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FORGING PSYCHIATRIC CATEGORIES:
A Philosophical Examination of the Creation and Stabilization of Diagnostic Knowledge

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Abstract
FORGING PSYCHIATRIC CATEGORIES:
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This dissertation explores the entanglement of ethics and epistemology in the domain of psychiatric research, considering the ways that measurement practices and ethical values interrelate. If psychiatric clinicians, researchers, and patients are not acutely aware of the relationship between experimental configurations and the meaning of scientific data, unhealthy realities that take psychiatric constructs as fixed parts of nature will persist.

The core of this work consists of case studies on the emergence and stabilization of two psychiatric diagnostic categories, Attention Deficit Hyperactivity Disorder (ADHD) and Depersonalization Disorder (DPD). Carefully examining the experimental configurations employed in the measurement of psychiatric disorders, this study shows how the neurobiological description of a psychiatric disorder is produced by employing a broad array of measurement techniques including psychopharmacological interventions, neuropsychological assessments, self-rating scales, historical studies, behavioral observations, psychophysical apparatuses, brain scanning devices, guided interviews etc. The dependence of each of these techniques upon others demonstrates the highly contingent status of diagnostic categories.

The findings of this study include the claim that the current best-confirmed neurobiological description of ADHD, the catecholamine hypothesis, could not have

been developed without the treatment and measurement of the disorder with psychostimulant medications. This observation and others suggest that the knowledge produced regarding psychiatric diagnostic categories needs to be carefully examined to avoid treatment plans that implement research data that is not well understood. I conclude that researchers, clinicians and patients are responsible for the data produced in scientific studies, and should therefore take a critical stance to conclusions drawn from scientific data when making decisions about treatment.

Keywords: Entanglement, Ethics, Epistemology, Psychopharmacology, Clinical Validity, Etiological Validity, Active Genealogy,

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Charles and Diana
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Introduction: The Entanglement of Ethics and Epistemology in Philosophy of Psychopharmacology

Interest in the philosophy of medical sciences has grown to the point where epistemological and ontological questions related to medical theory and practice are being posed with greater frequency and urgency. The special area of philosophy of psychiatry has garnered more attention in recent years. Examples of the types of compelling questions raised in this area can be found in Dan Stein's book *Philosophy of Psychopharmacology*.

Stein's book considers a broad array of topics in clinical psychiatry and the practices of pharmacological intervention. Using an "integrative approach" that draws on both classical and critical traditions in philosophy, Stein explores a range of issues about proper categorization of psychiatric phenomena and psychotropic compounds. Stein also considers what the use of psychotropic compounds might reveal about personal identity and emotion, the distinction between psychopharmacological and psychotherapeutic interventions for mental disorders, and the implications of psychotropic compounds for evolutionary and other theories of psychology. He delves as well into moral questions regarding human use of psychotropic compounds, including the issues of proper clinical treatment for psychiatric disorders, and the enhancement of psychological faculties in non-pathological contexts.

Throughout the text Stein also comments on various theoretical and practical issues of psychopharmacological treatment. One issue that comes up just briefly is a

question about how the practice of psychopharmacological intervention helps to establish the validity of mental disorders. This question warrants more attention than Stein gives it in the space of the book. Stein has acknowledged additional complexities in this area, ones that warrant deeper investigations into the relationship between diagnostic validity and knowledge making practices. My research explores the relationships between the diagnostic categories of Attention Deficit Hyperactivity Disorder (ADHD) and Depersonalization Disorder (DPD) and the pharmacological interventions used to treat them. These relationships provide unique cases exemplifying the entanglement of epistemology and ethics.[1]

Trained in both psychiatry and philosophy, Stein's knowledge of psychopharmacology is efficiently applied to the philosophical questions he raises. Using a series of philosophical examples based on clinical experience he discusses how responses to his target questions might be developed. Throughout the book Stein employs "cognitive-affective science,"[2] and an approach that integrates classical and critical traditions in philosophy[3] in order to respond to these questions.

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[1] My use of the term "ethics" in this paper is not to establish some normative principle and suggest that it has been violated, but to promote the view that individuals involved in the production of knowledge must become responsible for the way the questions are framed and solved within their discipline. I take this view, in particular, from Karen Barad's book *Meeting the Universe Halfway*.

[2] Stein describes this evidence as a combination of cognitive science, plus affective science (the study of how emotions are manifest in the nervous system), plus developmental psychology, plus evolutionary psychology.

[3] Specifically, Stein endorses "an integrative and realist view of the sciences, for a non-reductionistic naturalist approach to psychology, and for a view on categorizing medical and psychiatric disorder that goes beyond the classical and critical perspectives." Stein claims that this integrative view takes into account the considerations about the role of power in the scientific paradigms or research programmes levied by critical philosophers including Kuhn, Feyerabend, Barnes, Bloor, Latour & Woolgar, and Longino. However, his work never actually produces a critique of the scientifically produced cognitive-affective data he makes

Stein's integrative approach holds that "categories are intimately related to human actions and activities" and that "the brain-mind relies on prototypes and metaphors grounded in human experience, rather than on the formal manipulations of abstract representations." (Dan J. Stein, 2008) These and other considerations lead Stein to the conclusion that "a range of disciplines within the cognitive-affective sciences, including philosophy, provide increasingly sophisticated ideas and data on the embodied brain-mind and its categories." (Dan J. Stein, 2008)

Cognitive-affective data of the type that Stein offers, which includes studies in neuroscience, evolutionary biology, cognitive psychology, etc. are still generated within scientific contexts that do not actively critique the assumptions that are built into experimental configurations. The meaning of scientific data is fully understood only by examining the historical, social and evaluative contexts that have played a part in the production of that data set. When the relationship between epistemology and ethics is properly foregrounded medical scientists will be more responsible for their role in experiments, and these will lead to healthier realities for patients and clinicians.

One piece of evidence that suggests that Stein's work has not made these concerns properly explicit is a review of the book by a practicing psychiatrist. The book review contains the following troubling section:

> For many clinicians, delving into the ultimate meaning of making diagnoses, prescribing treatment, and changing people may feel a bit like driving a car on a busy freeway while contemplating why the road has eight lanes, … The gap between everyday practice and the

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use of. Instead, his position assumes that this type of data is produced in such a way as to avoid these critiques. No form of scientific data can avoid this critique unless it is submitted to a rigorous contextual analysis. Stein's claim to this effect therefore fails.

> ultimate meaning of what we do as clinicians was brought home to me when a very capable neurologist friend of mine told me about a patient he had just referred. "Perhaps you can explain the question she asked me," he said. "She wanted to know what your philosophy was. 'Is he a psychopharmacologist, a behaviorist, a Freudian, a Jungian, or a cognitive therapist? What is his orientation toward mental illness?'" "'He's a doctor,' I told her. 'He'll prescribe whatever treatment makes the most sense.'" (Dubosvky, 2009)

This type of response to theoretical questions from practicing clinicians demonstrates what I would refer to as an "uncritical attitude." The concern for the critical philosopher about the phrase "whatever treatment makes the most sense" is that various treatments come to "make sense" in a field of power relations that do not necessarily have all the patient's interests in mind. For whom, exactly, does the chosen treatment "make sense?" While Stein cannot be held entirely accountable for the response of his readers, this review suggests that the philosophical considerations he has advanced look more like an interesting puzzle than an indispensable ethical consideration.

In adding detail to his integrative approach, Stein suggests that the questions he poses in *Philosophy of Psychopharmacology* should be answered with a view that takes seriously the social influences on categorization, but retains a commitment to naturalist realism.[4] His view is that cognitive/affective data represents a form of evidence that takes into account social influence on scientific practices, and thus already contains a critical component. Stein develops a new series of thought experiments for each chapter and then brings in cognitive/affective data to ground his position on the relevant phenomenon. While this process can be effective for dealing with certain questions in this area of inquiry, dealing with the actual

[4] Stein describes this view as allowing that "natural and social sciences provide knowledge of real structures and their mechanisms."

scientific experiments presented in research papers provides a richer context for analysis.

First I will consider a point that Stein makes regarding the relationship between the diagnostic category of ADHD and its requisite pharmacological intervention, psychostimulant medication. I will maintain that the conclusion he draws does not accurately characterize the relationship between ADHD and psychostimulants. My critique draws on a case study of ADHD in relation to psychostimulant medication. This examination provides a more concrete basis on which to understand the relationship between psychiatric diagnostic categories and pharmacological interventions.

In particular I will defend the view that psychopharmacological interventions provide two kinds of support to the diagnostic validity of mental disorders, and show in turn how this has relevance to good decision making in science. My analysis will conclude by considering the difficulties for stabilizing psychiatric diagnostic categories without psychopharmacological treatments by drawing on a case study of Depersonalization Disorder.

Stein draws the following conclusion between the validity of ADHD and its accepted psychopharmacological intervention, psychostimulant medication.

> …dopamine releasing agents are not only effective in improving concentration in patients diagnosed with attention-deficit/ hyperactivity disorder (AD/HD), they may be used by ordinary college students or by military personnel to enhance cognitive performance, thus raising questions about the validity of AD/HD as a disorder. (Dan J. Stein, 2008)

Stein's reasoning here seems to suggest that because the stimulants used to treat ADHD are also effective outside of that context, the evidence suggests that the

medication acts as a type of cognitive enhancement, and not a cure for a mental disorder. If this medication is a cognitive enhancement, then the mental disorder it "cures" should not be considered a valid mental disorder. Mental disorders are taken to be a type of illness that affects the brain of the patient. The treatment via pharmacological intervention of the patient's neurochemical system is thus taken to be treating the cause of the disorder.

This issue reaches a core tension within the field of psychiatry today. There are many factors that determine the validity of a psychiatric disorder, and the etiological description of the disorder is only one of them. The problems facing the diagnostic category of the disorder in relation to its etiological description do not necessarily undermine the broader structures of validity for the disorder. There is also a "clinical validity" to be considered as distinct from the "etiological validity," and a disorder can be considered valid (i.e. treated as "a real medical entity") as long as its clinical validity remains stable.[5]

Sociologist Ilina Singh has carefully developed the distinctions between the scientific and cultural logics that comprise the reality of ADHD, and shown that these different logics can result in disparate treatment practices for the disorder. (Singh, 2005) The distinction between the medical model of mental disorders (which requires a neurobiological description) and the cultural context of a disorder has also been recognized by the psychiatrist Appelbaum as creating a tension between different notions of the validity of mental disorders. (Appelbaum, 2005)

[5] This is the case with disorders such as Axis 1 depression, which I discuss further below.

Stein's comment provides an excellent opportunity to explore the distinction between the scientific etiological validity of psychiatric diagnostic categories and the clinically based practical validity of mental disorders. What it means to say that a mental disorder is "valid" means different things in different contexts. In psychiatry, the "validity" of a diagnostic category is taken as a measure of how well the category corresponds to phenomena in the world. In the context in which psychiatry is taken as a clinical practice of treatment the term "valid" can mean that a particular constellation of psychiatric phenomena causes problems for the functioning of patients and needs to be treated with various therapies. In the context of scientific psychiatric research the term "valid" refers to a psychopathological description that has been measured using various *statistically* valid procedures, and has a well confirmed etiological description associated with it.

One way of distinguishing between these two forms of validity might be to recognize a divide between the theory and practice of psychiatry. While theory and practice no doubt *inform* one another in the psychiatric context, they do not necessarily *conform* exactly to one another. While clinical procedures are rigorously scientifically tested and proven, they rarely attempt to produce an etiological description of the disorder. They are focused instead on what provides relief to a suffering patient. Etiological descriptions of mental disorders can change radically without affecting the clinical validity of that disorder.

ADHD has both a broadly accepted clinical validity to it as well as a well confirmed neurobiological etiological description. These two types of validity serve

to bolster one another. The clinical recognition of the phenomenon has led the disorder to be "the most well studied disorder in childhood psychiatry." (Russell A. Barkley, 1998) The etiological description confirms the correctness of the most favored treatment for the disorder, psychostimulant medication. The catecholamine hypothesis of ADHD, --- the view that the disorder is the result of a deficit of the catecholamine neurotransmitters dopamine and norepinephrine in the prefrontal cortices of patients, --- has emerged through the development of psychopharmacological evidence. Looking carefully into the experiments done to support this hypothesis, it becomes apparent that this view is founded partly on the logic of chemical deficit or imbalance, which is part of the turn towards scientific psychiatry. Appelbaum has brought this discussion to light by noting the importance of psychopharmacology in the narrative of scientific psychiatry. (Appelbaum, 2005) He recognizes that in a conceptual framework in which mental disorders are characterized as medical disorders like any others, neurobiological descriptions of disorders that allow interventions with neurochemistry prove mutually reinforcing.

This feature serves to highlight the entanglement of ethics and epistemology. Entanglement occurs when treatment of a disorder is co-constituting[6] with the way that knowledge is produced about it. In the case of ADHD it suggests that the catecholamine hypothesis emerges from our practices of medication for the disorder, and that this hypothesis also supports the further medication of patients with the

[6] A relationship of co-constitution suggests that the relationship of causality between two concepts is not uni-directional, and thus cannot be understood under standard theories of causation.

psychostimulants that promote catecholamine release in the brain. Looking further into the history of the "paradoxical effect logic" of the disorder, we can see how Stein's contention about validity of the disorder in connection with the role of psychopharmacology has emerged. This will allow us to see the special role that the drugs play in the validities of ADHD.

Psychostimulants were first administered to children in the context of therapy in 1937, long before the diagnostic category of ADHD existed. (Bradley, 1937) As the remarkable effects of psychostimulants on attention and classroom behavior were recognized, studies that helped to determine the reason for these effects proliferated. By the 1980s the "paradoxical effect" narrative had become commonplace among clinicians and teachers. (V. E. Snider, Busch, & Arrowood, 2003) This view suggested that hyperactive children were hyperactive because they lacked certain brain chemicals that allowed them to sit still and concentrate. Furthermore, because Ritalin, the favored treatment for ADHD, was a stimulant it seemed obvious to conclude that the compound would cause normal children to behave more hyperactively. It was discovered subsequently that this paradoxical effect was false. Indeed, healthy controls also show increased concentration, etc. from the administration of psychostimulants. (Rapoport et al., 1980) This discovery damaged the neurobiological narrative that supported the etiological validity of ADHD. Mounting psychopharmacological evidence was used to assuage growing concerns among the public that ADHD was being over-diagnosed and psychostimulants over-prescribed. (Singh, 2009) In particular, it became established

that despite the fact that psychostimulants do not have special effects on ADHD patients, they still present the most effective treatment for the ADHD population.

It is this moment in the history of ADHD that Stein's claim is directed at. The discovery that healthy controls also received cognitive benefits from psychostimulants undermined the early etiological story of ADHD. While it seemed likely that this revelation would cause problems for the clinical validity of the disorder, this was not to be the case. The clinical validity of the disorder was already well established in the minds of psychiatrists, teachers and parents caring for ADHD children.

This situation regarding the disparity between clinical and etiological validity of mental disorders is not unique to ADHD. A similar process has occurred regarding the link between the neurotransmitter Serotonin and the phenomenon of clinical depression. As Stein has remarked:

> SSRIs are effective in depression, from which some deduce that serotonin is involved in the cause of depression. But it turns out that that is probably a step too far. First, SSRIs do act on the serotonin system, but via that they affect multiple other molecules, so their end effects are not specific. Second, it turns out that there really isn't a whole lot of evidence (if any) that serotonin is a single cause of depression. Indeed, any one of the ultimate places the SSRIs act on could contribute to depression. (Dan J. Stein, 2011)

This state of affairs has also been recognized recently by psychiatrist Alan Frazer. He suggests that the connection between depression and serotonin emerged because of the state of the science on Parkinson's disease when tricyclic antidepressants first became available. Specifically, Parkinson's is strongly associated with a dopamine deficit in the brain, and this thinking was immediately applied to the phenomenon of depression in relation to serotonin. Because these anti-depressants were known to act on the serotonergic system, the conclusion that depression is caused by a

serotonin deficit was easily reached. Frazer has said "There is no doubt in my mind that the Parkinson's story had a strong impact on the way that people were thinking about depression." He went on to state, "It became easy to speculate that depression was due to a deficiency." (Frazer, 2012) Regardless of the fact that this thinking has now been shown to be false, the justification for drug treatment of depression is no weaker than before; drugs work for treating depression, which is really all that is necessary to retain the clinical validity of the disorder and its treatment through SSRIs, etc.

When we examine the etiological studies that have been carried out to support the scientific validity of mental disorders the importance of psychopharmacological interventions for both forms of validity becomes apparent. Not only do psychostimulant medications help to establish the clinical validity of ADHD by assuaging the most noticeable symptoms of the disorder, they are also necessary for generating a large portion of the etiological evidence for the catecholamine hypothesis. This is the core issue to note when considering the entanglement of epistemology and ethics in this case. Insofar as the clinical practice of administering stimulants becomes the most well studied childhood psychopharmacological treatment, it becomes the centerpiece of the etiological narrative, which in turn lends more support to the practice of psychostimulant treatment. It is important for clinicians to be critically aware of the context in which the etiological data is developed. The experimental configurations in which ADHD is studied rarely fail to include psychostimulant medication.

Studies that produce a neurobiological picture of ADHD have traditionally relied on pharmacological, animal, neuropsychological, neuroimaging, and genetic evidence. It is important to recognize, however, that many of the studies that produce non-pharmacological evidence still employ and to some extent rely on pharmacological features.

Meta-analysis papers recognize that although research data focusing on the generation of neurological and genetic evidence is proliferating, psychopharmacological evidence has been emphasized most heavily in supporting the catecholamine hypothesis. Most importantly, these papers have observed that neurological and genetic studies rely on pharmacological interventions in order to produce statistically significant data. (Pliszka, 2005) (Arnsten & Li, 2005) Examples of this reliance include studies in which neuroimaging techniques are employed to differentiate between the brains of ADHD patients and those of normal controls. Neuroimaging data shows evidence for brain dysfunction in the PFC of ADHD patients, but relies on pharmacological data to complete the link. One review of the data supporting the catecholamine hypothesis cites findings in neuroimaging studies that show decreased blood flow or smaller brain volume in the PFC of ADHD patients, and then shifts attention to the psychostimulant evidence that makes these findings relevant in this context. "Notably, the frontosubcortical systems that control attention and motor behavior are rich in catecholamines, which have been implicated in ADHD by the mechanism of action of stimulants." (Faraone & Biederman, 1998) When researchers employ pharmacological interventions as part

of neuroimaging studies, the data express significant differences between ADHD and non-ADHD brains. (Semrud-Clikeman & Pliszka, 2005) (Pliszka, Glahn, et al., 2006) (Pliszka, Lancaster, Liotti, & Semrud-Clikeman, 2006)

Experiments that help the phenomenon of ADHD to appear as inherently neurobiological frequently rely on psychopharmacological intervention. The boundaries enacted within psychopharmacological experiments tend to lead towards the view that the drugs are not actually a part of the apparatus of measurement. Statements like the above demonstrate just how fundamental medications are for the generation of scientifically significant data.

This feature can also be seen when it comes to animal modeling data. Mice that have been administered a catecholamine-focused neurotoxin (one that selectively damages catecholamine transmission sites) exhibit hyperactive symptoms as well as severe learning disabilities. This evidence alone would not provide particularly strong support for the catecholamine hypothesis, since the damage to the rodent brain as a result of the neurotoxin is severe. Only after these same mice are subsequently treated with methylphenidate or amphetamine to test drug responses do the results bear much relevance to ADHD. (Shaywitz, Klopper et al. 1977) (Luthman, Herreramarschitz, & Lindqvist, 1994) These mice show reduction of post lesion learning deficits with the application of stimulants, which suggests that the deficits are "more attentional in nature than true learning deficits." (Ferguson 2001) The administration of the drugs is vital for differentiating learning deficits from strictly attentional deficits. Without the drugs, these studies would not

present any significant data for the catecholamine hypothesis. Again, the reliance of the etiological story of ADHD on psychostimulant medications is apparent.

Genetic studies of ADHD show a similar reliance on psychopharmacological interventions. Genetic studies of attention have been carried out using gene knockout mice, which are bred to lack dopamine transporter and/or receptor genes. These knockout mice showed hyperactive symptoms, which is significant for the catecholamine hypothesis. Researcher Michael Posner has noted that these gene knockout mice were first bred in response to results from pharmacological studies demonstrating the effects of stimulants on dopamine pathways. (Posner, 2004) Also, within such studies dopamine transporter knockout mice were treated with methylphenidate and amphetamine compounds in order to reduce "learning impairments" associated with genetic alteration. In meta-analysis it has been concluded that "such learning impairments prevent the usefulness of this model." (Ferguson 2001) Therefore, without the administration of psychostimulants, animal genetic models are significantly less useful for generating evidence about attention and attention deficits. The animal and psychopharmacological models are often cited as independent forms of evidence supporting the catecholamine hypothesis. However, in the cases I have just referred, to the psychopharmacological data provides the basis for the evidentiary significance of animal models. Animal models are less likely to produce supporting data for the catecholamine hypothesis if psychostimulants are not employed within the context of the experiment. It is for

this reason that critical examination of every experimental configuration is so important.

Meta-analysis literature repeatedly demonstrates the importance of psychostimulant data for evidence of the catecholamine hypothesis. In reports on the history of the hypothesis, the effects of psychostimulants are frequently noted as one of the initial pieces of evidence that led scientists towards this view. One such example can be seen in the following paragraph:

> Satterfield and Dawson (1971) were among the first to propose that ADHD symptoms were caused by frontolimbic dysfunction.[7] They suggested that weak frontal cortical inhibitory control over limbic functions might lead to ADHD. The success of stimulant medications and animal models implicating dopamine pathways were taken as support for this model. (Faraone & Biederman, 1998)

This early permutation of the catecholamine hypothesis is grounded in the success of psychostimulants. Some animal models cited to support this view take the form of mice and rats being administered psychostimulants, and so are actually better understood as psychopharmacological data. Other animal models are created using rodents and monkeys who have brain lesions that damage their frontal cortices. Hyperactive behavior amongst these test subjects, while consistent with the view that executive function occurs in frontal cortices, does not provide the same degree of specificity as the pharmacological data. The pharmacological data provides an essential ground for many of the animal models, and thus should be seen as the crucial form of evidence for the catecholamine hypothesis.

[7] This claim is not strictly correct because the classificatory heading "ADHD" and its requisite symptoms did not exist in 1971.

Other histories of the catecholamine hypothesis credit different scientists with initial observations about catecholamine dysfunction, yet still cite the psychopharmacological data as paramount.

> Basic pharmacological research established that the stimulant drugs augment central norepinephrine and dopamine. Reasoning in part from these data, Paul Wender proposed, in an influential book entitled *Minimal Brain Dysfunction* published in 1971, that alterations in catecholamine neurotransmission mediate the therapeutic effects of the stimulants in ADHD. (Solanto, Arnsten, & Castellanos, 2001)

This account also demonstrates that the earliest claims that ADHD is caused by catecholamine dysfunction are based on the success of psychostimulant medication for the treatment of the disorder. (Wender, 1971) The animal models that have been invoked in other accounts are not clearly separable from the pharmacological data, and in this account the pharmacological data is of paramount importance.

The view that the catecholamine hypothesis emerges from the success of stimulant medication is also shown in the current literature educating patients and parents of patients on the causes of the disorder:

> The effectiveness of stimulant medication, along with animal models of hyperactivity, also point to catecholamine disruption as at least one source of ADHD brain dysfunction. (NRCA, 2011)

The education of the public as to the etiology of ADHD centers on the success of psychostimulant medications. This is a crucial fact to recognize for understanding the relevance of psychopharmacological intervention with respect to the validity of mental disorders.

Animal models provide sources of evidence that are difficult to differentiate because the global effects of altered brains and genetic codes create severe learning deficits beyond those that can be attributed to attention in the absence of

psychopharmacological data. The tremendous success of treating ADHD patients with psychostimulants is the central justification for the view that the ADHD brain lacks the chemicals promoted by those psychostimulants. This view in turn justifies the further administration of psychostimulants and stabilizes the apparatus of measurement, thus promoting both types of validity of the disorder.

No other form of etiological evidence (i.e. neuroimaging, neuropsychological, animal or genetic evidence) is also capable of providing clinical evidence for the validity of the disorder. None of these other measurement practices also take the form of a therapy for the disorder, so none can occupy this special role within the practices of psychiatric research. The ability to intervene directly with the neurochemistry of patients produces one of the only forms of neurobiological data. Pharmacological data helps to produce the view that ADHD is caused by a lack of the exact neurochemicals that psychostimulants promote. Without an effective critical examination of psychopharmacological experiments, we allow the argument for their continued prominence in treatment to be, in a way, self-sealing.

