of the 20th century. I described the genesis of this field that was focused on integrating development into biology known as evo-devo and the provided a brief sketch of the field as well the potential for DEP that has been emerging out of evo-devo. I argued that DEP provides a better basis for evolutionary psychology than NEP because NEP fails to take both development and evolution seriously while DEP takes both seriously and represents a conception of evolutionary psychology compatible with modern evolutionary biology. While DEP provides a better foundation for evolutionary psychology some aspects of NEP research might still be valuable.

In chapter 4 I showed that NEP based evolutionary psychiatry was based on a conception of SE function that described mental disorders in terms of SE dysfunction. I argued that SE function was problematic and that SE dysfunctional accounts of mental disorders were not realistic and this concept of mental disorder was not as universally held as claimed by Wakefield. I suggested an alternative account of a DEP CR functional account of mental disorders. SE function can be utilized by DEP but CR function has more foundational role in DEP so I argued that it would be more appropriate to use as a way of defining mental disorders.

I argued that there are at least three goals in psychiatry which can require separate taxonomies in order to properly isolate the causal factors involved. I showed how CR function allowed us to set the goals and levels of our analyses which could help us to elucidate both the causal factors involved in mental disorders as well as the values involved. I argued that values and scientific information were both required to be used

together when defining mental disorders and that a DEP CR Functional account provided the best way to make our values explicit and provide us with the best scientific data regarding the causal factors we are interested in studying in psychiatry.

I also showed how DEP could explain why some evolutionary explanations do not fit under Murphy's taxonomy and why a DEP based evolutionary psychiatry is better situated to account for these types of explanations. A DEP based evolutionary psychiatry provided us with a strong understanding of the OCD examples and also showed how we could explore all of the causal factors necessary for generating more complete evolutionary explanations. I then considered some objections that helped further illuminate how a DEP CR functional account of evolutionary psychiatry might work.

Finally in the fifth chapter I offered some examples of how a DEP based evolutionary psychiatry might affect psychiatry itself beyond its role as a niche sub discipline. I suggested that it could offer psychiatry a way of developing a stronger account of how we might prevent mental disorders and why this might be important. I also suggested that DEP provided an impetus for developing a different understanding of responsibility in psychiatry. Then I suggested that some debates about treatment could benefit greatly from a DEP analysis that could highlight the important connections between both sides.

Now that I have summarized the thesis I wish to conclude by returning to the discussion at the beginning of the thesis about evolutionary medicine in general and the calls for evolution to be integrated into the medical school curriculum and the calls for evolution to be used in psychiatry specifically. First it should be obvious that I support Nesse and colleagues in his call for the integration of evolution in medicine and

psychiatry in particularly. However, this is obviously qualified support dependent on the kind of evolution that is to be taught. I have suggested that Murphy`s enthusiasm for evolution was dulled at least partially due to the assumptions of NEP which have led to many problems. NEP seems to suggest that for evolution to be of value to psychiatry natural selection must be at the forefront of discussions and it must be involved in dictating the concepts of mental disorders.

The account that I have developed does not require this as I envision evolution eventually being integrated into biological explanation of psychology as well as psychiatry. A biologically adequate understanding of evolution can be implicit even if evolutionary discussion is not present all of the time. I have aimed for an account of evolutionary psychiatry that need not be limited to the role of the niche sub discipline "evolutionary psychiatry". If we understand evolution and development and the relationship between them properly we can see that a DEP account of psychiatry is simply a biologically adequate and accurate account of psychiatry which has the potential to greatly add to the goals of psychiatry in general.

Evolution can be in the background when not required and come to the foreground explicitly whenever it is needed. In this way evolution can function as the basic science that Nesse also envisions. In one sense we may want to aim for an eventual collapse of the distinction between psychiatry and "evolutionary" psychiatry because when done properly evolutionary psychiatry simply means biologically adequate psychiatry.

The same applies for general medicine as well. Whether or not an evolutionary account will be of use depends on whether or not the version of evolution is biologically

adequate and represents a full history of evolutionary theory including the recent incorporation of development. DEP is still in its early stages so there is an argument to be made that a general medicine account of evolutionary biology might have even more to offer. I suggest that incorporating DEP into psychiatry might serve as a good model for how evolution should be incorporated into general medicine while the NEP based account is a model to avoid both in terms of content and methodology.

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