The preceding evidence is meant to show how our experimental practices are carried out in conjunction with our treatment practices to bolster the validity of ADHD in both etiological and clinical contexts. The neurobiological picture of psychiatric disorders provides the framework for the etiological narrative expressed in the catecholamine hypothesis. The successful treatment of ADHD by means of psychostimulant medication supports its continued use. The catecholamine neurotransmitters implicated in the etiological narrative of ADHD are the exact ones

promoted by Ritalin (methylphenidate) and Adderall (dextroamphetamine), the two most common psychostimulants used to treat the disorder. Both clinical and research psychiatrists have begun to propose that psychostimulant medication is the only necessary treatment for ADHD and that other forms of psychological treatment are unnecessary. (Jensen et al., 1999) (Abikoff et al., 2004)

Insofar as the treatment practices also form the basis for knowledge production of the etiology of the disorder they influence the recommended treatment program for the disorder. Because we often see pharmacological intervention as a treatment rather than a form of knowledge production, it is easy for the entanglement of ethics and epistemology to be overlooked in psychiatric contexts. The practices of philosophy can help to unearth the apparatus and foreground these entanglements.

Stein's claim about the validity of ADHD is problematic not because it is untrue, but because it fails to go deeply enough into the issue of validity to make plain the complexities of ADHD in relation to psychopharmacological interventions. While it is true that regarding psychostimulants as cognitive enhancements does "raise questions about the validity of ADHD as a disorder," these questions do not pose any serious threat to the acceptance of ADHD as a valid mental disorder, which the tone of the remark suggests.

Looking into the etiological and clinical validation practices of another psychiatric disorder, Depersonalization Disorder, we can see even more clearly how psychopharmacological interventions underpin both forms of psychiatric validity.

Depersonalization Disorder (DPD) is a little known psychiatric disorder and it bears a troubled history. Scientists have questioned the validity of the disorder on the grounds that it fails to be clearly separable from other disorders such as anxiety and depression. (Parnas & Handest, 2003) The reason for this is that the main symptom of the disorder, the experience of depersonalization or derealization,[8] is among the most common psychiatric symptoms and frequently expresses during the course of other psychiatric disorders. (Sierra, 2009)

The attempt to subsume DPD under the category of an Axis I disorder such as anxiety or depression failed partially because patients with DPD experience little relief from anti-depressants and anxiolytics. (Simeon & Abugel, 2006) While this evidence supports the view that DPD should be characterized as a separate disorder, it does little to help generate effective treatments or an etiological narrative for the disorder. Lacking these two crucial components for establishing the validity of a disorder, researchers Daphne Simeon and Mauricio Sierra and their colleagues have had to explore other means for generating the necessary evidence to establish the category as diagnostically legitimate.

These means encompass a diverse array of experimental practices, including explorations of the historical phenomenological stability of the disorder, (Sierra & Berrios, 2001) the creation and validation of a new self-rating scale for the disorder

[8] Depersonalization is described as the experience of not being in control of or connected to one's body. Derealization is usually characterized as the experience that the world and people surrounding the subject are unreal. These symptoms can express in the context of other disorders. It is only when these symptoms become chronic in nature and not better explained by the presence of some other disorder that the diagnosis of Depersonalization Disorder is made.

(Sierra & Berrios, 2000), the division of the disorder into differentiated symptom clusters, (Sierra, Baker, Medford, & David, 2005) (Simeon et al., 2008), and the production of psychophysiological data, (Giesbrecht, Merckelbach, van Oorsouw, & Simeon, 2010). These studies have all been generated alongside attempts to discover and prove the efficacy of psychopharmacological treatments of the disorder. (Sierra, 2008) Attempts to treat the disorder with lamotrigine (Sierra et al., 2006) (Sierra, Phillips, Ivin, Krystal, & David, 2003) (Sierra et al., 2001), fluoxetine (Simeon, Guralnik, Knutelska, & Schmeidler, 2002) (Simeon, Guralnik, Schmeidler, & Knutelska, 2004), and clomipramine (Simeon, Stein, & Hollander, 1998) have failed. Depersonalization Disorder has proven refractory to every well-established psychopharmacological intervention.

Despite this fact, researchers have worked to generate a neuro-anatomical perspective of the disorder by considering the phenomenological characteristics of the disorder. (Phillips et al., 2000) (Sierra & Berrios, 1998) This etiological description, the "cortico-limbic disconnect hypothesis" of DPD, while compelling in its explanation of the phenomenal character of the disorder, is not without its problems. Simeon suggests that the body of data generated about the disorder is "partially in accord and partially contradictory" with the cortico-limbic disconnect hypothesis. (Dell & O'Neil, 2009)

The difficulties of generating and confirming a neurobiological hypothesis for DPD are bound up with the relationship of the disorder to psychopharmacological practices. While DPD is refractory to all conventional pharmacological

interventions, it can be induced through patient interaction with various illicit compounds. (Medford, Baker, et al., 2003) (Simeon, Kozin, Segal, & Lerch, 2009) While DPD scientists are quick to note this fact about the disorder, using it as further evidence of the neurobiological presentation of the disorder, it is difficult to generate any more detailed evidence than this. (Reutens, Nielsen, & Sachdev, 2010) The reason for this difficulty is not hard to discern. Systematic measurement of the effects of illicit compounds in relation to DPD is basically impossible. Patients who report the onset of DPD symptoms after experiences with illicit compounds often do not immediately seek treatment. DPD researchers have not been able to pinpoint the etiology of drug-induced DPD. One meta-analysis paper concludes that the disorder is the result of "interaction between psychological and neurobiological factors." (Reutens et al., 2010)

Preliminary forays into the possibility of reverse engineering a psychopharmacological treatment for DPD based on data about drug-induced DPD have been abortive. Simeon noted the difficulty of developing new central nervous system drugs in a recent interview saying

> "It's a matter of market and cost; the pharmaceutical companies have made big announcements in recent years about cutting central nervous system (CNS) drug development because they are the most difficult to develop and test. [They are] unpredictable and uncertain and complex and costly and you can spend decades and get nowhere. They're cutting back in that category of drugs and there you have little depersonalization disorder in the midst: it's not schizophrenia and it's not depression…"(Simeon, 2011)

This comment shows the difficulty of creating new drug treatments for disorders that do not have high rates of incidence among the population.

The situation with DPD provides an effective foil for that of ADHD. Where ADHD is validated both clinically and etiologically as a result of an effective

pharmacological intervention, DPD researchers and clinicians have no such resource. The various types of measures that have been generated to validate the disorder among the clinical and research communities in psychiatry have been painstakingly developed by psychiatrists who care immensely about the patients they are trying to treat. The struggle to have DPD recognized by mainstream psychiatry is ongoing. While it is certain that Depersonalization Disorder will appear as a diagnosis in the upcoming edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM – 5), this was by no means a foregone conclusion when this DSM task force was first convened.

The practices of validation for DPD and ADHD offer a unique comparative space to consider the entanglement of ethics and epistemology in the medical sciences. In the case of ADHD, a highly effective psychopharmacological intervention produced early clinical successes. These successes then guided the epistemological practices that produced the etiological story of the disorder. The catecholamine hypothesis of ADHD suggests essentially that the disorder is caused by the lack of the very neurotransmitters promoted by the favored pharmacological intervention. This hypothesis bolsters our future clinical practices for treating the disorder. The difficulty of concluding a single causal direction between the clinical and etiological practices bolsters the claim that the two practices are co-constituting. Neither one can be claimed to causally precede the other, since psychostimulants were being administered to children for school problems before the diagnostic category of ADHD existed. Bradley, the author of the first child psychostimulant

study, made claims regarding the etiological presentation of the disorder in relation to psychostimulants within the space of his first publication. (Bradley, 1937)

In the case of DPD, the fact that the psychopharmacological interactions with the disorder are those of inducement rather than treatment leaves little to build a research program on. All of the imaginative measurement practices devised by DPD scientists to generate reliable data regarding the disorder still do little to support a clear neurobiological description. Because the psychopharmacological treatment for DPD does not exist, it is much more difficult to produce neurobiological data about the disorder. Intervention by means of illicit chemicals is precluded, and the development of new central nervous system drugs is unlikely.

The clinical validity of DPD is supported mostly by the subjective reports of individual patients in combination with emerging psychophysiological studies. The disorder (unlike ADHD) has very few behavioral outcomes, so the only way to diagnose someone with DPD is through clinical interview. The behavioral observations and neuropsychological evaluations that generate and support the diagnosis of ADHD can be administered without ever asking the patient to describe their symptoms. While this difference between the disorders is important, the relationship of the two disorders to psychopharmacological interventions can add detail to our understanding of personal identity.

The neurobiological requirement for the description of mental disorders means that our view of mental disorders has become a mechanical one. If we want to hold that mental disorders occur and can be treated only within the context of

neurochemical intervention, then we make a strong claim about the relationship of personality and behavior to the brain. As is expressed in Ian Hacking's book *Mad Travelers*, different forms of psychological unease manifest themselves differently in different historical contexts. (Hacking, 1998) The disorder known as "dissociative fugue" that was the subject of that book has now been subsumed under the category of "dissociative disorder, not otherwise specified" and is rarely seen.

If we maintain that every form of psychological suffering must be expressed according to a painstakingly crafted neurobiological description, we run the risk of denying treatment those whose suffering does not fit within such a description. The ethical situation for mental disorders bears a clear relationship to our forms of psychiatric knowledge production. Insofar as our willingness to recognize and treat disorders is related to the availability of psychopharmacological interventions to produce clinical and etiological validity, we must be extremely careful to critically examine the ways in which these conclusions are drawn. Stein's book has more to say about the relationship between psychopharmacology and personal identity. Recognizing that the brain-mind occurs within an embodied context is Stein's chief suggestion here, but more can be said. Whether we take a strongly reductionist approach like the position of eliminative materialism proposed by the Churchlands, or a more moderate position, recognizing that neurobiological descriptions of mental disorders may depend on the availability of psychopharmacological interventions is extremely important for our ethical decision making with respect to mental illness.

First Case Study: Attention Deficit Hyperactivity Disorder, Psychostimulants, and the Catecholamine Hypothesis

1.) Opening

This exploration of the relationship between the diagnostic category of Attention Deficit Hyperactivity Disorder (ADHD) and the psychostimulant compounds used to treat it takes place in four main sections. The first two sections consider the scientific and historical underpinnings of the best confirmed theory of the disorder, "the catecholamine hypothesis." This view of the disorder, which holds that ADHD symptoms are the result of the deficiency of certain neurotransmitters in the prefrontal cortices of patients, has its roots in science that dates back to 1937. The initial study documents the effects of Benzedrine, a psychostimulant, on the behavior of school children exhibiting an array of behavioral disorders. As the practices of psychiatry came to align more closely with medical science, the effects of psychostimulants on childhood behavioral disorders began to receive greater attention. The biological origins of mental disorders became more widely accepted in the latter half of the 20th century. As such, *The Diagnostic and Statistical Manual of Mental Disorders*, published by the American Psychiatric Association, began to codify the psychiatric disorders that could be understood as having biological rather than biographical etiologies. Childhood behavioral disorders responded well to pharmacological intervention, and thus could be shown to have neurological components, at which point research into the diagnostic category now referred to as ADHD proliferated quickly.

Although the scientific research into the biological basis of ADHD now contains many forms of evidence including genetic, neurological, neuroimaging and animal models of the disorder, I will show that these forms of evidence all depend in some way on the early success of pharmacological interventions. I will suggest that the stability of the catecholamine hypothesis is contingent upon the success of psychostimulant treatments for the disorder.

The third section of the chapter examines the relationship between the history of scientific psychiatry and the needs of ADHD patients, parents of ADHD patients and others. While the needs of ADHD patients and parents have traditionally aligned with the practices of scientific psychiatry to produce the disorder, the recent recognition of the serious side effects of psychostimulants by the FDA (among other factors) have damaged that alignment. Understanding how the needs of individuals and groups form aspects of the scientific apparatus is vital if we are to become responsible for the meaning of scientific data.

In the final section I explore the possibilities for remediation of scientific and cultural practices surrounding the phenomenon of ADHD to produce healthy realities for the individuals and groups who are concerned with the disorder.

2.) Norms, Psychopharmacology, and the etiological validity of ADHD

The 2002 "International Consensus Statement on ADHD" underscores the legitimacy of ADHD as a valid diagnosis according to medical science. The authors of the report say "We cannot overemphasize the point that, as a matter of science, the

notion that ADHD does not exist is simply wrong. All of the major medical associations and government health agencies recognize ADHD as a genuine disorder because the scientific evidence indicating it is so overwhelming." (R. A. Barkley, 2002) The document contains just two pages of expository text in a similar vein followed by 20 pages of supporting references and signatures of ADHD researchers. It strongly articulates the belief held by scientific researchers that ADHD is a meaningful and valid diagnostic category.

Despite repeated attempts by the ADHD research community to educate the public about ADHD, misconceptions and folk beliefs about the disorder continue to emerge. One *New York Times* article published recently, entitled "Untangling the Myths about ADHD," rehearses the same data provided by scientists in 2002; that ADHD is a neurologically based disorder with a clear genetic component, and that psychostimulant medication is a medically legitimate treatment for the disorder. (Klass, 2010) The fact that this view needs to be so frequently repeated to the public suggests that the message is not being successfully transmitted.

Medical anthropologist Andrew Lakoff has published an article exploring the historical emergence of ADHD and drawn insightful conclusions about the relationship between cultural demands and medical science. Responding to claims that ADHD and the requisite psychostimulant medications such as methylphenidate (Ritalin) and Dextroamphetamine (Adderall) are actually a covert means of social control imposed by the educational system,[9] Lakoff demonstrates that the

[9] (Fukuyama, 2002)

predominance of the ADHD diagnosis can be more consistently attributed to the needs of ADHD patients and their parents. His analysis shows that "Police and school authorities did not so much impose the diagnosis as parents and children insisted on the validity of the designation." (Lakoff, 2000) This history of the phenomenon supports the view that the diagnosis has become so widespread because children who underperform in school need to be normalized with respect to their peers. Lakoff's conclusion, that the model of ADHD that describes it as a "failure of executive function"[10] emerges from a set of techniques of measurement that stabilize it within its context, helps us to understand how science has responded to a process of "normalization." Lakoff's article focuses on the "behavioral checklists and cognitive tests (that) helped to stabilize the disorder and make it reproducible." (Lakoff 2000) In his article, Lakoff cites ADHD researcher F. Castellanos to bolster the view that it is patients and parents who have demanded a rapid and effective treatment for ADHD in the form of psychostimulant medication.

> The most vocal proponents of stimulant treatment for ADHD have been the parents of children with ADHD, but their arguments have been weakened by the imputation of self-interest and by the absence of a coherent explanation for the therapeutic utility of psychostimulants and an understanding of their limitations. (F. X. Castellanos, 1997)

The tension in this paragraph expresses the concerns about the relationship between psychostimulant medication and the validity of ADHD. Without a scientific justification for the positive effects of stimulants on schoolchildren, the people who need such an intervention would be without a crucial piece of evidence. Since the

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[10] For discussion see (R. A. Barkley, 1997)and (Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005).

publication of Lakoff's article a "coherent explanation for the therapeutic utility of psychostimulants" has gained a considerable amount of supporting evidence. I will contend that this model and theory of ADHD, referred to as "the catecholamine hypothesis," has emerged and gained support through the stabilization of measurement practices that employ psychostimulant medication as part of the experimental configuration. The emergence of the catecholamine hypothesis is multifarious and complex, but might be usefully understood as responding to different modes of reasoning. As such this case study will explore the currents of the various modes of reasoning or "strategic logics" that produce the catecholamine hypothesis. Observing the ways in which an endogenous conception of ADHD has emerged in modern psychiatric research, it will go on to consider how logical systems might be altered to produce healthier realities. The goals of psychiatric researchers and clinicians, ADHD patients, parents and other member of our culture focus on health, but often different groups disagree about what "health" means in a particular context. Recognizing the needs of various groups in relation to the diagnosis of ADHD will help to produce a reality in which each group is given an opportunity to have their needs expressed and met.

Understanding how an apparatus of measurement has been devised to stabilize the disorder and meet the needs of parents and teachers raising ADHD children is vitally important to seeing how the current best-confirmed theory of ADHD has been developed. The etiological description of ADHD as a deficiency of catecholamines (the neurotransmitters dopamine and norepinephrine) in prefrontal

cortices has emerged through a set of measurement practices designed to demonstrate the biological connections between stimulant medication and attention. The quotation from Castellanos produces a sign of the entanglement of the etiological and clinical validities of the disorder.[11] If the justification for psychostimulant medication requires "a coherent explanation of the therapeutic utility of psychostimulants" this explanation will have to come in etiological and biological terms. The etiological description of ADHD as a shortage of the very chemicals that Ritalin and Adderall promote in the brain stabilizes the diagnosis by giving a scientific explanation for why underperforming children can be normalized through medication. The validities of the ADHD diagnosis are not just bolstered by the success of psychostimulant treatments; they are deeply enmeshed within that success. If a convincing description of the action of psychostimulants on ADHD brains is available, the stability of the diagnosis is increased because the connection between etiological and clinical validities is strengthened.

Lakoff's view of the disorder is consistent with the Foucaultian notion that a "*dispositif*" (in this case an apparatus of measurement) is produced in part as a response to "to an urgent need" of a society or social group. (Foucault, 1972b) This "urgent need" might be expressed as a demand for American school children to conform to a certain norm in order to be seen as proceeding successfully. (Singh, 2005) The practices of the APA and their production of *The Diagnostic and Statistical*

[11] When I claim that the etiological and clinical validities of a mental disorder are entangled, I mean first that one cannot be understood properly without reference to the other. Secondly, I mean that what occurs within one will have outcomes for the other. Finally, I mean that no clear line of causality can be drawn from one form of validity to the other, meaning that the two phenomena should be regarded as "co-constituting."

Manual of Mental Disorders is one way to look into the creation of norms through diagnostic categories. The impending publication of the DSM – 5 has brought these issues to the fore as editors of previous editions voice their concerns about the difficulties of creating a manual that effectively produces the norms of a culture. Examining the debate surrounding the changes to the DSM helps to foreground the connections between matters of science and matters of society. As the testimony of previous editors of the DSM will demonstrate, psychiatric science cannot afford to begin posing ethical problems only after research has been carried out. The entanglement between ethics and epistemology is too serious to sideline.[12]

My description of the current state of ADHD science builds on and breaks away from Lakoff's analysis in a few ways. My analysis puts more emphasis on the way that psychopharmacological interventions have shaped the current theory of the disorder. My study focuses specifically on the experiments and meta-analysis papers about the catecholamine hypothesis. Because the catecholamine hypothesis has been the focus of a greater number of studies in the decade since Lakoff's publication, it is easier to see how pharmacological treatments have played a greater role in the stabilization of current models of the disorder. Additionally, although Lakoff's historical-anthropological approach has been invaluable in demonstrating the contingency of the diagnosis of ADHD, understanding the current theory of

[12] My focus on the entanglement of ethics and epistemology comes from my engagement with Karen Barad's book *Meeting the Universe Halfway* and from discussions with members of the Science & Justice Working Group. Taking ethics and epistemology as entangled processes means that they stand in a relationship of co-constitution. On this view scientific knowledge making practices inform our notions of correct action and vice-versa. Neither our knowledge nor our notion of correct action can be taken as primordial. These concepts are discussed at length in the concluding chapter of this dissertation.

ADHD requires closer attention to individual experiments and controversies within ADHD research. My development of these examples will help to underscore my interpretation of the disorder's development and the logical processes involved.

3.) Psychostimulants, the catecholamine hypothesis, and neurobiological etiology

The question of what makes a psychiatric diagnosis valid has been explored by Ian Hacking in several different works. (Hacking, 1995) (Hacking, 1998) (Hacking, 1999) His examination of the rating scales used to measure disorders such as Multiple Personality Disorder (now called Dissociative Identity Disorder) have helped to demonstrate the way in which a psychiatric diagnosis is made valid through the stabilization of measurement techniques pertaining to it. The importance of psychostimulant medication for the stabilization of ADHD measurement techniques is formidable, and this has not been fully recognized. Because stimulant medication is usually seen as a treatment for ADHD rather than part of a measurement techniques associated with it, it might be difficult to see initially how the previous statement could be true. When the experiments are examined, however, the evidence for this claim is abundant.

The catecholamine hypothesis of ADHD, the view that the disorder is caused by a deficiency of the neurotransmitters dopamine (DA) and norepinephrine (NE) in the prefrontal cortices of patients, is currently the most well confirmed theory of the disorder. (Pliszka, 2005) (Arnsten et al 2005) (Solanto et al., 2001) The reasoning supporting this theory can be summarized as follows:

1. The principal symptoms of ADHD are in the area of "executive functioning"[13] (including regulation of attention, planning, impulse control, mental flexibility, and the initiation and monitoring of action, including self-monitoring). (Solanto et al., 2001) (R. A. Barkley, 1997) (Arnsten & Li, 2005)
2. Executive functioning (EF) takes place as a result of processes occurring in the Prefrontal Cortex (PFC). (Posner, 2004) (Goldberg, 2009)
3. Executive functioning (or related abilities in the PFC) is shown to be markedly different in the following subjects (as compared to normal controls) (Pliszka, 2005) (Faraone & Biederman, 1998)
    a. Human patients or animals with lesions to the PFC (including Rats & monkeys) have reduced EF or EF-like processes.
    b. Human patients with reduced brain volume in the right hemisphere of the PFC have reduced EF.
    c. Human patients with reduced blood flow or metabolism within the PFC have reduced EF.
    d. Human patients with genetic abnormalities in their norepinephrine and dopamine transmitter sites have reduced EF.

[13] While weaknesses in executive functioning are believed to be one important feature of ADHD, it should be noted that they are not universally considered to be the necessary and sufficient conditions for ADHD. See (Willcutt et al., 2005)

e. Animals (rats, mice, monkeys) with heavily heightened or depleted stimulation to the D1 dopamine receptor have reduced EF-like processes.
f. Human patients with low to moderate norepinephrine functioning have reduced EF.
g. Human patients and monkeys with inhibition of their alpha-2-adrenoceptor show reduced EF or EF-like processes.
h. Rats and mice that are given low dose methylphenidate (Ritalin) have increased EF-like processes.
i. Human patients administered a-2-adrenceptor agonists show heightened EF.
j. Child and Adult human patients treated with Norepinephrine reuptake inhibitors show heightened EF.
k. Gene knockout mice missing dopamine transporters and receptors show decreased EF-like processes.
l. Child and human ADHD patients given low dose methylphenidate (or similar psychostimulant compounds such as dextroamphetamine) show heightened EF.

4. Because the preceding list of subjects have shown diverse EF reactions in association with catecholamine-related processes in the PFC, it is likely that catecholamines are implicated in executive functioning.

5. Because ADHD symptoms are primarily associated with executive function, it is likely that a.) ADHD symptoms are in some way related to deficit or dysregulation of catecholamines in the PFC (weak claim) or b.) ADHD symptoms are caused by a deficit or dysregulation of catecholamines the in PFC (strong claim).

The list of evidence recreated in (3.) from the referenced documents is not exhaustive. It is presented here to demonstrate to the reader the diversity and complexity of evidence supporting the catecholamine hypothesis. It should be clear from the nature of this evidence that necessary and sufficient conditions for ADHD have not been discovered, and that the disorder is understood through a complex amalgamation of evidence. As one commentator has expressed the issue "The neurotransmitter systems for NE and DA are implicated in the pathophysiology of ADHD. Exactly how these systems are dysfunctional in the disorder is unknown, but it is unlikely that a simple deficiency of either system accounts for the symptomology of ADHD." (Pliszka, 2005) Regardless of the lack of complete understanding, the catecholamine hypothesis provides the strongest scientific justification for the treatment of ADHD with psychostimulant medication. The types of evidence that appear in the list under (3.) fall into three broad categories: genetic, neurological, and psychopharmacological. It is partially the overwhelming diversity of studies used to support the catecholamine hypothesis that makes the theory of the disorder so compelling. ADHD researchers who are attempting to express the

catecholamine hypothesis are responsible for organizing, interpreting and extrapolating meaning from hundreds of studies. In this process, the space for detailed critical examination is lost. No doubt these researchers know far more about the hypothesis than can be expressed in a single publication, yet the practices of data presentation do not allow the level of detail that could be communicated in principal. For this reason some of the relationships between different types of data become obscured, and it is these relationships I want to foreground in this section. While genetic, neurological and psychopharmacological data are disparate in many ways, they all depend upon one another for important steps in reasoning that cannot be supplied by one technique of measurement alone.

In order to understand the situation in which the catecholamine hypothesis has emerged, an exploration of the genetic, neurological (animal modeling, neuropsychological data, neuroimaging) and psychopharmacological data is indispensable. It will be important later to note that the foregoing list only accounts for the endogenous (internal to the bodies of patients) factors of the disorder. In addition to these considerations, there are numerous exogenous (external to the bodies of patients) factors associated with the disorder that are rarely considered in relation to the catecholamine hypothesis of ADHD. It is my contention that these exogenous factors allow the phenomenon of ADHD to manifest in particular ways, and that both endogenous and exogenous factors must be understood in tandem in

order to properly explore the catecholamine hypothesis and its relation to psychopharmacology. [14]

Of some importance to my analysis is the final item on the list of evidentiary factors in (3.). While child and adult ADHD patients show heightened executive function when administered psychostimulants, the same is true of normal controls. (Rapoport et al., 1978) (Rapoport et al., 1980) The meaning of this data cannot be determined without carefully examining the experimental context in which it has been produced. The conclusions an ADHD researcher might draw from this data depends on the other data and hypothesis that are most relevant to them. It could be used to show that catecholamines are the main neurotransmitters implicated in all human and animal executive function, and that it ameliorates ADHD symptoms through direct action on the requisite system. Conversely it could be claimed with this data that increased NE and DA in the brain only mask the symptoms of ADHD rather than dealing with some as-yet-unknown cause of the disorder. What exactly we can surmise about the etiology of a disorder in relation to psychopharmacological intervention is still far from clear, and this issue warrants critical consideration. In this section and the next we will see how the controversy over the meaning of drug effects has contributed to the reality of the ADHD.

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[14] The distinction between endogenous and exogenous factors of ADHD is due to meta-analysis ADHD research conducted by Dr. Joel Nigg. Also, while some studies explore the effects of environmental features such as maternal smoking or lead poisoning on ADHD patients, the "exogenous factors" I have in mind are different. Prenatal trauma or early childhood poisoning are still related to the endogenous features of patients because their effect is on the permanent material structure of brains. The environmental factors I am concerned with pertain to things like classroom architecture and temporal design as well as psychosocial contexts of the disorder.

The concepts of "executive function" and "attention" mark off capabilities that normal humans employ and the possibility of enhancing these capabilities through pharmacological means obscures the boundaries between legality and illegality within our culture.[15] These concepts are also partially developed in conjunction with the science of ADHD, which is itself entangled with psychostimulant medication, and therefore complicates the issue of how to proceed when considering how these capabilities should be theorized in relation to ADHD.

Considering the three main types of evidence[16] used to support the catecholamine hypothesis of ADHD (both individually and in conjunction) will help to demonstrate how each of them works to produce the etiological and clinical validities of the disorder. I contend that of the three types, the psychopharmacological evidence plays the most important role in supporting the catecholamine hypothesis. Meta-analysis papers published in ADHD catecholamine science show that psychopharmacological evidence has been emphasized most heavily, and provide much of the basis for neurological and genetic research. (Arnsten & Li, 2005) (Pliszka, 2005) An entire book and several other studies have focused on understanding pharmacological data in relation to the catecholamine hypothesis. (Solanto et al., 2001) (Pliszka, 2005) These works show not only the importance of this data to the catecholamine hypothesis, but also the ways in which neurological and genetic research have roots in psychopharmacological studies. The three forms of evidence are rarely studied in isolation, and reading research

[15] The issue of enhancement is discussed at greater length in section 3.
[16] Genetic, neurological, pharmacological

findings carefully provides some valuable insights into the grounds of the catecholamine hypothesis.

Many studies using or generating animal models of the disorder also employ pharmacological interventions in the context of experiments in order to reach scientifically significant conclusions about ADHD. Mice who have been administered a catecholamine-focused neurotoxin exhibit hyperactive symptoms as well as severe learning disabilities. This evidence alone would not provide particularly strong support for the catecholamine hypothesis, since the damage to the rodent brain as a result of the neurotoxin is severe. Only after these same mice are subsequently treated with methylphenidate or amphetamine to test drug responses do the results bear much relevance to ADHD. (Shaywitz, Klopper et al. 1977) (Luthman et al., 1994) These mice show reduction of post lesion learning deficits with the application of stimulants, which suggests that the deficits are "more attentional in nature than true learning deficits." (Ferguson 2001) The administration of the drugs is vital for differentiating learning deficits from strictly attentional deficits. Without the drugs, these studies would not present any significant data for the catecholamine hypothesis. The evidence that supports the catecholamine hypothesis depends on the assumption that the requisite psychostimulants directly affect only attentional systems, yet the truth of that assumption is precisely what is at issue in the catecholamine hypothesis.

Others types of animal models of attention are developed using genetic evidence, which is made possible by examining the results of pharmacological data.

Genetic studies of attention have been carried out using gene knockout mice, which are bred to lack dopamine transporter and or receptor genes. These knockout mice show hyperactive symptoms, which is enough to make them a relevant model for the catecholamine hypothesis. Researcher Michael Posner has noted that these gene knockout mice were first bred in response to results from pharmacological studies demonstrating the effects of stimulants on dopamine pathways. (Posner, 2004) While researchers discovering new possible programs of genetic study as a result of pharmacological evidence is not in itself problematic, subsequent genetic studies provided little evidence for the catecholamine hypothesis unless psychostimulants were administered to genetically altered mice as part of the experiments.

For example, within such studies dopamine transporter knockout mice were treated with methylphenidate and amphetamine compounds in order to reduce "learning impairments" associated with genetic alteration. In meta-analysis it has been concluded that "such learning impairments prevent the usefulness of this model." (Ferguson 2001) The animal and psychopharmacological models are often cited as different forms of evidence supporting the catecholamine hypothesis; however in some cases the psychopharmacological data actually provides the basis for the evidentiary usefulness of animal models.

Meta-analysis literature repeatedly demonstrates the importance of psychostimulant data for evidence of the catecholamine hypothesis. In reports on the history of the hypothesis the effects of psychostimulants are frequently noted as

one of the initial pieces of evidence that led scientists towards this view. One such example can be seen in the following paragraph:

> Satterfield and Dawson (1971) were among the first to propose that ADHD symptoms were caused by frontolimbic dysfunction. They suggested that weak frontal cortical inhibitory control over limbic functions might lead to ADHD. The success of stimulant medications, and animal models implicating dopamine pathways were taken as support for this model. (Faraone & Biederman, 1998)

This early permutation of the catecholamine hypothesis is grounded in the success of psychostimulants. Some animal models cited to support this view take the form of mice and rats being administered psychostimulants, and so are actually better understood as psychopharmacological data. Other animal models are created using rodents and monkeys who have brain lesions that damage their frontal cortices. Hyperactive behavior amongst these test subjects, while consistent with the view that executive function occurs in frontal cortices, does not provide the same degree of specificity as the pharmacological data. The pharmacological data provides an essential ground for many of the animal models, and thus should be seen as the more important form of evidence.

Other histories of the catecholamine hypothesis credit different scientists with initial observations about catecholamine dysfunction, yet still cite the psychopharmacological data as paramount.

> Basic pharmacological research established that the stimulant drugs augment central norepinephrine and dopamine. Reasoning in part from these data, Paul Wender proposed, in an influential book entitled *Minimal Brain Dysfunction* published in 1971, that alterations in catecholamine neurotransmission mediate the therapeutic effects of the stimulants in ADHD. (Solanto et al., 2001)

This account also demonstrates that the main body of evidence showing that ADHD is caused by catecholamine dysfunction is the success of psychostimulant medication for the treatment of the disorder. The animal models that have been invoked in

other accounts are not clearly separable from the pharmacological data, and in this account the pharmacological data is of paramount importance.

The view that the catecholamine hypothesis emerges from the success of stimulant medication is also shown in the current literature educating patients and parents of patients on the causes of the disorder:

> The effectiveness of stimulant medication, along with animal models of hyperactivity, also point to catecholamine disruption as at least one source of ADHD brain dysfunction. (NRCA, 2011)

The education of the public as to the etiology of ADHD centers on the success of psychostimulant medications. Animal models provide sources of evidence that are difficult to differentiate because the global effects of altered brains and genetic codes create severe learning deficits beyond those that can be attributed to attention in the absence psychopharmacological data. The tremendous success of treating ADHD patients with psychostimulants is the central justification for the view that the ADHD brain lacks the chemicals promoted by those psychostimulants. This view in turn justifies the further administration of psychostimulants and stabilizes the apparatus of measurement, thus promoting the validity of the disorder.

Although cited less frequently as evidence for the catecholamine hypothesis, neuroimaging studies also produce important data for the theory. However, much like animal modeling studies, the strongest evidence of these studies comes from the measurement of stimulant effects on the brain. One meta-analysis review of neuroimaging studies concluded that most PET and fMRI studies of catecholamine dysfunction did not produce consistent data. However, the review did find that the

one stable piece of evidence from neuroimaging studies centered on drug effects. The review states that:

> The finding of no significant difference between the ADHD and normal control groups in total brain volume suggests that there may be a neurodevelopmental process that differs in children with and without ADHD. Methylphenidate and other stimulants may enhance functioning in the left hemisphere by inhibiting the over-response of the right hemisphere in attentional processes. (Semrud-Clikeman & Pliszka, 2005)

Neuroimaging studies have failed to show consistent evidence for the catecholamine hypothesis outside the context of pharmacological intervention. Without methylphenidate (Ritalin) and dextroamphetamine (Adderall), these neuroimaging studies would not provide any statistically significant support for the catecholamine hypothesis. This fact once again demonstrates the dependence of the theory on psychostimulant intervention.

Scientists recognize that the significant effect of psychostimulants on the catecholamine system allows for a special kind of contribution to psychiatric research.

> The catecholamines norepinephrine (NE) and dopamine (DA) have been hypothesized to be involved in a number of psychiatric disorders, primarily because a wide range of psychotropic medications affect these neurotransmitters. (Pliszka, 2001)

The visible effects of stimulants on executive functioning produce some of the most valuable observational data available to psychiatric researchers. This class of drugs produces a measurable effect on all humans and many animals, and thus provides an ideal tool for psychiatric measurement. Reasoning in this mode produces a definition of the disorder that emerges in relation to the pharmacological intervention. As Lakoff says in his book *Pharmaceutical Reason* "Illness comes gradually to be defined in terms of that to which it 'responds.'" (Lakoff 2005) This

"strategic logic" that Lakoff calls "pharmaceutical reason" has played the most dominant role in producing the reality of ADHD. The reality of the disorder has been at least partially determined by this mode of reasoning. Going deeper into the history of stimulant medication, it is easier to see how this "strategic logic" is being carried out.

Psychopharmacological data underpins a large amount of research into the science of attention and executive function in general. Genetic understanding of attention and attention deficits is as least partially based on the success of pharmacological data. In introducing one section of his volume *Cognitive Neuroscience of Attention* Michael Posner notes that "*pharmacological results* have made it possible to search economically for candidate genes relating specifically to the function of specific attentional networks." (Posner 2004) The presence in humans of certain alleles of genes implicated in dopaminurgic functioning (DRD4, etc.) are shown to relate to individual differences in attentional capability. What is interesting about this constellation of genetic evidence is that it has emerged in the context of psychiatric pharmacological research. While the diagnostic category of ADHD is only as old as the current edition of the *Diagnostic and Statistical Manual of Mental Disorders*, psychostimulants have been used to treat patients of ADHD's pathological predecessors (minimal brain dysfunction, hyperkinetic reaction of childhood, ADD) for decades. The practices by which certain genes have been implicated in the expression of ADHD were made possible by exploring the effects of psychostimulants on hyperactive patients and normal controls. The boundary

between pharmacological and genetic data has been enacted despite the fact that the two experimental programs are intimately linked; genetic evidence would be far more scant without the successes of pharmacological treatments.

Just as animal models for ADHD are entangled with pharmacological data, so the animal models which have been produced for studying attention in general are grounded in the same genetic-psychopharmacological emergence. Gene knockout mice (mice that have had certain parts of their genome altered to remove a particular gene or sequence) have expressed hyperactive behavior after having their DRD4 gene removed. (Grandy, 2004) This evidence provides support for the claim that this receptor is implicated in attention, but it similarly would not have been possible without the initial pharmacological results produced by research into the pathological predecessors of ADHD. The fact that scientists have been looking at genes associated with catecholamines since before ADHD existed as a diagnostic category is significant for understanding how our notion of "attention" has emerged alongside our understanding of "attention deficits." The availability and measurable effects of dextroamphetamine and methylphenidate on humans and animals has helped this domain of knowledge production to become successful. If these chemicals were not available or were used differently by clinicians and researchers, the catecholamine hypothesis would not be supported by the same wealth of evidence, and may never have been proposed. The success of the catecholamine hypothesis is contingent upon the historical availability psychostimulant compounds.

ADHD researcher Steven Faraone has noted that our human genetic studies would not have been possible without psychostimulant medication.

> "many of the genes implicated are directly related to the medications that work, for example methylphenidate and amphetamine both work on the dopamine transporter, atomoxetine works on the norepinephrine transporter. Of course that's why we chose the genes to study so it's not simply a coincidence. We chose those candidates because we knew they were involved in ADHD relevant systems." (Faraone, 2007)

The fact that genetic evidence is made possible by the effects of medications demonstrates the importance of medication in other areas of ADHD research. Other ADHD researchers have challenged the conclusiveness of genetic data noting that "The most robust finding in ADHD is its association with a polymorphism of the D4 receptor gene. The 7-repeat allele of the D4 receptor is more frequent in ADHD patients (23%) than in healthy subjects (17%)." (Gonon, 2009) The fact that the relevant mutation occurs in only 23% of ADHD patients as opposed to %17 of healthy controls suggests that these candidate genes would have been very difficult to find without the support of drug evidence. This situation further demonstrates the importance of medications for the catecholamine hypothesis.

In meta-analysis Joel Nigg has noted that there is still significant progress to be made before any single neurochemical hypothesis of ADHD can be confirmed. While catecholamines are clearly implicated, "other neurotransmitters, notably y-aminobutyric acid, are likely to prove equally important." (Nigg, 2005) Yet the role of these other neurotransmitters is difficult to measure due to the lack of effective pharmacological alteration of these chemicals.

Lakoff's own specific examination of ADHD precedes his larger research into "pharmaceutical reason," and thus does not go as deeply into foregrounding these

connections. Lakoff traces the initial observation of the therapeutic, calming effects of psychostimulants on unruly school children to Charles Bradley in 1937, and Bradley is widely accepted to have been the first to administer stimulants to children in the name of therapy. (Solanto et al., 2001) Examining Bradley's work giving Benzedrine to students in a "Rhode Island home for children with behavioral problems" Lakoff notes that Bradley "speculated that underactive centers of inhibition in the brain were stimulated by the drug, which 'reduced activity through increased voluntary control.'" (Lakoff 2000) In fact it turns out that Bradley's speculation fairly accurately describes the current state of the catecholamine hypothesis. The underactive centers of inhibition (now believed to be the prefrontal cortices) were stimulated by the drug (through catecholamine promotion in the synapses) which reduced activity through increased voluntary control (i.e. it allows to patient to control their attention and focus on their school tasks). The pharmaceutical logic upon which the catecholamine hypothesis is founded existed even in 1937.

Current ADHD researchers have noted "In hindsight, Bradley's observations concerning the effects of amphetamine treatment on primary symptoms, academic and social functioning, pharmacodynamics, and common side effects were remarkably astute." (Solanto et al., 2001) The fact that ADHD science has emerged along the currents of Bradley's work with psychostimulants should allow us to consider his astuteness in a particular light. An effective drug discovery makes whole new edifices and practices of knowledge production possible. Bradley was

also astute in another way, which is not mentioned by ADHD researchers. The last line of his publication warns that "any indiscriminate use of Benzedrine to produce symptomatic relief might well mask reactions of etiological significance which should in every case receive adequate attention." (Bradley, 1937) The psychopharmacological data has now become the most etiologically significant data available. While Benzedrine is no longer used in treatment, methylphenidate and dextroamphetamine provide powerful analogues with which to drive research. These chemicals have become a mainstay of ADHD research, and as such other lines of research into other possible etiological explanations have been underfunded.

The phenomenon of ADHD has never existed without its association to psychostimulant research. Even the oldest DSM pathological predecessor category, minimal brain dysfunction, was not officially codified at the time of Bradley's research. Bradley's original study was conducted by administering Benzedrine twenty-one boys and nine girls all with

> behavior disorders (that) were severe enough to have warranted hospitalization, but varied considerably. They ranged from specific educational disabilities, with secondarily disturbed school behavior, to the retiring schizoid child on the one hand and the aggressive, egocentric epileptic child on the other. The patients' intelligence was in general quite within the so-called "normal" range. (Bradley 1937)

The diagnostic categories that Bradley's subjects were labeled with bear little resemblance to the ones that currently exist in the DSM. Bradley's description says that all the students in the study are considered to have *normal intelligence* but had other problems connected with their behavior that hindered their educational progress. While the research has developed along with the emergence of new and specialized measurement techniques, the purpose of the apparatus of measurement

remains quite similar to that which Bradley's research was engaged with; how to allow children of normal intelligence to behave in school in such a way that they can perform according to scholastic norms appropriate to their requisite level of intelligence. The multifarious and (to us) unrecognizable disorders that Bradley's subjects were diagnosed with are unlikely to have been explained by the same chemical deficiency in the brain, yet over half of them demonstrated the desired effect; increased school performance. The development of science in this area has responded to the need to allow children of normal intelligence to perform normally, and the name for this phenomenon that is currently accepted is ADHD.

The "school performance" of these students was measured by the observations of teachers who "agreed that a great increase of interest in school material was noted immediately. There appeared a definitive 'drive' to accomplish as much as possible during the school period, and often to spend extra time completing additional work" (Bradley 1937). A typical response to the administration of stimulants, it becomes apparent that this kind of cognitive enhancement can be elicited from non-pathological patients as well.

Both Lakoff and Ilina Singh agree that the re-emergence of biological psychiatry in child therapy helped to bolster the stability of the diagnosis. (Lakoff 2000) (Singh, 2009) In particular, the view that biological problems could be treated with material interventions was especially consistent with this theoretical movement. Biological psychiatry has historically competed with psychoanalysis and other forms of psychiatric practice for acceptance. Lakoff and Singh both suggest

that the success of biological psychiatry within the area child psychiatry is due in large part to the success of treating childhood scholastic disorders with psychostimulants.

In order to better understand how ADHD science has been produced, it is helpful to view the phenomenon of ADHD and indistinct from the phenomenon of psychostimulant medication. Because these compounds are prescribed to ADHD patients as a treatment, medical science does not tend to view them as a fundamental feature of our knowledge making practices surrounding the disorder. Recognizing that a boundary that cuts stimulant medication *out* of the phenomenon has been enacted by the human agential practices is an insight made possible by considerations in Karen Barad's *Meeting the Universe Halfway*. (Barad, 2007) One of the chief values of examining scientific practices by examining apparatuses of measurement is that it becomes possible to see the boundary enactments[17] that cut various features in or out of experimental configurations. These boundary enactments allow data about a phenomenon to be produced by fixing phenomena in certain material and discursive ways.

[17] Barad's view holds that "Bodies are not objects with inherent boundaries and properties; they are material discursive phenomena." (Barad 2007) Because on this view "matter and meaning are mutually articulated" we can understand the phenomenon under investigation as one that takes on its features because of the engagements of agents within that phenomenon. The material and discursive features that ADHD expresses emerge out of the way that scientists and other actors perform acts of measurement employing certain standards, assumptions and experimental configurations to the exclusion of others. The way that ADHD is understood emerges out of the way that it gets produced within the world. By recognizing, as Niels Bohr did, that "we are part of the phenomenon we seek to understand" we can better grasp the way in which ADHD has come to be part of the world we take part in producing.

The data that is produced in any experiment is determined by the experimental configuration, and so studying the meaning of data in relation to experimental configurations is paramount. Because psychostimulants have been preferentially excised from the phenomenon, their role in the production of the catecholamine hypothesis has been virtually ignored by research science. This boundary enactment takes place in a context where psychiatrists are attempting to achieve a particular type of scientific certainty that is mistakenly attributed to other sciences such as internal medicine and physics. Barad's work in *Meeting the Universe Halfway* shows that data produced in science experiments must also be understood in the context of experimental configurations. Insofar as matter and meaning are co-constituting processes, it would be valuable for ADHD researchers to consider the catecholamine hypothesis and other conclusions about ADHD by exploring the relationship between data and experimental configurations.

One study entitled "Mining the Meaning of the MTA[18]" discusses ways that the experimental data comparing behavioral and psychopharmacological intervention might be more effectively produced. While this study does focus on experimental design, it does not adequately consider the crucial role of psychostimulants within the phenomenon, instead discussing the issues surrounding the practical administration of behavioral intervention. In the abstract, the publication expresses its goals to respond to six issues raised by competing interpreters of the data.

[18] The MTA is the massive "Multimodal Treatment Analysis" study carried out to compare the effectiveness of various ADHD treatments.

> "The commentary questioned the design and analysis of the MTA in terms of (1) the empirical criteria for selection of components of behavioral (Beh) intervention, (2) the effectiveness of the Beh intervention, (3) the methods for analyses at the group and individual level, (4) implications of the MTA findings for clinical practice, (5) the role of genetics in response to treatment, and (6) the lack of a nontreatment control group." (Swanson et al., 2002)

While each of these factors is important to understanding the meaning of the conclusions reached by the MTA study, none of them effectively address the issue of the role of stimulant medication within the phenomenon. The concern is clearly focused on the effectiveness of the behavioral treatment and the ways it was found to be inadequate. These commentators have failed to consider the possibility that it was actually the measurement practices surrounding psychopharmacological intervention that skewed results in favor of that treatment.

The researchers who defend the results of the MTA in this study invite people to use their data to propose new experiments, but they do not consider the possibility of collaboratively discussing experimental configurations and producing new ones. The adversarial tone of the document invites competition rather that collaboration, leading to a type of critical consideration that focuses on errors rather than embedded assumptions. Researchers who disagree as to the meaning of data regarding effective treatments for ADHD might discuss experimental configurations without turning to accusations of bias, instead collaboratively proposing new experimental designs that critically consider boundary making practices within experimentation. If these matters could be discussed in a way that the members of the public concerned with the ADHD diagnosis could participate, the scientific community also might have better success in transmitting their message regarding the validity of the diagnosis. To know that these conclusions have been reached

after a critically robust conversation had taken place would help those who doubt the validity of the diagnosis to see that the consensus that is ultimately produced is hard won and thus worthy of respect.

Scientific knowledge of the etiology of ADHD is contingent upon our pharmacological pursuits, and if this information could be transmitted to the public effectively, it might help people to make more informed decisions about how to best negotiate treatment practices. Because the etiology and clinical validities of mental disorders are entangled, this evidence shows also that the clinical validity of the diagnosis emerges at least partially from our pharmacological practices. However, as Lakoff says in his ADHD paper "to assert that the disorder is contingent is not to dismiss it as false." (Lakoff 2000) Rather, it should be understood that the diagnostic category of ADHD emerges in response to the creation of an apparatus in which the needs of various cultural groups are inherent. The diagnosis persists only insofar as a culture needs to achieve the norms of education that are created within it. If the public were educated according to this view, the diagnosis and its treatments could be more widely accepted, but different treatment practices could be explored and developed more effectively as well.

The ADHD diagnosis lies at the confluence of many societal needs, and has been scientifically bolstered by the availability of psychopharmacological interventions. What I have tried to show here is that the apparatus producing the etiological validity of ADHD includes the pharmacological interventions associated with it in non-trivial ways. In the next section I will explore more carefully how the

clinical validity of the ADHD diagnosis is connected to psychopharmacological interventions. This analysis will take into account the ways that the normalization of academic achievement in the United States has emerged in connection with the validity of ADHD. In section 4 I consider possibilities for remediation of our culture that would allow for new norms to be produced and affirmed.

4.) The clinical validity of ADHD and the issue of norms

In order to explore more fully how the validity of ADHD has been connected to psychopharmacological intervention, it will be helpful to consider some ideas brought to the fore by Dan Stein's *Philosophy of Psychopharmacology*. Stein makes some specific remarks about ADHD in relation to pharmacological interventions, and they can be used to elucidate the state of affairs concerning the clinical validity of the diagnosis. Stein suggests that the fact that psychostimulants work on non-ADHD control subjects raises "questions about the validity of ADHD as a disorder." (Dan J. Stein, 2008) Somewhat confusingly, this claim comes in a paragraph where Stein also suggests that "when a medication is effective, we cannot necessarily deduce a great deal about the mechanisms involved in the relevant disorder." (Stein 2008) His warning against surmising the mechanisms and etiology of a mental disorder based on the functions of psychotropic compounds is a valuable one, particularly in light of Lakoff's observations about the tendency of psychiatric practice towards a pharmacological definition of mental disorders. In *Pharmaceutical Reason* Lakoff suggests that if current trends continue, disorders will not be

categorized by standard observational headings, but instead by the drug-response profiles associated with them.[19] (Lakoff 2005)

Even with our most up-to-date brain scan technology, much is not understood about how drugs actually affect brain-mind states, and yet an increasing amount of confidence is placed in the ability of drug-response profiles to accurately determine the correctness of a diagnosis. The belief that the effectiveness of a particular drug-compound demonstrates reliable facts about brain states is a problematic leap in psychiatric practice, and Stein does excellent work to criticize this tendency. In personal communication, Stein has clarified his position by saying the following:

> SSRIs are effective in (treating) depression, from which some deduce that serotonin is involved in the cause of depression. But it turns out that that is probably a step too far. First, SSRIs do act on the serotonin system, but via that they affect multiple other molecules, so their end effects are not specific. Second, it turns out that there really isn't a whole lot of evidence (if any) that serotonin is a single cause of depression. Indeed, any one of the ultimate places the SSRIs act on could contribute to depression. (Dan J. Stein, 2011)

This series of statements is valuable for thinking through the issues of ADHD in relation to psychopharmacological intervention more carefully. While we should not surmise too much about the etiology of a disorder based on drug efficacy observations, there is still a feature of the clinical validity of a diagnosis that relates to observed drug effect. Stein has agreed with the view that the etiology and clinical validity of mental disorders are entangled, and that is the most basic lesson we can draw from these pieces of evidence about ADHD. The story we tell about the causes

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[19] Lakoff says explicitly that if current trends in psychology continue "the broad categories that govern psychiatric practice might be broken down in terms of medication response, so that diagnostic questions would appear no longer as 'is it bipolar disorder or schizophrenia?' but as 'is it a lithium or an olanzapine response profile'" (Lakoff 2005)

of the disorder is going to exist in relation to the stability of the diagnosis. Going further into understanding exactly how this relationship works requires an examination of the clinical practices that arbitrate the validity of a diagnosis.

Realizing that drug effectiveness does not necessarily tell us much about etiology should also help us to recognize that the clinical validity of a disorder is not based solely on what groups of people experience beneficial effects as a result of psychopharmacological compounds. Nevertheless, these considerations do come into play as concerned groups negotiate the clinical validity of a diagnostic category.

One strategic feature of "pharmaceutical reason" employed in the history of ADHD research was the "paradoxical effect" logic employed by scientists in the 1970s. During this era it was believed that psychostimulant medications had a "paradoxical effect" on ADHD patients. This meant that psychostimulants would help ADHD patients to focus where it made non-ADHD controls less attentive. As one group describes it "a reduction in hyperactive and inattentive symptoms following stimulant administration was often regarded as post hoc confirmation of the ADHD diagnosis." (Solanto et al., 2001)[20] The view held that stimulants should stimulate healthy controls into hyperactivity where they allowed hyperactive patients to function normally.

Scientists now agree that all normal children and adults experience increased attention and cognitive ability when psychostimulants are administered. (Rapoport et al., 1978) These experiences closely resemble those that Bradley observed in 1937.

[20] Scientists even went so far as to produce animal models demonstrating the "paradoxical effect." (Shaywitz, Klopper, & Gordon, 1977)

It is not hard to see why the collapse of the "paradoxical effect" logic might lead Stein and others to question the validity of the disorder. The paradoxical effect produced a useful narrative on which to rest claims about the action of psychostimulants on ADHD patients. Despite the fact that this initial justification for the administration of psychostimulant compounds to patients disappears from scientific view, other research programs had already appeared to fill this gap. If research toward the catecholamine hypothesis had not been already underway, loss of the paradoxical effect might have irreparably damaged the link between ADHD and psychostimulant medication. This is because, as Ilina Singh has put it, the fact that psychostimulants increase cognitive abilities in everyone shows that "to some degree, the medications enhance performance rather than treating the specific psychopathology." (Singh 2008)

The distinction between "enhancement" vs. "treatment" has become an increasingly visible issue in dialogues about ADHD in our culture. Our educational standards produce a norm through measurement of intelligence. Pinel's development of the IQ test is an extremely well recognized psychological measurement tool due in part to its longstanding tenure. The expectation that children conform to their measured degree of intelligence is a foundation of our scholastic formation. Because children with ADHD usually fall within normal intelligence ranges, just as Bradley's subjects did, administration of psychostimulants to help them perform at their requisite intelligence level is considered a treatment rather than an enhancement. If the IQ test developed by

Pinel and widely used in American public school systems were altered or abandoned, this justification might no longer exist.[21] This line of thinking might bolster Stein's concerns regarding validity of the diagnosis based on the loss of the paradoxical effect reasoning. If school performance expectations were not based on the measurement of IQ, the justification for such treatment would be damaged. Children would not have to match the standard predicted by their IQ scores, and would be allowed to perform according to some other norm.

Despite the loss of the "paradoxical effect" reasoning the clinical validity of the ADHD diagnosis has not suffered significantly. While it was probably a helpful piece of support for the catecholamine hypothesis during its incipient stages, support from the "paradoxical effect" claim has proven unnecessary for its long-term success. What remains, however, is a problem about how to differentiate treatment from enhancement. The question about whether or not to administer psychostimulants to a child is one that parents are faced with on a daily basis. Because these compounds carry potentially serious side effects, parents must think very carefully about what degree their child's academic performance is worth damage to their physical health.

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[21] Howard Gardner has proposed a theory of multiple intelligences which might alter the current norms that ground our expectations of academic performance. In his book *Frames of Mind* he says "In the heyday of the psychometric and behaviorist eras, it was generally believed that intelligence was a single entity that was inherited; and that human beings - initially a blank slate - could be trained to learn anything, provided that it was presented in an appropriate way. Nowadays an increasing number of researchers believe precisely the opposite; that there exists a multitude of intelligences, quite independent of each other; that each intelligence has its own strengths and constraints; that the mind is far from unencumbered at birth; and that it is unexpectedly difficult to teach things that go against early 'naive' theories of that challenge the natural lines of force within an intelligence and its matching domains." (Gardner, 1983)

The percentage of children diagnosed with ADHD has increased with time. Most estimates currently range from 5% of children to 4%-8% of children. Another way to view this data is to examine the rates of psychostimulant prescription in various countries around the world (see figure 1 in appendix 1). In 19 of the 20 countries where measurements were taken, psychostimulant prescription increased significantly over the four year period. Allen Frances, the psychiatrist who oversaw the publication of the DSM -IV has said he thinks the current diagnostic criteria for ADHD contained in that edition of the DSM make it too easy for people to obtain the diagnosis. In a very public debate both Frances and Robert Spitzer, lead editor of the DSM-III have voiced concerns about the development of the DSM – 5 and what features of diagnosis it will produce. In a recent radio interview Frances said

> I think we (the DSM –IV Taskforce) helped to trigger three false epidemics, one for autistic disorder that you mentioned, another for the childhood diagnosis of bipolar disorder, and the third for the wild over-diagnosis of attention deficit disorder.
> …with attention deficit disorder as an example, the prescription of stimulants has exploded. And what's happened is that, often, these are given, not for a mental disorder, but for performance enhancement. And getting a diagnosis of attention deficit disorder allows you to get that stimulant treatment, which, for many people, may not be for a mental disorder, but may just be so that they can do better in their everyday lives. Thirty percent of college students use stimulants to do better at school. (Frances, 2010)

The concern here about over-diagnosis and the line between treatment and cognitive enhancement is palpable. It has been suggested that lax criteria for the disorder have contributed more than any other factor to the tremendous prevalence of the diagnosis and the abundance of psychostimulant medication in the United States. Ilina Singh notes that "Studies have found that a diagnosis of ADHD is 3–4 times

more likely if DSM-IV criteria are used than if ICD-10 criteria are used."[22] (Singh 2008)

So, to add to the "treatment" vs. "enhancement" debate, there is a connected issue about over diagnosis. One proposal for dealing with this issue is to change not only the diagnostic criteria for ADHD in the DSM, but instead to shift the entire process of psychiatric diagnosis from a categorical to a dimensional model. Despite the fact that the ICD and DSM produce different rates of diagnosis, they still employ techniques of categorical assessment, which categorize patients based on the frequency and intensity of a list of symptoms (see appendix 2). As opposed to categorical assessment, dimensional assessment employs a battery of tests to determine not only the presence but the severity of a psychiatric diagnosis. The goal of instituting this technique of measurement would be to create a severity spectrum for ADHD that would more accurately determine what type of treatment, pharmacological or otherwise, is appropriate for a particular individual. (Singh 2008)

The proposal to institute "dimensional assessment" as part of all diagnostic evaluations for ADHD carries several issues. While many therapists believe that a full neuropsychological battery of tests is needed to properly diagnose ADHD, this practice is not currently required to diagnose ADHD. This neuropsychological battery might provide the grounds on which to produce a dimensional diagnosis of ADHD. If this diagnostic practice were adopted in the context of ADHD a distinction between the sick and the merely suffering (and thus between treatment

[22] The ICD-10 is used by the World Health Organization to diagnose all medical disorders including mental disorders. It is the reference manual used by European psychiatrists.

and enhancement) could emerge and gain statistical validity. The notion that subjects of study would fall somewhere within a dimensional model of the disorder would also carry various other treatment recommendations that might include non-stimulant therapies. However, while the move to dimensional analysis looks promising for proposing more effective treatments, there are significant hindrances to it being adopted wholesale by the APA. The tests have not been developed long enough to ensure their accuracy and statistical validity.

> Michael First, a Columbia University psychiatrist who headed up the DSM-5's Prelude Project to solicit feedback before the revision, believes that implementing dimensional assessment right now is a tremendous mistake. The tests, he says, are nowhere near ready for use; while some of them have a long track record, "it seems that many of them were made up by the work groups" without any real-world validation. Bad tests could be disastrous not just for the profession, which would erect its diagnostic regime on a shaky foundation, but also for patients: If the tests have been sanctioned in the DSM, insurance companies could use them to cut off coverage for patients deemed not sick enough. (Greeneberg 2010)

If these tests were adopted prematurely, the consequences for patients and diagnosing clinicians could be seriously problematic. While dimensional assessment promises more accuracy once the technique is properly developed, it will take much more science to institute the practice in way that can be considered responsible. In addition to these concerns, questions emerge about how the cut-points between different sections of the spectrum might be affected by the availability of psychostimulants. The pharmaceutical logic described by Lakoff in *Pharmaceutical Reason* will be part of the production of the dimensional model, and unless the role of psychopharmacological interventions in measurement is critically examined by scientists, the issue of treatment and enhancement is likely to go unresolved. Stein provides a useful example from the history of pharmacology that may help to demonstrate how cut-points between normal and pathological can be influenced by

pharmacological intervention. Stein notes that the threshold for condition of "high-cholesterol" has changed as cholesterol reducing agents have become more readily available. Stein explains

> "Prior to the advent of cholesterol-lowering agents, rather high levels of cholesterol were defined as "normal." After the advent of studies which showed that cholestatins were useful in lowering cholesterol and medical morbidity, the cut-off for "normal" cholesterol was lowered. Furthermore, with lowered purchase cost of older cholestatins, and studies demonstrating that treatment of relatively low levels of cholesterol was cost-effective in preventing medical morbitidy, the cut-off was again lowered." (Stein 2008)

This data is explored in greater detail in Jeremy Greene's book *Prescribing by Numbers*. (Greene, 2007) In the book, Greene shows how medical and public concern about high cholesterol has changed over the years in relation to the availability of cholesterol reducing drugs. Greene claims that "the fall and rise of cholesterol as a risk factor were intimately tied to the performance and promotion of discrete drug entities." (Greene 2007) If a dimensional model of ADHD gets adopted as the new form of diagnosis for ADHD, similar effects could emerge for the diagnosis of ADHD. The historical relationship between stimulant medication the measurement of ADHD is strong enough to make this extremely likely.

Alternatives to the wholesale adoption of the dimensional model exist. Joel Nigg suggests that adding neuropsychological findings to the behavioral diagnostic criteria of ADHD would be valuable assuaging public concerns about the validity of the diagnosis. (Nigg 2005) This is because a more robust set of measures is more likely to convince people of the scientific accuracy of the diagnosis. However, Nigg also points out that some neurological theories of the disorder are "closely related to psychopharmacology," thus demonstrating the proximity of these two measurement practices. (Nigg 2005) The neurological effects of pharmacological interventions are

clear both ADHD and non-ADHD patients, and there is a concern that any cogntive discrepancy in a subject might warrant medication. It seems adding features of the dimensional model could lead to a clearer decision about when medication is necessary, but it might also simply justify the use of "cosmetic psychopharmacology" (Kramer, 1993) amongst individuals who have the slightest neurological expression of symptoms.

The issue of non-diagnosed college students using psychostimulants to do better in school is related to the availability of the chemicals through their ADHD diagnosed classmates, and the increased availability of controlled substances is a major public concern. If the number and volume of psychostimulant prescriptions were reduced amongst college students by instituting a dimensional model that refused or restricted high volume stimulant medication to more people, it could help reverse this trend. Alternatively, if our systems of measurement for scholastic achievement were continuous rather than episodic, the situations where psychostimulants seemed necessary for non-ADHD students might be reduced. Studies have shown that illicit psychostimulant use is focused mainly around periods of evaluation, (Hall, Irwin, Bowman, Frankenberger, & Jewett, 2005) (Carroll, McLaughlin, & Blake, 2006) such as exams or due dates for long essays. Such episodic measurement, in which the grade given to a student depends on their performance on just a few assignments leads students to "cram for an exam" or "pull an all-nighter" with chemical support. A redesign of the measurements systems not only for the diagnosis, but also for academic success might allow for a clearer

boundary between treatment and enhancement. The public perception of the disorder is damaged partially because stimulants are used for enhancement so abundantly. The clinical validity of the disorder is harmed because the treatment which supports it so effectively can be re-appropriated for illicit and cosmetic use.

Another issue that causes a break between the medical and public perceptions of the disorder is an issue of authenticity with regard to childhood ADHD patients. There is a public perception that many ADHD symptoms are normal childhood behaviors, and need not be medicated outside the context of school. The difficulty of distinguishing between normal childhood behaviors and ADHD behaviors is one that especially affects the parents of ADHD children. Ilina Singh has developed a study focusing on the daily decisions made by parents of ADHD children about when psychostimulants should be administered. Because most psychostimulants tend to come in a short acting form, ADHD school children usually need to be medicated three times a day; once after breakfast, once after lunch, and once in the afternoon to get through the homework period. (Singh 2005) Because the medication needs to be administered so frequently, the parents of ADHD children are required to make a decision each time they give their child a pill. Although the side effects of these psychostimulants were not recognized officially by the FDA until 2007[23], the cognitive effects of stimulant medication on children is noticeable and thus raises concerns about its value in different contexts.

[23] Since February 2007 all FDA-approved drug treatments for ADHD (methylphenidate, dextroamphetamine and atomoxetine) have carried a warning that their use can involve risk for cardiovascular effects, growth suppression and the development of psychosis or other psychiatric conditions. Rare cases of sudden death have been reported among children using

In this study Singh interviewed several groups of parents, separating fathers from mothers during interviews. The study produced several important findings; mothers and fathers feel differently about their children's disorders, their justifications for medicating them emerge in different contexts, and (perhaps most importantly), the concept of authenticity grounds the decision about whether to medicate the child at a particular time. (Singh 2005)

Focusing on this third issue, the most difficult decision seems to come on weekends, when the child is not expected to perform school tasks. The question that emerges for parents is whether they are making their child more or less normal by giving them medication outside of a school context. Which child is the *real* child; the one under the influence of psychostimulants, or the one that is free of that influence? In summary, what Singh concludes is that when parents medicate their children during the weekdays, allowing them to perform well in school, the medication is allowing the child to be his or her "*real self*." One mother says "The real Joseph is the one on medication. That's the real one. I know that because he doesn't like what he is not on medication. So that can't be the real one. The real one is the one he is most comfortable being." (Singh 2005)

The narrative that Singh heard repeated by parents emerges in the context of the view of medical science, which says that the ADHD patient suffers from a medical disorder which must be treated at all times. One commentator on Singh's paper, speaking from the position of a medical practitioner, has suggested that the

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stimulant medications for ADHD. The FDA warns that the use of these medications by children with heart conditions should be avoided or undertaken with great caution (Singh 2008 pg 960)

justification for medicating ADHD patients emerges from the narrative of "scientific psychiatry," in which the stigma of mental disorders is removed by placing them on the same level as any other medical disorder. (Appelbaum, 2005) In the same vein, "Ritalin is likened to insulin for diabetes, or to glasses for myopia" e.g. (Hallowell & Ratey, 1994) in clinical contexts, producing a relationship of identity between medical and mental disorders. According to this narrative, psychostimulants are a necessary treatment for a disorder which hinders the patient *at all times*. In this context, the real patient is the one who has his or her symptoms mitigated by the medical intervention, thus allowing their true, un-disordered personality to emerge.

Conversely, during the weekend parents of ADHD patients will often give their children "drug holidays," sometimes at the recommendation of the prescribing pediatrician. This decision may be because of the risks associated with psychostimulant medications, however Singh also found a competing narrative of authenticity at play in this decision making process. In another interview, the mother of an ADHD child claims "Why should we drug him on the weekend? That's who Stuart is. If he wants to be off the walls, why not?" (Singh 2005) Here the authentic child is seen as the one free of the influence of psychotropic medication. The narrative that is being expressed to produce this notion of authenticity is clearly not the same as the one that emerges under the dictates of scientific psychiatry. Singh suggests that ideas about typical boyhood behavior (particularly ideas held by fathers because their son's behaviors reflect their own, un-medicated boyhood behavior) might help to produce this view. This narrative is very troubling for the

proponent of scientific psychiatric narrative because it "reveals the apparent triumph of the narrative of scientific psychiatry to be strikingly incomplete." (Appelbaum 2005)

The reality of ADHD patients, even down to their core identity, is produced on a day to day basis by the presence of psychostimulant medication. The parents' decision making practices emerge out of larger cultural contexts which produce particular habits of reasoning. Parents instantiate these reasoning practices each time they make a decision, and each time a different reasoning practice is employed, a different reality is produced. When the narrative and reasoning practices associated with scientific psychiatry is employed, the authentic patient is the one who is drugged. Returning to the similes of insulin for diabetes or glasses for myopia Singh says that "mothers used both these similes to justify Ritalin treatment for their sons. But for these similes to mean something, mothers have to hold to *a logic* in which a child's behavior is a disrupted or disordered part of the body that is at the same time not part of the child's real self." (emphasis added) (Singh 2005) It is precisely the logic employed by the mothers that takes part in the production of various realities for ADHD patients. I take the term "logic" here to be used much in the same way as Andrew Lakoff uses it when he discussed the emergence of "pharmaceutical reason." Lakoff says, referring to the cultural edifice that includes diagnostic standardization, clinical protocols, drug development and molecular genetics that "this constellation of heterogeneous elements is joined together by a strategic logic I call 'pharmaceutical reason.'" (Lakoff 2005) Indeed, the form of

reasoning referred to as the scientific psychiatric view maps extremely well onto Lakoff's notion of pharmaceutical reason. This form of reasoning emerges and gains success because it falls at the intersection of the needs of two different groups. One is the need expressed by parents of ADHD patients to escape the stigma sometimes associated with raising a child who does not behave. It also helps parents to guide their children into alignment with the norms and expectations of school performance. Additionally, Appelbaum has expressed a state of affairs in which psychiatrists work to achieve a medical-scientific standard that mirrors other medical sciences. It is this set of needs, in Appelbaum's view, that has led psychopharmacology to "usurp psychotherapy as the primary instrument of treatment." (Appelbaum, 2005)

When the need for school children to achieve according to scholastic standards or behave in certain ways is removed in non-school contexts, parents no longer adhere to the logic that says the disorder is on par with diabetes or myopia. The "authentic child" is produced differently depending on the reasoning practices of the parents, which ultimately can be traced back to their needs; having a child that performs well in school as opposed to one that conforms to the behavior of the parents when they were children.

Singh concludes "the substance of these moral concepts, and parents' moral resolutions to dosing dilemmas, are anything but predictable; rather they are inconsistent, contradictory, strategic and incomplete. Parents of ADHD children hold a definition of authenticity that shifts according to what they value in particular

contexts." Singh's use of the word 'strategic' here produces another point of connection to Lakoff's view of pharmaceutical reason, which he calls a "strategic logic." This phrase is helpful for describing the way in which the reality of ADHD is carried out through particular practices of reasoning. When we read Singh's work through Lakoff's, we can recognize that notions of the self and identity can take on radically different forms depending on what "strategic logics" are employed when producing that self. Of great importance to this issue is also the fact that the actual subject, the ADHD child, has not been interviewed in the context of this study into his or her authenticity. To what extent do these competing processes of reasoning produce the experience of ADHD for the patient?

Beyond the strategic logics that differentially produce the identity of ADHD patients, there is more to say about the extent to which the diagnostic category of ADHD itself emerges in relation to strategic logics. The way that strategic logics play out in the production of reality might be understood in response to the notion of the "ratiogenic" as developed by Val Plumwood. Plumwood explains that the word "ratiogenic" literally means "reason generated." In her book *Environmental Culture* Plumwood suggests that "Reason has been captured by power and made an instrument of oppression." (Plumwood, 2002) While I would eschew the notion that "reason" itself could be villainously kidnapped, I am sympathetic to the concern that some strategic logics can become overly dominant. Certain logics can be strategically and dominantly employed such that competing logics are made nonsensical or irrational.

Plumwood's concern in *Environmental Culture* is that a form of economic reasoning has come to determine human decisions about the environment, and that this dominant form of reasoning has now become so well entrenched as to serve only the needs of capital and not the needs of penguins, which are her critters of interest in this book. When we look at the struggle of scientific psychiatry to establish itself as the dominant logic of the reality of mental disorders, we can see that it has had less success than the economic logics that Plumwood believes dominate our environmental decision making practices. In my view, Plumwood's mistake is in thinking that "reason" can ever be only a single practice. While I agree that the enlightenment notion of "Reason" is perhaps the most successful and dominant logic ever practiced by humans, there are many logics that vie for self-expression in human realities.[24] We need not suggest that reasoning is held by one power at the expense of another. Rather, I would suggest that different strategic logics are instantiated by various groups of humans and non-humans in order to respond to needs.

What Plumwood expresses most usefully about the ratiogenic is that reasoning practices (i.e. strategic logics) are capable of taking part in the production of our reality. Plumwood says

> Ratiogenic patterns of thought and organisation – monological, rationalist, hyper-capitalist, colonising and centric – seem at first to be ghosts, shadowy, insubstantial figures, mere phantoms of the real world of political action. But as we scrutinise them more closely we can learn to recognise their very real and material traces intertwined in our lives (Plumwood 2002)

[24] I am also concerned that Plumwood seems to hold nature and reason in contrast. In my view nature and logics emerge from one another.

In *Environmental Culture* Plumwood demonstrates ways in which our practices of reasoning produce real outcomes. In exploring what Singh and Lakoff have said about how the reality of ADHD gets produced, we can see that strategic logics play an important role in the notions of identity and authenticity of subjects. The question that drives the final section of the chapter will be: how can we use the idea that strategic logics take part in reality production to make healthier realities possible?

The various strategic logics that are carried out with respect to the diagnostic category of ADHD have different outcomes for patients, clinicians and researchers. Psychiatric researchers might be said to instantiate the strategic logic of scientific psychiatry when carrying out the experiments that produce the catecholamine hypothesis. Appelbaum, a psychiatric researcher himself, describes the "deliberate process of self-redefinition" undertaken by psychiatry in the 20th century in this way. He recognizes that the rebirth of psychiatry is initiated "from a desire to have mental disorders and their treatments accepted as essentially equivalent to other medical disorders and their interventions." (Appelbaum, 2005)

Appelbaum goes on to foreground the ways that Singh's analysis shows that the incompleteness of the re-creation of scientific psychiatry. "For the scientific psychiatrist, it suggests a substantial failure to persuade the public to embrace a medical explanatory model for behavioral disorders." (Appelbaum 2005) This discord between individuals and groups that are concerned with ADHD provides an opening for reconfiguration of measurement practices. If we take the category of

ADHD to be a ratiogenic one, produced at the confluence of multiple strategic logics, then it devolves on those involved to examine their own engagement within that category. If the entanglements between the needs of various groups can be better understood, a potential for healthier futures emerges from the site of that understanding.

5.) The endogenous logic of ADHD and the design of our culture

One helpful entry point into the measurement of ADHD is a formation I refer to as the "endogenous conception of ADHD." The endogenous conception of ADHD is a complex set of measurement practices which serves the strategic logic of scientific psychiatry, but seems at odds with widespread public acceptance. By employing measurement techniques that focus on determinants of the disorder that are *within* children rather than being related to environmental factors, scientific psychiatry achieves a disorder that is purely neurobiological, and must be treated through medical means. While this functioned in accord with the desire for exculpation for the parents of ADHD children, this set of measurement practices no longer aligns with the goals of ADHD parents. Parents want to feel involved in their children's upbringing, and since the endogenous features of the disorder are capable of being manipulated most effectively by means of psychopharmacological intervention, the endogenous conception of the disorder no longer serves the strategic logics employed by parents of ADHD patients. The opening for the

possibility of other types of intervention works only when exogenous features of the disorder are properly studied. Data shows that scientists have systematically avoided the exogenous features of the disorder in their experiments.

The clearest demonstration of the claim that exogenous factors are being ignored comes from a meta-analysis document written by ADHD researcher Joel Nigg. Looking systematically through hundreds of ADHD research papers, Nigg demonstrates that ADHD researchers are more focused on the endogenous features of the disorder, i.e. those features that are internal to the bodies of ADHD patients; genetic, neurological, neurochemical etc. Exogenous features, such as psychosocial, environmental and other correlates of the disorder have been studied significantly less than the brains and genes of ADHD patients. (Nigg 2003) The strategic logic of scientific psychiatry, which depends upon the demonstration of a medical deficit for mental disorders, demands the endogenous framework of investigation. An apparatus of measurement, which responds to the needs of scientific psychiatry, has been constructed in order to produce the type of evidence that further bolsters its strategic logic. The exogenous features of the disorder have been excised from the apparatus of measurement so that the view of the disorder according to scientific psychiatry gains support.

Ilina Singh has demonstrated how the emergence of scientific psychiatry converged with issues of parenting in the middle of the century to cement the view of ADHD as a disorder of the brain to be treated pharmacologically. As the needs of patient communities and medical communities have aligned, the strategic logics

employed by these communities have supported the catecholamine hypothesis. However, while ADHD patients and parents of patients have demanded legitimacy for the disorder, other cultural currents are at flow through the phenomenon. Concerns over administering chemicals with FDA recognized side effects to children are abundant. The notion that ADHD is a disorder of the brain initially worked within the culture to absolve parents (especially mothers) of blame for childhood misbehavior. Singh says "An increased emphasis on the organic underpinnings of childhood behavior problems meant that mothers were more often assured that mental illness was not their fault." (Singh, 2002) However, Singh argues, this same move towards a neurological conception of the disorder has also divested parents of their ability resist medical intervention or to promote other types of intervention.

The goal of producing healthier children is the first priority of parents, medical practitioners and researchers, but how to achieve that goal is becoming an increasing source of contention. The difficulty here is to produce a position which accepts that while catecholamines are the main neurotransmitters involved in ADHD and that stimulants support this function, the volume of psychostimulant medication must to be reduced where possible. The continued contestation of ADHD's legitimacy as a diagnosis is, in Singh's view, an attempt to re-embody the disorder in a social context, rather than viewing it purely from the perspective of scientific psychiatry. Singh argues that

> Scientific understanding of ADHD in this decade has actively promoted a disembodied reality to ADHD through a brain-based discourse of neurotransmitters, receptor sites, and chemical processes. The brain, divorced from the body, is divested of time and history in which the body moves, and so ADHD, which resides in the brain, would also appear to have no history. (Singh, 2002)

The public has been unwilling to fully accept the view of the disorder furnished by scientific psychiatry. In light of this fact, new openings must be made in order to produce realities for the disorder that can be deemed healthy by both psychiatrists and patient groups. In order to see a way that the logics that produce the catecholamine hypothesis might be opened in order to produce new possibilities for treatment, these groups must look more carefully into the apparatuses of measurement that are employed to support the views of scientific psychiatry.

The endogenous conception of ADHD is not one that is intentionally carried out by some specific individual or group. Rather, apparatuses of measurement emerging from various strategic logics support the needs of those concerned with ADHD. Agents within these apparatuses preferentially cut exogenous features of the disorder out of the phenomenon of ADHD while continuously refining the understanding of endogenous factors. The problem here is that unless the exogenous features of the disorder are properly understood, the disorder will preferentially materialize in ways that fail to consider exogenous context as a relevant feature. For example, in experiments that compare the effectiveness of drug treatments to behavioral therapy, if the architectural and psychosocial contexts of the disorder are not properly theorized, the many of the beneficial effects of behavioral treatment are underrepresented in the data that is produced.

The exogenous features of the disorder I am most interested are not the kind of environmental factors that are usually invoked in public discussion. Newspaper articles frequently bring up the omnipresence of television, video games, and

increasingly "smart phones" as sources of continual distraction for children and teenagers. One *New York Times* writer suggests that our current technological world makes kids "wired for distraction." (Richtel, 2010) While some studies have shown that maternal habits such as alcohol and nicotine use during pregnancy increase the likelihood of ADHD in children, other environmental features such as television, video games and sugar intake have shown no correlation to ADHD symptoms.[25] (Linnet et al., 2003) (Nigg 2003) Studies such as these have been carried out partially in response to public beliefs about the causes of ADHD, and they demonstrate that electronic and digital technology is not to blame.

The exogenous features that are most relevant to ADHD are a far less visible form of "environmental factor" than electronic devices. The main site of the emergence of the phenomenon of ADHD is the elementary school classroom, and this environmental factor is systematically under-theorized. The architecture, temporality, social practices and expectations of elementary school classrooms are major features of the apparatus of measurement for ADHD, and scientists rarely discuss this feature in when they produce and present data. Looking carefully at apparatuses of measurement for ADHD can help us to see how strategic logics are co-constitutive of experimental configurations. The experimental configurations employed in ADHD science produce a type of data that is used to reinforce the logic of scientific psychiatry because these configurations emerge from that logic.

[25] It should be noted also that while maternal prenatal habits might be called "environmental factors" they are not really "exogenous" in the sense I am trying to express here. Maternal habits have the effect they do because they affect the material structure of the brain in permanent ways. What I am trying to describe with the term "exogenous" is the architectural, temporal and psychosocial contexts of the phenomenon of ADHD.

The definition of ADHD contained in the *Diagnostic and Statistical Manual of Mental Disorders* delineates symptoms of the disorder in a way that assumes a particular classroom configuration. Symptoms in the diagnosis of ADHD such as "Often fidgets with hands or feet or squirms in seat," or "Often gets up from seat when remaining in seat is expected," or "Often runs about or climbs when and where it is not appropriate" (APA, 2000) require a certain classroom configuration in order to be observed. The phenomenon of fidgety children emerges in part because of the way that classroom chairs and the expectations surrounding them are designed. In order for the DSM diagnostic symptoms to correlate meaningfully to teacher observations, chairs must be standard four-on-the-floor seats that children are expected to sit still in for hours at a time, and the space of the classroom must be divided into acceptable and unacceptable zones. In order to diagnose a student with the hyperactive features of ADHD, they must behave a certain way within this particular spatial configuration. While it is true that ADHD must be observed both in school and in another setting such as the home or in day care, the primary observational venue of the disorder is the classroom.

The phenomenon of ADHD emerges in certain contexts, and it is most visible in the classroom. The design of the classroom reflects certain values, and these values need to be examined in order to test their timeliness and usefulness. When we look at the architectural form of the elementary school classroom we can see how it has been designed to meet the needs of the society it was designed for. It has been noted frequently that these goals closely reflect the goals of pre-industrial and

industrial society. (See for instance Sir Ken Robinson's "Changing Education Paradigms") (Robinson, 2011) Because the United States is moving into an era that could be considered "post-industrial" in that it focuses more on the production of services rather than goods, our school system will need to be updated to reflect new needs, and these may not include the traditional classroom model as it has been currently imagined and created.

Our elementary education system was designed to reflect the economic and social ideals of the industrial revolution, and the classroom architecture in many ways is designed to prepare students for work in this kind of economy. Some area of personal space is granted to the student, usually taking the form of the desk and chair, which they are expected to keep tidy. Additionally, students are expected to complete work according to a particular temporal formation, and not to disturb the work of others around them.[26] Children are expected to work on a single task at a time. If collaboration with other students is required, this collaboration is to take place in a fashion still ordered by the standard desk-and-chair model. The design of the traditional classroom takes place on both material and social levels, as the behavior of students is ordered around the material configuration. The phenomenon of ADHD is measured within this context, and yet this context is never adequately theorized within the course of experimental design. In ADHD experiments, a boundary has been enacted that removes the classroom architecture from theoretical consideration, and thus the meaning of data does not reflect the way that this

[26] Some writers have noted that the temporal structure of schools associated with the ringing of bells reflects the temporalization of the factory and the steam whistle.

architecture takes part in the production of data. Data produced in such a configuration is more easily interpreted as supporting the conclusion that psychostimulant medication is the only necessary treatment for ADHD.

The endogenous conception of ADHD emerges partially from the fact that classroom architecture has not been well theorized within ADHD science. Evidence has shown, for instance, that substituting inflatable exercise balls (a.k.a. therapy balls or yoga balls) for traditional four-footed chairs has helped reduce problematic behaviors associated with ADHD. One study concludes that "use of therapy balls (instead of chairs) for students with ADHD may facilitate in-seat behavior and increase legible word activity." (Schilling, Washington, Billingsley, & Deitz, 2003) When behavioral treatments are compared with psychopharmacological treatments in large comparative studies, the classroom configuration is rarely discussed.

Many rating scales for the observation of ADHD contain similar or identical criteria to the DSM, which means that any study that employs these rating scales to test for the effectiveness of psychostimulant treatments is unlikely to fully account for the role of classroom architecture in the phenomenon. These ratings scales account for a major feature of the stability of the phenomenon, and this stability is in turn based on the fixedness of classroom architecture. Meta-analysis of pharmacological intervention studies has shown that "important demographic and descriptive information, such as…classroom settings, were often poorly defined" in ADHD experiments. (Trout, Lienemann, Reid, & Epstein, 2007) This way of measuring ADHD encourages the endogenous description of the disorder in at least

two ways. First, it fails to consider the possibility that alterations of classroom space have measurable effects on ADHD behavior. Second, it creates a situation where psychostimulant medication appears to be more favorable than other kinds of intervention for the disorder.

Many evidence-based psychosocial interventions for ADHD exist, such as behavioral parent training and behavioral classroom management, and yet psychostimulant medication alone is by far the more popular option. The largest ADHD crossover treatment study to date, the multimodal treatment study (MTA), has been used frequently to tout the superiority of psychostimulant medication. (Jensen et al., 1999) Theorizing the design of clinical trials such as the MTA has not been carried out systematically. Researchers Pelham and Fabiano worry that the cross over studies and meta-analysis documents supporting the effectiveness of psychostimulant medication have focused too much on large, short term cross-over studies that do not adequately reflect the benefits of behavioral interventions. (Pelham & Fabiano, 2008) This type of study design reflects a set of epistemological practices that focuses the on measurement of ADHD through endogenous determinants. Pelham and Fabiano suggest that the quantitative methods of large scale studies do not adequately consider the environment of ADHD patients. Single patient treatment studies, which more frequently explore the possibilities for environmental remediation, tend to be excised from meta-analysis documents.

Derek Bolton has argued randomized, controlled trials present special epistemological problems for psychiatric research. (Bolton, 2008) In the article

Bolton suggests that this type of trial is a.) more effective for the measurement of psychopharmacological efficacy than for other types of treatment and b.) issues from the movement of psychiatric measurement towards practices of natural science. Again we can see how the logic of scientific psychiatry produces an apparatus of measurement that favors an endogenous conception of ADHD and its treatment by psychostimulant medication. Scientists need better theoretical and critical tools to understand the meaning of their data in relation to experimental design. Carefully considering the way that strategic logics are produced in response to needs, and how these logics relate to apparatuses of measurement might provide a useful tool in this arena. Additional examples of how this view of experimentation works are produced below.

Another feature of the classroom that is often not well theorized in ADHD experiments is the role of the elementary school teacher in observation of the phenomenon. Because a significant portion of ADHD cases are diagnosed at the recommendation of elementary school teachers, observations of classroom behavior are a vital part of the manifestation and measurement of the disorder. (V. E. Snider, Frankenberger, W., & Aspenson, M., 2000) The temporality of ADHD in the classroom is also heavily dependent on the behavior of teachers and the expectations involved with teaching an elementary school class.

In the course of most comparative drug studies teachers and parents are asked to rate their students' behavior in order to compare the effectiveness of pharmacological and behavioral therapies. Problematically, these same teachers and

parents are also expected to *administer* behavioral therapy during the course of the experiment. The teacher's role as a feature of the apparatus of measurement and part of the phenomenon under investigation has not been well theorized. In the following example, one study that concluded that stimulants were more effective than behavioral therapy for treating ADHD was altered in significant ways in order to deal with the issue of teacher roles. The publication of this study contains the conclusion that "The fact that parents, teachers, and psychiatrists were not blind to treatment and that parents were involved in treatment delivery *should have biased the results* in favor of psychosocial treatment" (Abikoff, Hechtman et. al. 2004, emphasis added). Here the experimental designers assume uncritically that if a parent or teacher is involved in treatment they must preferentially rate the positive effect of their own intervention on the behavior of the child. They give no evidence or argument for this view. In order to control for the effects of this type of bias, which they suggest occurs in other studies, these researchers tell the reader that "we did not implement active intervention in the classroom" (Abikoff et al., 2004) during the course of the study.

Pelham and Fabiano, who vigorously contest the outcome of this study, point out that by having teachers "not actively intervene" with ADHD behaviors, the experiment measured a situation in which teachers did not engage in the normal interventions that are needed to control behavior in an elementary school classroom. They suggest that by altering standard processes that occur within the classroom, this experiment produced results that were biased in favor of pharmacological

intervention. (Pelham and Fabiano 2008) The accusations of bias have not helped the disputants in this debate to make headway. Instead, if these groups of researchers would collaboratively and critically consider the way that previous experiments have been designed to produce certain types of data, new and more effective experiments could be produced. These collaborations could take place effectively within spaces like the one created during meetings of the Science and Justice Working Group. In these meetings, participants are encouraged to think with each other about how new configurations might be produced that move beyond the problems facing older experiments.

The features of the classroom environment are part of the phenomenon and measurement apparatus of ADHD. If parents of ADHD patients wish to take a more active role in their children's treatment, it is the exogenous features that provide the greatest promise for this possibility. Just as scientists have been willing to study the effects of videogames on ADHD behavior, studies that focus on the spatial and temporal contexts of ADHD will emerge if these concerns are well voiced. This will open the way for a new hybrid logic of ADHD, one that accepts that genetic and neurological factors can be altered by means other than psychostimulant intervention, thus responding to the needs of psychiatric researchers and parents of ADHD patients.

If new possibilities for the spatial and temporal features of classroom design can be explored, new forms of measurement might provide data on non-

pharmacological interventions for ADHD. The most pressing question here is how to go about considering the possibilities for new designs.

How to redesign the classroom so that the apparatus of measurement can reflect new possibilities for ADHD treatment will be a matter of societal collaboration. Leigh Star's work into the remediation of infrastructure can help us to begin considering how this might be carried out. Although this iteration of Star's diagram (Fig 2) is focused on the design of cyber infrastructure, it can be effectively abstracted to discuss the infrastructure of classrooms as well.

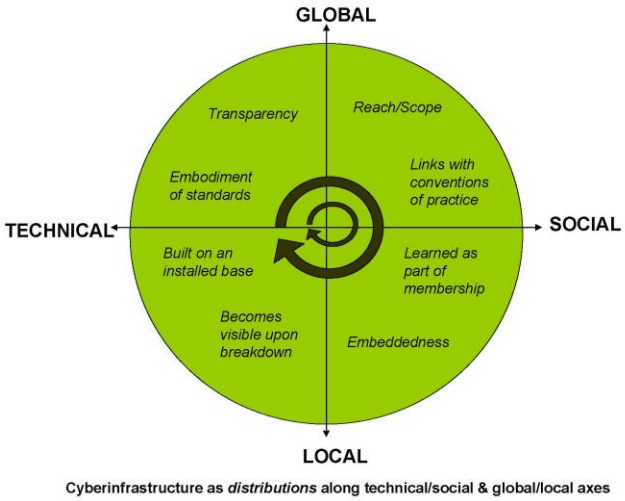

Fig 2.

I have already suggested one way in which the spatiality of elementary school classrooms might be altered to include inflatable balls in addition to four on the floor chairs. It is at first difficult to accept the idea of giving a hyperactive child a bouncy ball inside the classroom, and this reflects a technical expression of global practices. The western style of education has been successful in helping many countries to achieve massive industrial development, and is thus extremely well

entrenched. However, if social practices can be changed at the local level and their efficacy demonstrated, then technical fixes adopted by local communities successfully can become part of global practice. Anecdotal evidence has suggested that allowing children to occupy spaces that are not dominated by traditional writing desks also helps ADHD children to sit more comfortably. Elementary school teachers have gone as far as to remove the legs from the desks in their classroom, allowing the children to sit on the floor in circular spaces marked off by tape. These teachers have observed decreases in the frequency and severity of symptoms their ADHD students express. The embodied aspects of the disorder are somatic in a way that cannot be captured merely by gesturing towards the brain, and altered spatial practices reflect this.

When we look into the history of education comparing the teaching styles of Plato and Aristotle, we can see that the architectural formation of the modern school system mirrors Plato's Academy, a stand of trees later enclose by a wall where students and teachers engaged in lecture and dialogue. Aristotle taught in the peripatetic style at the Lyceum, and it is this active style that may be a more effective learning formation for some students. Nietzsche concluded in *Twilight of the Idols* that "only thoughts reached by walking have value," (Nietzsche & Large, 1998) and this thought ought to be remembered when we consider the architectural formation of the classroom.

Additionally, rethinking the temporality of the classroom might help to remediate teaching practices in ways that are beneficial to ADHD students. One

elementary school teacher who teaches at a Waldorf school explained to me that they do not use bells to break up the school day. Instead, this school employs a temporal design they call "breathing in and breathing out." Instead of dividing the school day into distinct subject periods, this new temporal design focuses on doing individual activities for shorter periods of time, and balancing kinetic with auditory and visual learning. Usually, the "breathing in" phase will focus on some sort of written lesson, while the "breathing out" phase allows the children to be more interactive, and to learn through tactile and kinesthetic means. Because ADHD patients are allowed to use their bodies more energetically, many of their hyperactive systems are abated in this context.

While some children might have ADHD so severe they need to be repeatedly dosed in the day, a more careful examination of what points in the day ADHD symptoms are most visible might help to reduce the volume of psychostimulants currently deemed necessary.

The possibility of exploring different styles of intelligence through varied learning techniques has also been understudied in relation to ADHD behaviors. Howard Gardner's theory of multiple intelligences might allow different standards and techniques to be implemented for different kinds of students, thus producing new norms and standards. These new norms might well reduce the extent to which certain children need to be medicated, because they will allow children of one type of intelligence more leeway in areas where their intelligence is not the strongest. I am not suggesting that educational standards should be in any way lowered.

Rather, if children's success was measured in areas that they felt interested in and comfortable with, their willingness to explore other forms of learning might be encouraged in ways that have not been discovered yet, leading to new types of academic success. This new kind of measurement practice might help to assuage some of the symptoms associated with "inattention."

Symptoms such as "often does not give close attention to details or makes careless mistakes in schoolwork, work, or other activities," "often has trouble keeping attention on tasks," "often does not follow instructions and fails to finish schoolwork, chores, or duties in the workplace," and "often avoids, dislikes, or doesn't want to do things that take a lot of mental effort for a long period of time (such as schoolwork or homework)" have not been systematically examined to determine whether they manifest more prominently during areas of study that are not congruent to the patient's brand of intelligence. If experiments were designed with this new form of classification in mind, the data regarding various forms of treatment might be significantly different from what currently exists.

6.) Conclusion – The Future of the Catecholamine Hypothesis

The catecholamine hypothesis is not a static view of ADHD but a continuously evolving narrative through which the etiological validity of the disorder is established. Earlier descriptions of the disorder that express ADHD as a simple deficiency of a single neurotransmitter have been abandoned. (Pliszka, McCracken, & Maas, 1996) A rore recent picture of the ADHD brain as failing to

properly regulate catecholamine function in the PFC has become commonplace. (Arnsten, 2011) (Arnsten & Pliszka, 2011)

One very notable feature of recent publications discussing the role of psychostimulants in the treatment of ADHD has been the acceptance of methylphenidate and amphetamine as cognitive enhancers. (Berridge & Devilbiss, 2011) The recognition that low doses psychostimulants help to regulate the PFC functions of both ADHD and non-ADHD individuals has been adopted into the narrative of the catecholamine hypothesis. New studies that feature descriptions of the disorder as a failure of catecholamine regulation now invoke the need for psychostimulants as a way to regulate a system that otherwise has difficulty regulating itself. Insofar as this is the case, we are entering a new phase of the diagnostic category that accepts it as a weakness in cognitive ability. The difficulties for patients caused by the disorder are those associated with work stress, etc. rather than a distress caused directly by the neurochemical state itself.

Proposed future research programs for psychostimulant treatment of the disorder tend to focus on the detailing the precise process by which well-regulated catecholamine levels in the PFC allow for optimal functioning. As one paper expresses it "Although the biochemical mechanisms responsible for this effect are well known, the conceptual framework for understanding the physiological basis of MPH efficacy in ADHD treatment or its ability to improve cognitive function in normal individuals is lacking." (Agster et al., 2011)

The important consequence of more recent scholarship on catecholamine function is to recognize how the etiological and clinical validity of the disorder have re-synced in order to provide a cohesive narrative, despite the fact that this new story alters our broader definition for mental disorders. The notion that a mere cognitive deficit can provide the basis for a full-fledged mental disorder is novel, and yet this new state of affairs has not been fully addressed. Where does the line for a cognitive deficit get drawn within our culture if the notions of suffering are change with the requirements for performance? The metaphor from cholesterol and cholestatin administration might once again be relevant here. The cut-off for high cholesterol has lowered significantly with the increased availability of cholestatin drugs. (Greene, 2007) How will our culture's notion of cognitive normalcy change now that a cognitive deficit can be described as a psychiatric disorder?

Second Case Study: The Stabilization of Depersonalization Disorder

1.) Introduction to the phenomenon of Depersonalization Disorder

Depersonalization Disorder (DPD) is a member of the class "dissociative disorders" as delineated in the *Diagnostic and Statistical Manual of Mental Disorders*. (APA, 2000) Dissociative disorders are those characterized by a tendency of patients to break from normal integrated conscious functioning. Although the phenomenon manifests in different ways, the most common expressions of *the experience of* depersonalization (as opposed to the disorder of the same name) describe it as the feeling that one is not in control of, or connected to, one's body. More detailed descriptions of the experience include watching oneself from a distance, "acting one's part" in an interpersonal situation, feeling detached from one's image in the mirror, not feeling in control of speech or bodily movements, and feeling detached from thoughts and emotions.[27] These experiences are often accompanied by or expressed as the feeling that one's surroundings are *unreal*. The experience of unreality is sometimes referred to as "derealization" and although this symptom is included as a separate category in the DSM – IV (TR), it has been recommended for inclusion under the Depersonalization Disorder diagnostic heading by the DSM – 5 dissociative disorders subcommittee.[28] (Spiegel et al., 2011)

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[27] This final symptom description includes symptoms of emotional blunting and physical numbing.

[28] The diagnostic manual used by European Psychiatrists, the International Classification of Diseases (ICD-10) uses the term Depersonalization-Derealization Syndrome to delineate the category, whereas the DSM takes derealization as a symptom of Depersonalization Disorder.

The experience of depersonalization is thought to be the third most common psychiatric symptom after depression and anxiety. (Sierra, 2009) Depersonalization researchers suggest that the experience of depersonalization occurs most commonly in humans who are placed in situations of high anxiety or have experienced some type of emotional trauma. (Dell & O'Neil, 2009) When the experience of depersonalization is chronic in nature and is not better explained by the presence of another disorder it becomes classified as a separate diagnostic category rather than a symptom. This category is referred to as Depersonalization Disorder (DPD).

In this case study of the science of Depersonalization Disorder I will consider the ways that knowledge making practices have produced the reality of the diagnostic category. The core focus of this case study will be the neurobiological and psychological approaches to the measurement and treatment of the disorder. I will ask the reader to consider the possibility that these two approaches are producing different material-discursive realities for the disorder, and that understanding these different realities may help to create an effective treatment program for the disorder where none currently exists.

2.) **The Status of DPD in Relation to Differing Research Approaches**

In previous eras the status of Depersonalization Disorder (DPD) as a psychiatric diagnostic category has been contested. As recently as 2003, psychiatric researchers have gestured toward the disorder as lacking "convincing empirical justification" for separation from other, more mainstream diagnostic categories, such

as anxiety and depression. (Parnas & Handest, 2003) When I mentioned that I was interested in the phenomenon of Depersonalization Disorder to a recently trained psychiatrist in 2009, his first question for me regarding the disorder was: "Do you think it's real?" In the past, concerns about the status of dissociative disorders have arisen, particularly around Dissociative Identity Disorder, and this may have caused increased skepticism regarding DPD when the body of recent medical evidence for the disorder was smaller. Since 2003 increasing layers of empirical evidence for the disorder have helped to assuage this concern amongst psychiatrists. The diagnostic category will appear in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* edition five. (DSM – 5)

For the researchers who have devoted careers to the study of the disorder, the most vexing issue is the dearth of effective treatments for the disorder. One recent comprehensive book written about the disorder laments that "unfortunately, the condition has been shown refractory to most conventional medications used in psychiatry, and at the time of writing, there is no officially recognized treatment." (Sierra, 2009) This fact is recognized by other prominent DPD researchers who note that there is a "failure of chronic depersonalization to markedly respond to any of our standard pharmacological interventions." (Dell & O'Neil, 2009) The lack of an effective treatment remains the most powerful obstacle for the widespread acceptance and diagnosis of the disorder. As one psychiatrist has put it "the diagnosis does not seem to be particularly useful for patients, as depersonalization

symptoms are extremely resistant to both medications and psychotherapy." (Reeves, 2010)

The process by which DPD has been stabilized as a diagnostic category has gone through many phases. The stability of a psychiatric diagnosis changes within the larger context of psychiatric research and treatment. Disorders have emerged and disappeared at various moments in history depending on various factors.

The current standards of evidence within psychiatry are best described as a propensity towards "scientific psychiatry," in which mental disorders must be described in terms of a neurobiological situation. The movement towards scientific psychiatry and neurobiological description has had many effects including the de-stigmatization of mental illness, as well as the replacement of psychotherapy by psychopharmacology as the primary instrument of treatment. (Appelbaum, 2005)

The formation of scientific psychiatry has encouraged the development of a neurobiological description for Depersonalization Disorder. Psychiatric researchers have worked to understand the disorder primarily through a description of biological and/or neurochemical processes in the brain. The current best-confirmed theory of the disorder: "the cortico-limbic disconnect hypothesis," holds that DPD involves a disruption of the relationship between sensory stimuli and psychological processing of emotional content. Speaking in neurological terms, these processes occur in and between "cortical areas involved in integration of sensory and somatic processing" and the limbic system, which is implicated in the processing of emotional data. (Guralnik, Giesbrecht, Knutelska, Sirroff, & Simeon, 2007) (D. J. Stein

& Simeon, 2009) The disconnection between cortical areas and the limbic system fits the phenomenological descriptions that have emerged for DPD. Depersonalization experiences occur normally in humans when situations of high anxiety occur, allowing individuals to continue to act despite tremendous emotional stress. The function is made possible by the dissociation of emotional experiences from their other cognitive processes. As one research team has described it: "an evolutionary perspective suggests that attenuation of emotional responses, mediated by deactivation of limbic structures, may sometimes be advantageous in response to inescapable stress." (D. J. Stein & Simeon, 2009) It is this description of depersonalization experiences which underscores the current neurobiological picture of the disorder. The pathological experience of depersonalization occurs when this "attenuation of emotional responses" persists outside the expected context.

The recent history of research focusing on the disorder is marked by the development of a new measurement scale for the disorder: the Cambridge Depersonalization Scale (CDS). (Sierra & Berrios, 2000) Following the development of the CDS, the phenomenological stability of the disorder was tracked by comparing depersonalization cases dating from before 1946 to modern diagnoses. This research showed that clinicians' observations and patient reports of the phenomenology of the disorder have remained remarkably consistent throughout the 20th century. (Sierra & Berrios, 2001) Next, separate research groups at centers in the U.S. and U.K. used an array of measurement techniques to differentiate the

symptom clusters of the disorder. The agreement between these various studies was strong, thus providing greater support for the stability of the diagnosis and demonstrating the specificity of symptoms across different experimental groups. (Sierra et al., 2005) (Simeon et al., 2008) Using evidence of symptom clusters and clinical observations, researchers have been able to produce an increasingly convincing neurobiological picture of the disorder. (Phillips et al., 2000) (Phillips & Sierra, 2003) (Medford, Brierley, Brammer, David, & Phillips, 2003) (Ketay, Hamilton, & Simeon, 2010)

Although the previous paragraphs present scientific findings according to the date at which they were published, these forms of research (the exploration of phenomenological stability, the creation of a rating scale, and the division of the disorder into symptom clusters) are all inter-latticed and implicated in one another. Despite the fact that the publications appear in successive years the different forms of research take place concomitantly. Each form of research creates and adds to a description of the disorder which forms part of the neurobiological picture.

When discussing the stability of the diagnosis it will be necessary to better understand how the relationship of materiality and discursivity functions within psychiatric research. In order for the disorder to *materialize* in the way the cortico-limbic hypothesis suggests the discursive practices (e.g. what descriptions of the phenomenon come to be deemed significant for the purposes of diagnosis) by which this materialization takes place must be stabilized. While scientific psychiatry requires that mental disorders be understood in material terms, it must be noted that

mental processes are always implicated in a discursive formation in order to be given meaning and made intelligible.[29] By the phrase "discursive formation" I mean a set of practices by which experimental configurations and logical processes are carried out. For example, if I were to approach the phenomenon of schizophrenia as psychiatrist in the 1950s using behaviorist techniques as opposed to cognitivist ones, I would come out with different conclusions about the patient and the nature of the disorder.

The cognitivist and behaviorist approaches to psychiatric phenomena constitute disparate discursive formations: they employ different experimental configurations and logical processes from one another, and they reach different conclusions about the nature and proper treatment of mental disorders. For instance, where attempts to treat schizophrenia from the behaviorist approach focused on token economy practices (Wong, 2006) cognitivist approaches focus on the alteration of particular thought processes associated with the disorder. (Grant, Huh, Perivoliotis, Stolar, & Beck, 2011) Each approach has different triumphs and failures with respect to treatment, which is one reason for the merging of cognitive and behavioral techniques into a single form of therapy (cognitive-behavioral therapy) in the 1980s. Additionally, these approaches also create different material processes associated with mental disorders. While behaviorism traditionally eschews talk of mental states in favor of brain states, cognitivist models tend towards a reduction of mental states to representations or symbols. These

[29] I will explore the meaning of the terms "material" and "discursive" (and their adjectival and adverbial forms) throughout this chapter to draw out the ways in which scientific practices produce the reality of the diagnostic category of Depersonalization Disorder.

approaches mean different treatment practices, which in turn alter the state of affairs within individual patients undergoing a particular form of treatment. This material context unfolds both within and outside the body of the patient.

The differences between the discursive formations that are being used to produce knowledge about DPD are less significant than the differences between cognitive and behaviorist models of psychiatry, yet they do produce different conclusions about the manifestation and proper treatment of the disorder. I will endeavor to show that there are at least two discursive materializations for the phenomenon of DPD that are drawn together as a single category in the *Diagnostic and Statistical Manual of Mental Disorders* through the work of distinct epistemic practices.[30] In the case of DPD the two discursive formations I will focus on include a neurobiological approach and a psychological approach.

Depersonalization Disorder researchers have used a broad array of evidentiary techniques in order to differentiate Depersonalization Disorder from Axis I mood and anxiety disorders and to stabilize it as a separate diagnostic category. These evidentiary techniques include neurobiological, psychopharmacological, cognitive-behavioral, cognitive-affective and phenomenological techniques. (Sierra & Berrios, 1998) (Simeon, Knutelska, Nelson, & Guralnik, 2003) (Hunter, Phillips, Chalder, Sierra, & David, 2003) (Guralnik et al., 2007) (Sierra, 2008) (D. J. Stein & Simeon, 2009) While the separation of the

[30] Discursive materializations can be shown to be distinct in the context of psychiatry when the research practices and treatment recommendations associated with each are not employed in conjunction with one another. There is no need for distinct discursive materializations to be incompatible in principle. Their distinctness is merely a function of the reality they produce in relation to the phenomenon in question.

diagnostic category from other disorders has been well established, the disorder continues to be underdiagnosed. (Sierra, 2009) The infrequent diagnosis of the disorder has been attributed to "limited familiarity" with the disorder on the part of many clinicians, reluctance on the part of patients to describe or seek help for their symptoms, inability on the part of patients to describe their symptoms linguistically, and misdiagnosis by clinicians as a variant of depression or anxiety. (Dell & O'Neil, 2009)

Another reason for the under-diagnosis of the disorder is that is has proven extremely difficult to treat effectively. Certain psychopharmacological interventions have shown increasing promise, but none have generated the kind of response that warrants a large scale study to prove the effectiveness of any single compound. Fluoxetine (Prozac) has been found largely ineffective (Simeon et al., 2004). Another SSRI, clomipramine, may be effective, but only one study has demonstrated this and its sample sizes are too small to draw dependable conclusions from. (Simeon et al., 1998) Lamotrigine is considered effective as an add-on treatment to SSRIs by some researchers (Sierra et al., 2006) but lacks sufficient evidence to be employed in a widespread manner. (Sierra, 2008) One recent study of lamotrigine-only treatment has shown promise, but the findings contradict previous studies and need to be replicated. (Aliyev & Aliyev, 2011)

In order for the diagnosis to become one that all psychiatrists are confident in conferring it must be not only stable but treatable. While the stability of the disorder has been sufficiently established, the search for an effective treatment continues. To

this end many different non-pharmacological techniques have been explored including Transcranial Magnetic Stimulation, (Mantovani et al., 2011) and various forms of psychotherapy including cognitive-behavioral therapy and psychoanalysis. (Hunter et al., 2003) (Hunter, Baker, Phillips, Sierra, & David, 2005) (Guralnik & Simeon, 2010)

Cognitive-behavioral treatments offered some promising possibilities for the treatment of DPD, but have been understudied in the past five years. (Sierra, 2009) While psychoanalytic approaches to treatment have been disfavored in "scientific psychiatry"[31] because of their resistance to empirical proof or disproof, (Appelbaum, 2005) forms of psychotherapy developed by practitioners of psychoanalysis may provide promising treatment possibilities for DPD patients. (Guralnik & Simeon, 2010) Because psychoanalytic treatments have not traditionally provided the same standards of evidence required by scientific psychiatry, they may not align properly with the research that has been done to stabilize the disorder in mainstream psychiatry.[32] While cognitive-behavioral approaches fit better with the medical model of psychiatry, publications discussing new developments in cognitive-

[31] I take the phrase "scientific psychiatry" from the referenced article by Appelbaum, who tracks the movement of psychiatry in the 20th century towards a medical analogue, in which mental disorders are seen as medical ailments and should therefore be described and treated in biological terms.

[32] While efforts have been made by psychoanalytic researchers to provide scientific psychiatric evidence for the effectiveness of psychoanalytic treatments, none of this type of research has been carried out with respect to proposed psychoanalytic treatments for Depersonalization Disorder.

behavioral therapy have been limited as compared with psychopharmacological research into the disorder in the last five years.[33]

3.) Different Approaches to the Study of DPD

Research into Depersonalization Disorder has increased heavily in the past fifteen years. This increase can be attributed largely to the creation of two independent Depersonalization Disorder Research Centers. One, located in New York, NY is led by Daphne Simeon. The other, located first in Cambridge, England and now in London, England, is led by Mauricio Sierra. Looking deeper into the Depersonalization Disorder research that has been carried out over this period, we can see how psychiatric research into DPD has been shaped by trends in neurobiological and psychological research.

In the past two years Daphne Simeon, Director of the Depersonalization & Dissociation Program at Beth Israel Medical Center, has participated in the publication of two articles regarding Depersonalization Disorder that embrace what might be considered divergent research programs. Employing the most up-to-date methods for creating neurobiological evidence of the disorder has been the goal of DPD researchers for the past decade, and much of Dr. Simeon's work has followed this line. Using psychoanalysis to treat the disorder moves in a direction that might be said to threaten the work that has been done to convince mainstream psychiatry of the stability of the diagnosis. Specifically, if the disorder cannot be fully

[33] This conclusion can been drawn by looking at the measurement technique and treatment discussions employed in Depersonalization Disorder publications contained in the "Web of Science" database maintained by Thompson Reuters.

understood and treated in neurobiological terms, this could damage its acceptance amongst those who adhere only to the program of scientific psychiatry.

The first article I will discuss here is entitled "Skin conductance and memory fragmentation after exposure to an emotional film clip in depersonalization disorder." (Giesbrecht et al., 2010) This publication reports data from a complicated experiment intended to demonstrate the material and "objective" aspects of the phenomenon of depersonalization. During the experiment two groups (the first consisting of 14 individuals diagnosed with Depersonalization Disorder and the other consisting of 14 healthy controls) are connected to a psychophysiological apparatus. This apparatus consists of a Biopac skin conductance measurement device and a computer running software which calibrates and records the measurements of the device.[34] The test subjects are then shown a 12.5 minute clip from the film *Silence of the Lambs*. (Demme et al., 1991) The clip is meant to induce strong emotions in the viewer. The data from the skin conductance measurement equipment is taken continuously throughout the duration of the clip and for five minutes after the clip has ended.

The skin conductance data provided by averaging and comparing the measurements of the DPD group and the healthy control (HC) group is meant to demonstrate the autonomic response of the subjects during various intervals of the film clip. This assumption is not foregrounded within the publication, probably because the direct correlation between high galvanic skin response levels and human

[34] The device measures the galvanic skin response between the ring finger and middle finger of the non-dominant hand of the subject in microsiemens.

anxiety levels has been well documented for decades. (Lader, 1967) (Epstein & Clarke, 1970) While the DPD group exhibited a shorter time to peak skin conductance levels, the intensity of the peak was similar to the levels exhibited by the HC group. This data is meant to create a psychophysiological expression of the differences between anxiety levels of DPD patients and healthy controls. It also accords with hypotheses drawn from previous studies about the neurobiological functioning of DPD patients. As one research group has expressed the function of DPD brains: "The pattern of neural response to aversive stimuli in depersonalized patients suggests that whilst appraisal of the emotional significance of emotive stimuli is intact, an affective state is not induced in response to this material in these patients."(Phillips & Sierra, 2003) In other words, normal depersonalization occurs in humans when they need to disconnect their emotional processing from extremely stressful stimuli in order to take action rather being petrified by fear. The notion that this is an evolutionarily selected trait is expressed by Stein in the following way: "An evolutionary perspective suggests that attenuation of emotional responses, mediated by deactivation of limbic structures, may sometimes be advantageous in response to severe stressors."(D. J. Stein & Simeon, 2009) People who experience chronic depersonalization exhibit this tendency with abnormal or continuous frequency, and their skin conductance levels provide psychophysiological evidence of the fact that their emotional response to aversive stimuli exhibits an abnormal pattern.

The skin conductance charts from this experiment produce data that is in line with the neurobiological picture of the disorder. DPD patients have a higher resting

skin conductance level (i.e. higher resting anxiety levels), but when they are shown an aversive stimulus such as the *Silence of the Lambs* clip, they do not respond with greater anxiety as the HC group does. Instead, DPD patients undergo a neurological response that inhibits the increase of anxiety levels by detaching their sensory experience from the emotional response experience they are undergoing, thus producing the feeling of unreality. That DPD patients break from normal integrated conscious functioning as a response to emotionally negative or aversive stimuli is well established in other studies in which DPD patients and normal controls are shown aversive photographs and their skin conductance and other physiological responses are measured. (Medford, Stringaris, Sierra, David, & Phillips, 2004)

The phenomenon of depersonalization might be described as a type of situational emotional blunting. When the anxiety level of the DPD patient increases past a certain point, their emotional response attenuates; the patient comes to experience the world as *unreal* and thus anxiety levels cease to increase. This data fits the cortico-limbic disconnect hypothesis of the disorder which holds that DPD symptoms can be described by

> "a pattern of reduced activity in regions important in the response to emotional stimuli, i.e. subcortical regions including amygdala and striatum, and increased activity in regions potentially involved in the regulation of emotional responses, i.e. predominantly prefrontal cortical regions" (Phillips & Sierra, 2003)

This data is exactly the kind of psychophysiological evidence that is valuable for stabilizing the disorder amongst adherents of scientific psychiatry. It fits a type of material evidence into a neurobiological theory of the disorder, thus giving psychiatrists a measure of the disorder that can be counted as objective in the sense

required by scientific psychiatry. The device used to measure the disorder gives data in microsiemens, a numerical unit of electrical charge. This type of measurement does not rely on self-rating questionnaires, which might be seen as unreliable because they describe the subjective experience of the respondent.

In addition to the data provided by the skin conductance equipment, the subjects also provide responses to ten different questionnaires as part of the experiment. Of the ten questionnaires, five focus on the history of the subject's mental states, two on the subject's current mental states and three on the subject's memory of the content of the film clip. The last three questionnaires are designed to measure the accuracy of the subject's memories regarding the clip.

Two of the last three questionnaires are unlike typical psychiatric self-rating scales because they do not ask the subject to report on their emotional state. Instead, they require the subject to chronologically sequence various moments in the film clip. Healthy controls demonstrated a higher degree of accuracy in correctly identifying the order of events in the clip than the DPD group. This data is meant as objective evidence for memory fragmentation experienced by DPD patients.[35] One of the symptoms of DPD has been labeled "temporal disintegration," (Simeon et al., 2008) and the researchers conducting this study want to present evidence of this feature of the phenomenon. The evidence that the DPD group performed noticeably less well in temporally sequencing moments of the clip from *Silence of the Lambs* is

[35] The experimental designers refer to one of the sequencing tasks as the "objective memory fragmentation task."

meant to provide evidence for the view that the temporal experience of the DPD patient is distinctly different from HCs.

These two measures, the skin conductance data and the temporal sequence task demonstrate crucial ways in which the experience of DPD patients can be expressed in both psychophysiological and cognitive terms. The psychophysiological measures are important to the study of DPD because the disorder has been notoriously difficult to measure in this way. The cognitive differences between DPD subjects and healthy controls also provide an "objective" distinction between these two groups. Because the stability of a diagnostic category in scientific psychiatry depends on these types of measurability, psychiatric researchers have gone to great lengths to find psychophysiological apparatuses that make the experience of depersonalization materialize as a neurobiological phenomenon. As DPD researcher Mauricio Sierra has put it

> "Unlike the case with most psychopathological phenomena where subjective reports are usually associated with objective behavioral manifestations, the distressing complaints of patients with depersonalization do not seem accompanied by observable changes in behavior. In spite of this, there is now a growing body of evidence revealing a number of neurobiological abnormalities in patients with depersonalization disorder."(Sierra, 2009)

The ways in which the disorder is brought to the material expression within this experiment requires close inspection. One way to understand the development of the material expression of the disorder is to consider it through the concept of discursivity. The concept of "discursivity" has been theorized differently by various philosophers including Michel Foucault, Ian Hacking, and Karen Barad. While the notion of the discursive has typically focused on issues pertaining to discourse, there is an increasingly strong recognition among philosophers that the notion of the

discursive should not be restricted merely to what is said, or what is capable of being said, but instead should contain all the objects, concepts and strategies that are employed in the creation of a set of scientific practices. Going a step further than this, Barad has said that "discursive practices are not human-based activities but specific material (re)configurings of the world through which boundaries, properties and meanings are differentially enacted." An important feature of this view is that "discursive practices and material phenomena do not stand in a relationship of externality to each other." (Barad, 2007) To take materiality and discursivity as external to one another in this context would be to treat the materialization of DPD as a process occurring separately from the discursive practices used to produce data about the disorder. This separation might lead one to believe that DPD has a material process that occurs in a single way regardless of the interventions we use to measure the phenomenon. Reading the data provided by this experiment through various notions of discursivity gives us a different understanding of the meaning of the data.

The experimental configuration contains many different features including movies, skin-conductance measurement equipment, several forms of questionnaires, as well as the diagnostic equipment used to differentiate DPD subjects from Healthy Controls, etc. Each of these elements is part of the materialization of the disorder in this context, and each one affects the meaning of the data that is produced by the experiment. Understanding the phenomenon of depersonalization as materializing neurobiologically according to the discursive formations of scientific psychiatry

requires recognizing the way material-discursive practices produce the reality of the disorder.

The discursive materialization of this experiment has outcomes for the conception and treatment of the disorder. The treatment program for Depersonalization Disorder is in a nascent stage, and the options for the development of this program depend on the discursive materialization of the phenomenon.

In graphs that chart galvanic skin response of DPD and HC subjects, the DPD subjects are divided into two groups; one group that is not currently undergoing pharmacological intervention, and a larger DPD group including both drug free subjects and subjects undergoing pharmacological intervention. The galvanic skin response curves of these two groups are remarkably similar in shape, but the drug free group expresses an average .15 microsiemens increase in galvanic skin response. The peak conductance level exhibited by drug free DPD subjects reaches around .5 microsiemens, so this difference is significant. In the discussion section, the authors note the fact that "not all participants were medication free" as a limitation of the study, but they go no further in describing the significance of this feature of the study on the data.

Additionally, the authors note the limitation that "we employed an excerpt from a well-known video. Therefore, prior knowledge could have affected semantic and episodic memory." If DPD patients are less likely to have seen *Silence of the Lambs* recreationally because the film is known to be anxiety inducing, it might give

preference to HCs who are able to properly sequence the events of the clip because they have already seen the film. The considerations of possible experimental limitations by the authors demonstrate the ways in which the notions of materiality and discursivity are being employed. The feature of the experiment that gives data about medicated and non-medicated DPD patients merely expresses a lack of homogeneity amongst research subjects. For the authors of the study it does not warrant consideration as a potentially confounding factor in the cognitive feature of the study. This is a problem because it fails to recognize the possibility that these drugs are altering the cognitive abilities of DPD patients.

The fact that nearly half of the DPD patients are under the influence of chemicals meant to control anxiety is not considered as a possible reason why the DPD group expresses reduced cognitive ability. If galvanic skin response is taken to be a measure of affective response, and the galvanic skin response of medicated DPD patients is an average 33% below non-medicated subjects, then it can be concluded that the types of pharmacological medication administered to these subjects heavily blunts their affect.

The effect of mood states on cognitive abilities has been studied with respect to several different disorders and circumstances. Several studies that have been published suggest that emotional states have important cognitive outcomes. (Bartolic, Basso, Schefft, Glauser, & Titanic-Schefft, 1999) (Melcher, Born, & Gruber, 2011) (Cohen, Henik, & Mor, 2011) Additionally, the drugs that that were administered to experimental subjects may have altered their cognitive functioning

during the memory tasks. The publication lists the drugs that were administered to the DPD patients in the study. They include sertraline, venlafaxine, quetiapine, ramelteon, tranylcypromine, donepezil, and lamotrigine. At least two of these compounds has been found to impair cognitive and memory performance. One study concludes that "ramelteon (8mg)[36] significantly impaired driving performance, cognitive, memory, and psychomotor performance." (Mets et al., 2011) Another, entitled "Donepezil Impairs Memory in Healthy Older Subjects" also demonstrates the possibility that psychopharmacological interventions administered to experimental subjects may have affected data. (Balsters et al., 2011) I raise these considerations not for the purpose of rejecting the conclusions of the experiment, but to draw out the complicated features of employing psychophysiological and cognitive measurement techniques within the frame of a single experimental apparatus. Each measurement technique employs a different discursive-material context, and these contexts express different manifestations of the disorder that are not necessarily compatible. The aspect of the apparatus that materializes meaningful data regarding galvanic skin response is sensitive to the effects of pharmacological intervention, and shows usefully that the affects of medicated vs. non-medicated DPD patients is distinct. The other aspect of the apparatus expresses cognitive features of DPD patients as opposed to healthy controls as measurably distinct, but this data is disclosed in a way that may vary in relation to the psychophysiological features of the apparatus. The authors of the study claim that "the inclusion of both

[36] 8mg is the same dosage received the by subject of the DPD cognitive experiment.

psychophysiological and cognitive measures" is an important strength of the experiment. While the attempt to include two different research programs in a single study is admirable, it also changes the material-discursive manifestation of the disorder by uniting them in a single experimental configuration. There is no issue in principle with this, as all experimental designs reflect different considerations, but it is important for experimental designers to carefully foreground these issues in publication. The results of the experiment are meaningful in relation to the boundary enactments that are imposed as parameters, and these enactments warrant careful discussion in the literature.

Employing a distinction between the material and the discursive that takes one as ontologically prior to the other, an observer of this experiment might be tempted to say one of two things. An observer who holds that discursive practices produce material reality might suggest the differing discursive formations that were employed produce two different, possibly conflicting materialities of the disorder; one psychophysical and the other cognitive. This observer might be tempted to claim that the data is problematically situated by the fact that these two materialities cannot coexist.

Conversely, an observer who holds the view that the material features of the phenomenon of depersonalization are prior to the discursive practices that describe it might conclude that there is a single material phenomenon being described in two different ways. To hold neither the material nor the discursive features of the experiment as ontologically prior might lead to the view that different material-

discursive formations are capable of producing entangled realities for the disorder. On this view these realities only become stable according to momentary boundary enactments expressed by the engagement of agents in experimental practices.

The approach employed by the scientists is silent with regard to these issues, and it might be concluded that this is the proper attitude for scientists to take with respect to data. However the data produced by the experimental configurations in this study have consequences for the treatment of DPD patients, showing how the epistemic practices of the science have ethical outcomes. The psychophysical and cognitive approaches employed in the aforementioned paper provide different enactments of the phenomenon of depersonalization disorder that are neither mutually exclusive nor unified.

The second paper Dr. Simeon has recently published is a clinical paper describing a psychoanalytic approach to the treatment of DPD. (Guralnik & Simeon, 2010) This paper brings in an array of concepts from the practices of psychoanalysis that are anathema to the goals produced by scientific psychiatry. The paper attempts to situate the phenomenon of depersonalization and specifically Depersonalization Disorder in a discursive context in order to make recommendations for the treatment of DPD individuals. In particular it is the assertion of the authors that depersonalization emerges in the "spaces between" recognition and interpellation, where "recognition" is taken to be the disclosure of the self within the gaze of the other, and "interpellation" is an interpretation of the sense employed by Althusser, used "to capture the process by which one is recognized by the State to become part

of the social order." (Guralnik & Simeon, 2010) The authors of the paper go deeply into describing the relationship between interpellation and the discursive structures of our culture. Their view of depersonalization holds that the phenomenon is caused by a problematic relation between a "self-in-itself"[37] and the features of our socio-political discourse that disavows important features of that self. The use of the term "discursive" in this paper is closer to the sense that would take discursivity as ontologically prior to materiality. The central case study of the article focuses on a subject who has found it difficult to cope with the various expectations that U.S. culture has placed upon her and her body.

The suggestion for treatment of depersonalized patients emerging from this clinical paper calls for a "listening anew." This approach takes account of the therapist's position within the discursive structures of our culture (as a mediator and intervener) in order to allow the patient to reinterpret the shame produced by cultural confinements and emerge as a person who functions as an integrated subject. This call for a new kind of listening from the therapist offers different possibilities for the treatment of depersonalization that move beyond the scientific psychiatric understanding of mental disorders as confined to neurobiological processes. The approach taken in this paper that asks therapists and patients to consider the social context of the disorder is valuable in that it considers how

[37] The authors of the study specifically mention Kant's thing-in-itself when they invoke the concept of the self-in-itself and state their belief that "individuals come with a quasi-natural load or *matter*." The invocation of Kant carries the purpose of allowing the authors to remain silent regarding a full description of this primordial self that is constituted by discourse but which also has a material expression.

discursivity affects materiality, however it remains handicapped by that fact that it presumes these contexts can be readily differentiated from one another.

The challenge for psychoanalysts who want to find new ways for effectively treating depersonalized people is to make their suggestions function on a register that can be accepted by mainstream scientific psychiatry. If therapists working within the psychoanalytic tradition believe they have found effective ways to treat depersonalized people that cannot be achieved by psychopharmacological approaches, then the task will be to create a new kind of treatment that invites the larger psychiatric community to understand the disorder in a way that is open to this kind of treatment.

One problem for psychoanalytically influenced clinicians is that by thinking of materiality and discursivity as fundamentally separate facets of reality, they re-inscribe the very distinction that allows their work to be ignored by mainstream psychiatry. Because they focus on the discursive aspects of the phenomenon, without explicitly considering the material features of the phenomenon, they not only make a philosophical mistake, they also preclude the possibility of being understood by the tradition of scientific psychiatry. Recent publications discussing the integration of neuroscientific data and psychoanalytic practices suggest that there is interest in pursuing a course that reconciles or "integrates" these approaches. (S. Castellanos, 2010) (de Lima, 2010) (Hershberg, 2011) It will be important for such researchers to find a way to avoid privileging either material or discursive approaches in this research. I return to these possibilities in more detail in section 7.

4.) The Emergence of Depersonalization Disorder as a Neurobiological Phenomenon

Significant advances have been made in DPD research within the past decades, beginning in earnest in the 1990s. In 1997 Simeon and other researchers used the Dissociative Experiences Scale to examine 30 cases of Depersonalization Disorder, subsequently expanding the study group to a larger scale 117 subjects in 2003. (Simeon et al., 1997) (Simeon et al., 2003) These studies focused particularly on the differentiation of the disorder from other diagnostic categories. The authors conclude that the data supports "the conceptualization of depersonalization disorder as a distinct disorder with a characteristic course that is independent of mood, anxiety, and personality symptoms." (Simeon et al., 1997) The fact that the study uses the Dissociative Experiences Scale (DES) is significant because that scale was first devised by scientists in order to measure and stabilize Multiple Personality Disorder (MPD), now called Dissociative Identity Disorder. Ian Hacking has devoted an entire book, *Rewriting the Soul*, to exploring the development and history of MPD, and pays special attention to the DES.

Hacking's discussion of the DES focuses on its development amongst a small group of psychiatrists, noting that this community struggled with mainstream psychiatry to have the diagnosis accepted. Those who have read Hacking's analysis of the DES are likely to pay close attention to the way in which this self-rating scale is being used to measure Depersonalization Disorder. Hacking himself concludes

that while these rating scales are a common technique employed to achieve psychiatric objectivity, he suggests that the objectivity they confer is only stable if the measurement technique is calibrated to the shared judgments of the entire psychiatric community. (Hacking, 1995) Having undergone numerous revisions on its path to becoming a well validated psychiatric measure, the DES has become more widely accepted since being the target of Hacking's critique. It is unclear, however, that these revisions would assuage Hacking's concerns that the scale was not calibrated to the shared judgments of all or even most of the psychiatric community.[38] Indeed, the statement advanced by Simeon that "clinicians experienced in the diagnosis and treatment of dissociative disorders can recognize depersonalization symptoms without much difficulty"(Simeon & Abugel, 2006) is consistent with Hacking's concern that the stability of some dissociative disorders might be maintained by a special community of therapists. The specific concern is that those clinicians who focus on dissociative disorders are more likely to diagnose a patient with DPD through clinical interview.

In discussing the employment of the DES in the early measurement and differentiation of DPD, Simeon noted that every disorder is in some sense "haunted by its history." (Simeon, 2009) The more recent acceptance of the stability of the diagnostic category by mainstream psychiatry may have been somewhat hindered at the outset by its association with MPD. The creation of a new measurement scale for

[38] The DES has been validated to a greater degree since the time Hacking's comments were published, but this process of validation has been carried out mostly by researchers who work predominantly in the field of dissociative disorders.

DPD is therefore one of the most important steps for the stabilization of the diagnosis.

In order to understand this new rating scale we must turn our attention to the work of Mauricio Sierra, head of the Depersonalization Research Unit at King's College, London. Sierra has, like Simeon, explored several different approaches to the disorder during the course of his career. Importantly, Sierra is one of the first proponents of the "cortico-limbic disconnect hypothesis," which is currently the best confirmed neurobiological theory of DPD. He is also one author of the Cambridge Depersonalization Scale, the first well validated self-rating scale devised specifically to measure DPD. In what follows I will explore the history of the stabilization of Depersonalization Disorder through the various apparatuses that have allowed it to materialize in accord with the cortico-limbic disconnect hypothesis. Considering the development of this hypothesis and the practices which have helped it to stabilize as a well confirmed theory will allow us to better understand the orientation of the diagnosis within the formation of scientific psychiatry.

The publication of the first study validating the Cambridge Depersonalization Scale (CDS) marks a crucial development for DPD research. (Sierra & Berrios, 2000) The CDS is considered more effective for the measurement of DPD than the DES for a number of reasons. The abstract of the paper introducing the CDS touches on many of these features. The first sentence of the abstract notes the "dubious face validity" of other rating scales, also asserting that these scales fail to address the "phenomenological complexity" of DPD. The second beneficial

feature of the CDS is that it is able to reliably differentiate individuals clinically diagnosed with DPD from patients with other disorders including anxiety and temporal lobe epilepsy, which also frequently carry dissociation symptoms with them. The CDS therefore offers further support for the claim that DPD should be considered a separate and stable diagnostic category. The CDS correlates well with the Depersonalization Subscale of the DES, therefore demonstrating its superiority for the measurement of DPD. Finally and perhaps most importantly, the CDS boasts high reliability and internal consistency measures, and can therefore "be profitably used in both clinical and neurobiological research." (Sierra & Berrios, 2000)

The fact that the CDS and DES are self-rating scales – they pose questions to the subject regarding his or her subjective mental experience – means that they are only the first step in achieving full stability in the context of scientific psychiatry. If a neurobiological theory of the disorder is the ultimate aim of scientific psychiatry, then the marks left by test subjects on a piece of paper do not create an indisputable psychophysiological representation of the disorder. While Hacking suggests in *Rewriting the Soul* that "empirical psychology has created its own genre of objectivity" by means of the self-rating scale, the standards of objectivity for scientific psychiatry have come to demand neurobiological explanation. (Appelbaum, 2005) It is for this reason that the authors of the CDS take special care to mention the implications of the rating scale for further neurobiological research. As this examination of the measurement practices and the neurobiological theory of DPD progresses, it will be important to return to the roots of our current

understanding of the disorder in these self-rating scales. As the neurobiological description of the disorder emerges, the way it has become possible to speak of it in neurobiological terms has been de-historicized. Possibly in part to escape the haunting associations of MPD, and perhaps also to give a picture of the disorder in strict material terms, DPD scientists focus less and less frequently on the significance of self-rating scales in research. The new forms of evidence used to stabilize the neurobiological perspective constitute a response to a new set of requirements for objectivity in psychology.

One relevant insight to emerge from Hacking's analysis of MPD in *Rewriting the Soul* is his development of Foucault's distinction between *savoir* and *connaissance*. Hacking interprets Foucault's use of these words to differentiate ideas that he says are roughly akin to Chomsky's distinction between "depth grammar" and "surface grammar" respectively. Hacking devises the concepts of "depth knowledge" and "surface knowledge" in order to differentiate between "particular items counted as true, or as false" (*connaissance)* as opposed to the "underlying set of rules that determine…what is up for grabs as true-or-false" (*savoir).* (Hacking, 1995)

One of the most important features of the past decade of DPD research is that it has had to align itself with a new type of *savior* in psychiatric research. No longer satisfied with the self-rating scales employed by older forms of empirical psychology, scientific psychiatry has produced new rules for what kinds of sentences admit of truth or falsity in psychiatric research and practice. Hacking

draws on Foucault's insights on psychiatric discourse in order to color this distinction. Foucault says that *savior* can be conceived as

> "a group of elements that would have to be formed by a discursive practice if a scientific discourse was to be constituted, specified not only by its form and rigor, but also by the objects with which it deals, the types of enunciation that it uses, the concepts it manipulates, and the strategies that it employs." (Foucault, 1972a)

Foucault's discussion of discursive practices here is important for properly understanding the significance of self-rating scales in the stabilization of DPD. The sentences that admit of truth and falsity in the logic of scientific psychiatry both emerge from and produce the material expressions of the disorder. While the current picture of the disorder is expressed foremost in its materiality, the emergence of this materialization is disclosed in its discursive context. This is important for the understanding of the stabilization of the diagnostic category. In order to become a widely accepted diagnosis, the phenomenon of DPD must be brought to express itself in a way that fits with the rules of scientific psychiatry. While other disorders, such as ADHD, have not proven difficult for researchers to express according to these rules for various reasons (e.g. clearly effective pharmacological treatments), DPD researchers have had to explore other means for stabilizing a material expression of the disorder.

5.) Pharmacological Intervention and the materialization of DPD

Throughout the history of DPD research various pharmacological interventions have been tested, yet none of them have proven particularly

successful. Even in the early stages in which DPD research was directed at differentiating the disorder from other diagnostic categories, researchers noted that DPD had proven refractory to SSRIs and benzodiazepines; two classes of pharmaceutical common in the treatment Axis I mood and anxiety disorders. This fact has created a special context for Depersonalization Disorder. Because the medications usually used to treat depression or anxiety are not effective for treating DPD, this provides additional evidence for the view that chronic depersonalization should be regarded as a separate category of disorder. (Dell & O'Neil, 2009) However, it is also in part because of this feature of the disorder that such a complex apparatus of measurement has had to be constructed in order to stabilize the category. As my previous chapter on ADHD suggests, a successful pharmacological intervention can provide an extremely powerful basis for producing a neurobiological picture of a disorder, thus situating it effectively within the logic of scientific psychiatry.

It is also important to reflect on the fact that although DPD is refractory to pharmacological *treatment*, a small but significant percentage of DPD cases fall under the heading of "drug-induced depersonalization disorder." Several different intoxicants including cannabis, MDMA (ecstasy), ketamine, psilocybin, and LSD have been known to trigger both short-lived and chronic cases of depersonalization. (Mathew et al., 1999) (Simeon, 2004) (Sierra, 2009) While this data is well documented, it occupies a problematic position within mainstream psychiatric research. Each of these compounds is listed as a "schedule-I" controlled substance

with the U.S. federal government Drug Enforcement Administration, and is therefore extremely difficult to obtain research permits and IRB approval for. Even if research into these compounds were easily permitted by the government, administering these compounds to research subjects is difficult to justify ethically, precisely because they are believed to induce unpleasant and/or debilitating psychological states. Despite the fact that John Halpern has consistently established the minimal negative neurophysiological effects of MDMA on users who avoid other illegal drugs, obtaining FDA permits for therapeutic research using illegal compounds remains extremely difficult. (Halpern, 2004) (J. H. Halpern et al., 2011) (P. H. P. Halpern et al., 2011) The Multidisciplinary Association for Psychedelic Studies (MAPS) is currently conducting successful clinical trials for treating Post-Traumatic Stress Disorder by employing MDMA in a clinical therapeutic setting. Despite MAPS's successes in this area, research into pharmacological compounds relevant to DPD is hindered by governmental controls.

For these reasons DPD research has not been able to rely directly on pharmacological data in order to advance its goals with respect to the category. While researchers have been able to gesture towards pharmacological evidence in order to make general claims about the neurobiological correlates of the disorder, the situation does not lend itself readily to systematic research. As Simeon has expressed it "several neurotransmitter systems have been implicated in Depersonalization Disorder, although evidence for each is scant and partly indirect." (Simeon, 2004)

The recent document published by the DSM – 5 dissociative disorders subcommittee notes that "In a review of the (limited) psychopharmacological treatments for DPD, Sierra suggested that possible agents and neurobiological targets for DPD might include CB1 cannabinoid agonists, k-opioid agonists, NMDA antagonists, and 5HT2C agonists." (Spiegel et al., 2011) The compounds described in this list are not readily attainable in the human pharmacopeia. The development and production of compounds that affect the central nervous system is extremely expensive, and so these drugs are not produced in large quantities, if at all. This situation underscores the widely held belief that the pharmacological treatment prospects for DPD are not good. Reverse engineering a legal drug from illicit drug data is extremely difficult.

An example of this difficulty is the situation surrounding the amino acid N-methyl-D-aspartate (NMDA), which mimics the neurotransmitter glutamate in the brain. Glutamate is present throughout the brain areas thought to be relevant to DPD. Ketamine is an NMDA agonist which can induce both chronic and transient depersonalization experiences. The effects of Ketamine can be attenuated through pre-treatment with the anti-convulsant drug lamotrigine, which is an NMDA *ant*agonist. While small scale studies treating DPD patients with lamotrigine did show promising results in a few subjects, the placebo controlled trial did not produce conclusive data. The promise of lamotrigine was such that Sierra devoted several studies across five years to studying its effects. (Sierra et al., 2001) (Sierra et al., 2003) (Sierra et al., 2006) The compound is still being studied, but the current

data is such that many studies will have to be produced to resolve current conflicts. (Aliyev & Aliyev, 2011)

Many other attempts at finding pharmacological interventions for DPD are developed in a similar fashion. Researchers consider which drugs can induce depersonalization, explore neurobiological information about the effects of these drugs, and then find out whether compounds that inhibit the same neurotransmitters these drugs promote can be developed. For instance, recent papers have noted that "the k-opioid receptor agonist enadoline has been shown to induce feelings of depersonalization, suggesting that the development of k-opioid receptor *ant*agonists may have potential therapeutic applicability."(Reutens et al., 2010) In a recent phone conversation, Simeon noted that "a kappa-opioid antagonist would be interesting to try" but that there are significant barriers in the way of a development of such a compound. Simeon commented further

> "It's a matter of market and cost; the pharmaceutical companies have made big announcements in recent years about cutting central nervous system (CNS) drug development because they are the most difficult to develop and test. [They are] unpredictable and uncertain and complex and costly and you can spend decades and get nowhere. They're cutting back in that category of drugs and there you have little depersonalization disorder in the midst: it's not schizophrenia and it's not depression…"(Simeon, 2011)

The development of new drugs that can be reverse engineered from the effects of illicit compounds is becoming less and less likely because it is not a cost effective proposition for pharmaceutical companies. Although the human pharmacopeia is extremely diverse, it seems that the neurobiological circumstances of DPD happen to be of the type that is difficult to alter pharmacologically. The treatment of DPD using psychotherapeutic and other means has a substantial amount of literature, but these means are more difficult to measure neurobiologically.

Brain scanning techniques used to measure the efficacy of psychostimulants on ADHD brains have provided a significant amount of the neurobiological data that supports the catecholamine hypothesis of ADHD. While interesting studies have produced data about the brain regions implicated in depersonalization experiences, they do not provide the specificity required to develop a complete neurobiological picture of the disorder. (Reutens et al., 2010) For instance, PET and fMRI measurement techniques are effective for measuring blood flow to various brain areas, and this has produced valuable information about the neural processes of drug-induced depersonalization. (Mathew et al., 1999) Theories of ADHD, however, have benefitted tremendously from such measurement techniques because the psychostimulant treatment of ADHD lends itself easily to repeated measurement by brain scanning devices. It is at least partially for the lack of effective pharmacological interventions that DPD researchers have had to pursue other means for constructing and supporting a picture of the disorder that fits with the standards of evidence required by scientific psychiatry. One such unconventional means is the focus on the phenomenological stability of the disorder.

6.) The Phenomenological Stability of Depersonalization Disorder

In the array of measurement practices in psychiatry, the exploration of the "phenomenological stability" of a mental disorder is a less frequently employed technique. Shortly after publishing the paper announcing the creation and validation of the CDS, Sierra also published his research into the historical

manifestations of the disorder.[39] The stated objectives of this study are to establish the stability of the disorder throughout the 20th century and to explore symptoms of the disorder that are potentially relevant to a neurobiological description. (Sierra & Berrios, 2001) Understanding the phenomenological presentation of the disorder is important for the development of the neurobiological view because the cognitive and affective experiences of patients must be consistent with brain regions claimed to be implicated in the disorder. The authors of the study are also concerned that "theoretical biases" have played a role in clinical descriptions of the phenomenon, which may be one reason for the low rates of diagnosis of the disorder in recent decades.

Additionally, the discussion of phenomenological stability is intended to create a space for expanding the description of the disorder beyond that which is contained in current editions of the DSM and the International Classification of Diseases (ICD).[40] Both manuals currently contain only a single distinct symptom criterion,[41] which describes patients as having "feelings of unreality" or having "persistent or recurrent experiences of feeling detached from, and as if one is an

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[39] The publication of the Cambridge Depersonalization Scale also notes that the historical study was important for the development of the CDS and the symptoms clusters contained within it.

[40] The ICD is the standard diagnostic text used by European psychiatrists.

[41] The other criteria are negative criteria or criteria meant to distinguish the disorder from other disorders e.g. "The depersonalization or derealization symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., complex partial seizures). The depersonalization or derealization symptoms are not restricted to the symptoms of another mental disorder (e.g.,schizophrenia, panic disorder, acute stress disorder, posttraumatic stress disorder, major depressive disorder, or another dissociative disorder)."

outside observer of, one's mental processes or body (e.g., feeling [as if] one is in a dream)." (APA, 2000) The dearth of accurate descriptions for the symptoms of the disorder may be one explanation for the under-diagnosis of the disorder. (Simeon & Abugel, 2006) Simeon also recently commented that the description of patients as being "an outside observer of mental processes" (Simeon, 2011) is problematically vague for the purposes of effective diagnosis of the disorder. For this reason the dissociative disorders subcommittee for the DSM – 5 has recommended that additional descriptors including "perceptual alterations; emotional and/or physical numbing; distorted sense of time" be added to the description of the disorder in the DSM – 5. (Spiegel et al., 2011) These additional descriptors are intended to make the diagnosis more "user friendly," and thus easier for clinicians to apply. It is likely that these new descriptors will be adopted in the DSM - 5. While evidence for separate diagnostic criteria is not strong enough to add to the official category, researchers hope that additional descriptors will make the disorder easier to identify.

By exploring the clinical descriptions of the disorder which have emerged in the last century, DPD researchers effectively expand the lexicon of psychiatric manifestation for the disorder. Additionally, because each symptom of the disorder may have a distinct neurological expression, it is important to accurately describe and document each feature of the phenomenon so that more effective neurobiological research can be carried out. (Sierra & Berrios, 2001)

One interesting feature of this historical meta-analysis is the discussion of the "theoretical biases" that have caused inconsistent symptom reporting over the

course of the 20th century. The study divides cases of depersonalization using the year 1946 as a boundary, and then compares these two groups to a 3rd group, which has been diagnosed with DPD using a clinical interview. The year 1946 is chosen as the boundary by the authors because they suggest it marks a shift in the theoretical views of clinicians regarding depersonalization. Before 1946 the authors claim that clinical views of the disorder were dominated by the theory advanced by Krishaber, who suggested the disorder was explained by "pathological changes in the sensory apparatus" and in particular the visual modality. For this reason the symptoms of visual derealization were reported more frequently than other symptoms of the disorder during this era. Additionally, a view put forward by Schilder seems to have heavily influenced the clinical community towards a visual metaphor for describing the disorder, and the authors of the study claim that descriptions created during this era suggest "the presence of a theoretical bias favoring vision." For these reasons, the authors believe, the sensory features of the disorder were given special attention during this era, whereas after 1946, more attention was given to other cognitive features of the disorder, such "mind emptiness" and "temporal derealization."

Despite the fact that these theoretical biases have been uncovered, the authors conclude that the most distressing symptoms of depersonalization are spontaneously reported throughout the 20th century, and that these should form the core symptoms of the definition of depersonalization. The issue of theoretical bias in clinical observation is relevant to this discussion because it can help us to

understand the techniques by which the neurobiological theory of the disorder is being developed. Depersonalization Disorder materializes in alignment with a set of discursive practices, and the work done to establish the phenomenological stability is a central discursive practice. The phenomenological stability paper concludes with the following thoughts:

> "Two lessons can be drawn, one that there is no such a thing as an atheoretical psychopathological description; the other, that the identification of theoretical biases is essential to the understanding of both the structure and frequency of symptoms and for current neurobiological research on depersonalization."(Sierra & Berrios, 2001)

While I agree that any theoretical biases in psychopathological description ought to be well understood, I have some concerns about how the authors of this paper use the concept. First, the means by which the phenomenological study was carried out warrants further attention. The authors note that they did not include each case of depersonalization they uncovered during their historical research. They describe the exclusion criteria they used in the following way:

> "The original cohort of 275 cases was pared down to 200 by excluding reports with: a) a poor phenomenological description (poor is defined here as a report that mentions less than 3 components of the syndrome), and b) insufficient information to satisfy the first three DSM-IV criteria for depersonalization disorder. (Criterion D, that "the experiences does not occur exclusively during the course of another mental disorder..." was not used as the objective of the study was to obtain as complete a clinical description of the depersonalization phenomenon as possible.)" (Sierra & Berrios, 2001)

The last sentence is particularly important because later in the paper the authors of the study claim that all the case reports they employ meet the criteria for DSM diagnosis of depersonalization disorder. This claim is puzzling, since by abandoning "criterion D" they do not exclude cases that might be diagnosed with some other disorder if they were diagnosed today, as per the rules of the DSM which state the diagnosis of DPD should not be applied if depersonalization symptoms

appear in the context of another disorder. (APA, 2000) Another issue with these exclusion criteria is that clinicians consider it irresponsible to diagnose a patient that they themselves have not personally interviewed. The authors of the study explicitly refer to clinical diagnosis through interview as the "gold standard" of diagnosis. Therefore, claiming that all the subjects in the historical sample could be diagnosed with DPD is cause for some concern. Later in the same article the authors consider the possibility that

> "Because cases come from different diagnostic epochs, it could also be argued that they do not refer to the same disorder. However, because all historical cases met DSM-IV criteria for depersonalization disorder, this is unlikely to be the case." (Sierra & Berrios, 2001)

This is not strictly true, considering that one important feature of the DSM-IV criteria for the disorder is the one which prohibits conferring the diagnosis on individuals suffering from other disorders. The exclusion criterion, "criterion D" was left out of the phenomenological study, meaning that many of the cases included in the study might currently be diagnosed as some other disorder in which the symptom of depersonalization occurs. Although it's not clear what effects these selection practices may have on the phenomenological description that ultimately emerges from the document, the authors themselves would have to admit that the psychological description they produce occurs in relation to their own theoretical biases. Their stated goal at the beginning of the paper is to produce a phenomenological description of the disorder that will allow them to do more detailed neurobiological research. In this way, the phenomenological description accords itself with the rules of neurobiological research. The symptom clusters which ultimately get included in the CDS are based on this phenomenological

description. Symptom clusters are important to the discussion of the validity of the disorder because they provide the most promising link the neurobiological foundations of the disorder. As philosopher Hanna Pickard has noted

> if the division of symptoms into clusters can be validated, the process of discovering an underlying, scientific property, which is not only correlated with the cluster, but potentially explanatory of it, can begin. Cognitive models for clusters of symptoms can be constructed. Bridges to neuroscience can then be built. And causal and development conditions can be explored. A focus on clusters of symptoms opens the door to scientific explanation, in a way that a focus on categories of mental illness currently does not. (Pickard, 2009)

The authors write that the questions on the CDS are explicitly devised "based on a comprehensive study of the phenomenology of this condition." (Sierra & Berrios, 2000) The symptom clusters that will later be verified in 2005 by applying the CDS to DPD patients all fit with the neurobiological perspective that was first published by Sierra and Berrios in 1998 in a paper called "Depersonalization, Neurobiological Perspectives." (Sierra & Berrios, 1998) (Sierra et al., 2005) The neurobiological perspective was developed before the symptom clusters that describe it were officially created. The emergence of the cortico-limbic disconnect hypothesis is concomitant with the evidence that is used to support this view. The researchers who first presented this hypothesis then set about clarifying and supporting it with the experiments they carried out.

The two papers using the CDS to differentiate symptom clusters of the disorder are published in 2005 and 2007, several years after the CDS and phenomenological stability papers are published, yet the creation of the symptoms clusters actually began before the publication of the CDS. This is not especially surprising, given that the clusters must first be created before they can be verified; however the measurement technique by which they are verified, the CDS, was

devised with these symptom clusters in mind. The first of these papers, authored by Sierra and Berrios, provides evidence for four distinct symptom clusters including 'Anomalous Body Experience ', 'Emotional Numbing', 'Anomalous Subjective Recall' and 'Alienation from Surroundings'.(Sierra et al., 2005) A second paper, authored by Simeon et al. distinguishes the symptoms of 'physical or emotional numbing', 'unreality of self', 'perceptual alterations', 'unreality of surroundings', and 'temporal distortion or disintegration'.[42] (Simeon et al., 2008) These symptom clusters accord very well with the neurobiological view offered in the 1998 paper by Sierra and Berrios. The phenomenological stability research done into the disorder not only stabilizes Depersonalization Disorder historically, it stabilizes it in accord with the neurobiological perspective that has come to be the touchstone of psychiatric objectivity. There is nothing wrong with this, as a neurobiological perspective is helpful for understanding the disorder for future research. It should be noted however that the material context in which the disorder is disclosed requires a particular discursive structure in order to take form. Whether or not this discursive structure amounts to what should be called a "theoretical bias" is not what is at issue. Instead we might recognize that the material-discursive formation of neurobiology grounds the cortico-limbic disconnect hypothesis from the outset. Depersonalization Disorder thus emerges as a neurobiological phenomenon because the process of stabilization for the disorder has been carried out that way.

[42] While it might seem problematic that the two papers contain a different number of clusters, Dr. Simeon has pointed out that this degree of agreement is actually extremely promising at such an early stage.

The various features of the cortico-limbic disconnect hypothesis (e.g. the phenomenological/historical data, the neurological data, the rating scales etc.) are not really separate forms of data. Instead, each form of data is enmeshed and implicated in the others. The phenomenological history of the disorder helps to determine the symptom clusters. The rating scales confirm the distinctness of those same symptom clusters. The symptom clusters help to pinpoint neural processes and direct diagnostic practices for further research. But the neurological view of the disorder existed well before any of the supporting evidence was produced. The disorder materializes neurobiologically because the research program that has been carried out is designed that way.

Had the set of practices described in the psychoanalytic research that focus on the configuration of social practices gained greater attention from the psychiatric community the diagnostic category of DPD might have different guidelines for diagnosis. The same could be said for cognitive-behavioral approaches. This is not to say that the neurobiological and psychoanalytic approaches are incompatible, indeed research in both areas is in many cases carried out by the same scientists. Both Simeon and Sierra have published many studies from neurobiological and psychological approaches. From their perspective these approaches constitute a unified research program. However, only by looking at the nuanced relationships between different experiments and publications can the developments of a scientific research program be understood. The guidance scientists take from a discursive formation, neurobiological, psychoanalytic or otherwise, allows the phenomenon of

the disorder to manifest itself in certain ways. These manifestations are made distinct by the practices of treatment associated with each formation. Neurobiological approaches favor pharmacological intervention and psychological approaches favor talking treatments.

Scientific research arrives at its outcomes through the practices of one or more material-discursive formations. These outcomes have implications for the success or failure of such fundamental structures as the diagnostic categories themselves. Data produced within experiments connected with the neurobiological formation guides the rhetorical practices that have allowed a small group of scientists to convince the psychiatric community of the usefulness of the diagnosis. The data regarding phenomenological stability of the disorder is used by Simeon in her book *Feeling Unreal*.

> "Despite the differences between modern medical practice and that of the nineteenth century, many of the earliest observations remain valid with few, if any, modifications, largely because what was being described by patients then is described so similarly today. Depersonalization is what it is, then and now."(Simeon & Abugel, 2006)

Establishing the phenomenological stability of Depersonalization Disorder constructed through a history of clinical reports is an important step in creating a discursive context which allows the disorder to materialize as a neurobiological phenomenon. The phenomenological description of the disorder must be fixed in a consistent way in order for the materiality to emerge within the formation of scientific psychiatry. The neurobiological features of the disorder have proven difficult to measure through more standard techniques such as psychopharmacological intervention, behavioral observation or brain scanning

techniques. This has meant that the disorder has been somewhat resistant to a neurobiological perspective. The context in which the disorder traditionally expresses itself is basically confined to the clinician's interview, and this has created a special discursive formation for the study of DPD. The neurobiological perspective has been supplemented in many ways by psychological research into the disorder, and this has meant that it is regarded differently than some other mental disorders. These distinctions come to the fore when DPD is compared with a more mainstream disorder like ADHD.

While DPD has expressed extraordinary phenomenological stability, the diagnostic category of ADHD has had no fewer than three categorical predecessors in the DSM, which might suggest a lack of phenomenological stability. Through the history of the DSM the heading for the shifting symptom cluster we now call ADHD has changed from minimal brain dysfunction, to hyperkinetic reaction of childhood, to ADD, to ADHD with various numbers of subtypes. The proposed changes for the DSM – 5 include another alteration of subtypes. This history might suggest that the phenomenological stability of ADHD is weak, and thus its status should be challenged. However, phenomenological stability does not matter for the overall stability of the diagnostic category of ADHD. There is a widely effective pharmacological treatment and clear behavioral symptoms associated with that disorder, which allow it to materialize uniformly in different contexts. In the case of DPD, since such measures are largely unavailable, different techniques of discursive

materialization need to be employed to in order to produce a neurobiological description of the disorder.

7.) A multiplicity of perspectives and new possibilities for treatment

While there is strong evidence that DPD patients have been experiencing a similar group of symptoms for many decades, the neurobiological description of the disorder does not dominate DPD research the way it does with ADHD. In addition to the neurobiological research, a diverse array of research from other theoretical perspectives persists. The stability of the disorder in neurobiological terms has been effectively established using both current and historical clinical reports, self-rating scales and available pharmacological data, yet the neurobiological perspective is not associated with an effective treatment, and thus leaves open space for psychological approaches. One material-discursive formation manifests the disorder according to neurological descriptions, but because a psychological description may actually be more effective for treatment possibilities it occupies a prominent place in the literature. This is distinct from the situation surrounding ADHD, in which psychological interpretations of the disorder have mostly abandoned. In the case of ADHD the neurobiological picture and its requisite treatment, psychopharmacological intervention, form a unified interpretation which circumscribes the perceived need for psychological interpretations.

The development of a neurobiological picture tends to rely heavily on pharmacological data, as is the case for other disorders such as depression, anxiety

as well as ADHD. The data that has been used to produce the neurobiological picture of DPD lacks a major feature, effective pharmacological intervention. As a result, the neurobiological literature has placed greater emphasis on the relationship between drug-induced DPD and illicit compounds. The possibility of inducing the disorder through pharmacological means provides an important link in the neurobiological picture, although unfortunately not as a means of treatment.

Studies have concluded that drug-induced DPD is not significantly different from DPD that emerges in other contexts. (Simeon et al., 2009) While this conclusion has been largely accepted by DPD researchers, the studies that compare drug-induced DPD to other forms of the disorder have developed different conclusions about the context in which drug-induced DPD emerges. (Reutens et al., 2010) One study concluded that patients were likely to experience DPD after undergoing a "bad-trip" under the influence of an illicit chemical, (Simeon et al., 2009) while the other focused on a link to previously existing anxiety disorders in patients. (Medford, Baker, et al., 2003) These two conclusions express different views of the disorder and the context in which it emerges, and these views are both accepted equally by the DPD research community. One view suggests that the trigger is due to psychological trauma induced by the drug, while the other suggests it relates to hereditary psychological characteristics in interaction with the drug.

While the two views are not mutually exclusive, they propose a different framework under which to make sense of the evidence, and neither is strictly neurobiological in nature. Dr. Simeon has noted that drug-induced DPD may be

more consistently understood as being caused by "psychological triggers" rather than purely neurochemical events. (Simeon & Abugel, 2006)

One meta-analysis paper ultimately concludes that the findings produced by the drug-induced DPD studies are consistent with the view that DPD emerges from an "interaction between psychological and neurobiological factors." (Reutens et al., 2010) In the discussion section of the 2003 paper exploring drug-induced DPD, the authors consider several possibilities for the origins of drug-induced DPD. They suggest that DPD that emerges in drug contexts may be the result of the altered psychological states that can be produced by these chemicals, but also consider the possibility that a direct psychopharmacological effect produces the depersonalization symptoms during the experience, which then have prolonged neurobiological effects. It may be argued that the willingness of DPD researchers to consider both neurobiological and psychological perspectives is the result of a general lack of understanding regarding the disorder, but more can be said. The differing material-discursive contexts in which DPD manifests also emerge from and produce different treatment recommendations for the phenomenon. Psychological and neurobiological treatment options for the disorder are still being explored alongside one another.

The cognitive-behavioral model and treatment suggestions for DPD have been studied in earnest by the same researchers who have worked to produce a neurobiological description of the disorder. In 2003 Hunter, Phillips and Sierra, some of the pioneers of the neurobiological perspective, developed a thoroughgoing

cognitive-behavioral model for understanding and treating DPD. In his recent book *Depersonalization: A New Look at a Neglected Syndrome* Sierra says of the cognitive-behavioral perspective that "the lack of quantified scientific studies makes it difficult to assess the value of (cognitive-behavioral) approaches used." (Sierra, 2009) While the cognitive-behavioral treatments for DPD may have received slightly less attention in publication in the past decade, they have not been abandoned. This is far different from the situation with ADHD, where only a small minority of researchers still promotes an integrated treatment paradigm.

The view expressed in the cognitive-behavioral research suggests that Depersonalization Disorder may be caused by cognitive processes similar to those described in individuals who exhibit panic disorder: symptoms that occur normally in individuals are interpreted by the patient as catastrophic, and thus intensify into still more distressing symptoms. One example given for individuals with panic disorder is that they experience shortness of breath as impending suffocation. Similarly the paper suggests those who experience DPD may associate feelings of depersonalization, which occur at some time in the life of 70% of the population, with a fear of impending insanity or death. These "catastrophic attributions" are cognitive states that cause DPD sufferers to believe that "something has gone wrong with my brain," and indeed one study found that 80% of DPD patients surveyed would assent to this statement. (Hunter et al., 2003) The view that the disorder emerges from fears that a certain symptom or experience are indicative of a life-threatening or sanity-threatening condition casts a different light on some other data

about individuals with DPD. First of all, the paper notes that a significant percentage of DPD patients have immediate relatives who suffer from mental disorders, which may cause them increased fear of insanity. Similarly, those who have experienced "bad-trips" under the influence of illicit chemicals also frequently report a fear of impending death or insanity, which may link the psychological factors of these experiences to long term DPD. Finally, it is interesting to note that the neurobiological picture of the disorder affirms the claim "something has gone wrong with my brain" to DPD sufferers. A neurobiological theory of mental disorders attempts to describe the phenomenon in terms of abnormal brain function, which is a precise expression of one fear that may actually have a hand in triggering chronic depersonalization. These considerations invoke the question of the relationship between the brain and the mind, but are difficult to address within the space of psychiatric publication.

While the cortico-limbic disconnect hypothesis has guided research through the decade, several data points have emerged in conflict with this neurobiological perspective. Daphne Simeon's overview of the disorder notes that subsequent data collected regarding the disorder is "partially in accord and partially contradictory" with the cortico-limbic disconnect hypothesis. (Dell & O'Neil, 2009) In the face of this, Simeon returns to what she believes is the core symptom of the depersonalization experience: that of unreality. She suggests that understanding the disorder requires "identifying the core feature" of the experience, which she reasserts is unfamiliarity with surroundings. Following this she reasserts that the

two brain areas implicated in this experience involve "limbic structures and sensory association cortical areas." (Simeon, 2004) While this assertion is much more general than the cortico-limbic disconnect hypothesis proposed by Sierra, it implicates the same brain regions. Simeon's recent publication of the clinical paper exploring psychotherapeutic treatments from the position of psychoanalysis suggest she is willing to explore other discursive materializations of the disorder, since the current neurobiological approach is providing little help for her patients.

Simeon's interest in both psychological and neurobiological views opens the possibility for treatment practices that hybridize the different material-discursive formations involved with the knowledge of DPD. A new therapeutic approach to the treatment of depersonalization might be one that challenges that distinction-between or primacy-of different material-discursive approaches. One serious difficulty here will be to create a dialogue that considers material-discursive processes in a way that also retains the stability of the diagnostic category among mainstream psychiatrists, who tend to favor the neurobiological view. Because Simeon has worked for a long time in both traditions, and is one of the most well-recognized researchers of Depersonalization Disorder, she is in a unique position to foster the dialogue between the scientific psychiatric and other, psychological approaches. Simeon has stated explicitly that "novel therapeutic approaches are clearly needed to help individuals experiencing this refractory disorder." (Simeon, 2004) If done carefully the consideration of the discursive materialization of DPD could allow researchers and clinicians to create a "hybrid approach" to treating

DPD. In order to create such a hybrid approach various options might be considered.

Looking first to the "integrative approach" that has been employed for Axis I disorders such as depression and anxiety we can see models that simply employ both psychopharmacological intervention and psychotherapy concomitantly. This integrative approach employs psychopharmacological and psychotherapeutic techniques in ways that complement each other, but do not inherently change either practice. One integrative approach being studied by the Santa Cruz non-profit organization MAPS uses psychopharmacological intervention *only in the context* of psychotherapeutic intervention, and alters the practices associated with each in order to fit this very different approach to treatment.

It is not only Simeon's recent publications that show a diverse set of approaches to the phenomenon of DPD. Her publication record shows a career that is committed to understanding and treating DPD through whatever means show promise. Of her 40 publications available on the "Web of Knowledge" database, roughly half focus on neurobiological data, while the other half focus on psychological, cognitive-affective or psychoanalytic approaches. By looking into the material-discursive formations that have been produced and considering the ways in which drug therapy and psychological approaches might be used not just alongside one another but through one another, new treatments may yet emerge from a hybrid understanding of the disorder.

The psychoanalytic approach employed by Simeon and colleagues, like the cognitive-behavioral approach employed by Sierra and colleagues, represent a willingness to explore new material-discursive practices for producing knowledge about the disorder. These approaches differ distinctly from each other and from the neurobiological perspective in the kinds of treatments they recommend for the disorder, and there is no reason why one approach ought to be closed off in favor of the others unless it produces a treatment program that is shown to be effective for most DPD patients. It is possible that the current material-discursive formations will never produce an effective treatment as they are currently conceived. Perhaps only through exploring the interrelations between these approaches and moving beyond them can an effective approach be devised. It is also possible that the integrative approach that employs both pharmacological and psychological intervention will be the most effective. Insofar as the psychological and neurobiological approaches employ different assumptions about the relationship between brain and mind, it might seem that these issues would have to be resolved before a unified approach can be conceived, but this is not necessarily the case.

Philosopher/psychiatrist Dan Stein has used the term "brain-mind" in order to continue working while remaining agnostic about the precise relationship between the separate concepts of "brain" and "mind." The view from cognitive-affective neuroscience, the approach that Stein espouses, offers an interpretation of the disorder that takes the neurobiological and psychological features of the disorder

at the same value, thus offering an integration of perspectives that may prove very promising.

Four main theoretical approaches have been the most conspicuous in DPD research over the past two decades. The first is the neurobiological perspective that focuses most carefully on what can be said about the function of the brains of DPD patients. (Sierra & Berrios, 1998) (Phillips et al., 2000) (Phillips & Sierra, 2003) (Simeon et al., 2008) (Simeon et al., 2009). The second most common is the cognitive-behavioral approach which considers the thoughts that appear in the minds of DPD patients, and how they might best be treated through cognitive-behavioral therapy. (Hunter et al., 2003) (Heidenreich, Michalak, & Michal, 2006) (Guralnik et al., 2007) Less frequent approaches include the cognitive-affective neuroscience approach, which attempts to draw together neuroscientific and cognitive data in order to form a broader picture of the disorder, (D. J. Stein & Simeon, 2009) and the psychoanalytic approach, which explores the possibility of treating the disorder using psychoanalytic therapy. (Guralnik & Simeon, 2010) While it might be proposed that these various approaches do not alter our understanding of the disorder, I will suggest that each one provides a slightly different discursive materialization of the disorder. Each of these approaches considers evidence for the disorder from a different perspective, and draws different conclusions on how treatments might be carried out. While it is clear that none of the aforementioned perspectives need preclude or proscribe the others, it is valuable

to explore the ways in which the practices that produce knowledge about the disorder cast it in a different light.

The multiplicity of discursive formations used to study the disorder is connected to the materiality of the disorder in many interesting ways. From a philosophical perspective, it might be argued that the current diversity of approaches is connected to the incompleteness of our neuroscientific capabilities. Insofar as the linguistic practices associated with non-neurobiological approaches are required to describe the experiences connected with the onset of DPD, it seems irresponsible to force the conclusion that a purely neuroscientific explanation is a more valuable standard. This may align me more with Feyerabend than the Churchlands, but ultimately I would suggest a pragmatic consideration grounded in the notion of effective treatment is the one that should prevail with respect to the status of various sciences in their knowledge production regarding psychiatric phenomena.

8.) DPD and DSM - 5

The forthcoming edition of the DSM has been surrounded by tremendous controversy. The attempt made in the DSM – IV to achieve an atheoretical description of mental disorders was carried out in order that disorders could be identified without suggesting or requiring any particular treatment program. Allen Frances, Chair of the DSM – IV task force has been publishing a blog called "DSM – 5 in Distress" on the website *Psychology Today*. The blog connects the issues he sees

with the emerging DSM – 5 to his own experiences in leading the publication of the previous edition. In one post Frances focuses on the proposal advanced by some psychologists that a single theoretical model might underpin the entire structure of psychiatric diagnosis. He divides these proposals into three groups; brain biology, psychological dimensional models and psychodynamic/ethological models. Of these he says

> "Unfortunately, none of these approaches, however elegant, is remotely ready for inclusion in the official system of psychiatric nomenclature. DSM must by its very nature be a conservative document that follows and never leads the field. The problem with all of the suggestions to replace the admitted DSM jumble is that there are so many contenders, none of which has been proven or has attained wide acceptance from the field." (Frances, 2011)

In our interview, Daphne Simeon also noted the desire expressed by the psychiatric community to move towards a single theoretical approach for the DSM. She said "Supposedly the DSM – 5 was going to be the first instrument that was to be more informed by neurobiological and other theories." (Simeon, 2011) Instead, the DSM – 5 will attempt to avoid allowing symptom criteria to be influenced by any particular psychiatric theory. Simeon notes, however, that in the description of the disorders there will be more information about what is known about these disorders from a neurobiological perspective. The descriptors that are being included with respect to DPD emerge from the neurobiological perspective insofar as they connect to the symptom clusters developed in the Cambridge Depersonalization Scale. On the other hand, the notes about treatment practices are likely to emerge from a psychological perspective. In this sense the new edition of the DSM will be multi-theoretical rather than atheoretical. It will be helpful if these theoretical associations are foregrounded within the text in order for the diagnosing clinician to be more

effective at picking out the perspective underpinning any particular suggestions made regarding the disorder.

Looking at the process by which the diagnostic category of DPD is generated, it can be concluded that the definition of Depersonalization Disorder contained in the upcoming edition of the DSM emerges from a "joint commitment," in the sense that Margaret Gilbert describes in her work in the field of social epistemology. In her chapter "Remarks on Collective Belief" (Gilbert, 1994) Gilbert describes and elaborates upon her notion of the "joint commitment." A joint commitment is a marker of collective intentionality insofar as the belief formed or held by a group that has entered into the joint commitment need not accord with the belief of any single individual within that group.[43] As Simeon says of the DSM – 5 classification for DPD, "although no one agrees one hundred percent there is a great effort to come to a consensus." (Simeon, 2011) Each member of the dissociative committee holds a slightly different perspective with regard to the category of DPD, yet they must be willing to abandon certain features of this perspective when it comes to the task of creating a description of the disorder for the purposes of diagnosis. This unified description may not accord completely with the views of each individual, yet the practice of joint commitment allows a single diagnostic category to be forged.

[43] The simple example provided by Gilbert describes the parents of a teenager, George, who are setting a curfew for their son. George's father thinks he should be home by ten. George's mother does not feel that George needs a curfew. Together they agree to set George's curfew at midnight. Their joint commitment holds them collectively to the belief that George's curfew should be midnight, although individually neither one believes this. The joint commitment can only be broken through mutual consent, and if either individual alters their belief without consent, the other has recourse to social rebuke.

The joint commitment that allows various researchers of dissociative disorders to assent to a single diagnostic category is one of the most important pluralist moves made for DPD research. Because there are many perspectives on the disorder being explored in terms of its proper etiological description and clinical treatment, it would be problematic to attempt to restrict the diagnostic category to a single theoretical framework or context. Instead, the diagnostic category of DPD reflects the diverse theoretical engagements that discursively materialize the phenomenon in various attempts to understand and treat it.

The purpose of this case study has been to demonstrate how a multiplicity of approaches, which I have described in more detail as being material-discursive formations, have been used to develop the diagnostic category of Depersonalization Disorder. Although these approaches are contained under a single diagnostic heading, it should be concluded that there are disparate discursive materializations of the disorder. This distinction is evidenced by the disparate (and thus far unsuccessful) treatment approaches to the disorder. New approaches to treatment need not collapse the different material-discursive formations into one another, but instead should attempt to foreground the ways that formations produce treatment approaches, thus bringing out the assumptions implicit in treatment plans that have been produced to date. Further thoughts in this area are issued in the concluding chapter of this dissertation.

Conclusion: Producing Healthier Realities through Active Genealogy

This concluding chapter is comprised of two sections. In the first section I present some ideas on rethinking causality in psychiatric neurobiological etiology. In the second section I discuss the practice of "active genealogy" I have employed in writing the case studies that form the core of this dissertation.

Part I: Psychiatric Neurobiological Etiology and Causality

The notion that psychiatric disorders are "caused" by neurobiological states warrants scrutiny. The neurobiological descriptions of psychiatric disorders that are becoming the focus of psychiatric etiology often lack any reference to the exogenous determinants of psychiatric phenomena. Although psychiatric research does sometimes mention the relationship between e.g. genetic factors and environmental factors these relationships are often under-theorized in the context of neurobiological description. This can be seen in the case of ADHD research, where meta-analysis has demonstrated the predominant focus on endogenous determinants of mental disorders such as brains and genes. (Nigg, 2003) Throughout this dissertation I have examined the troubled and problematic nature of causal descriptions of psychiatric disorders by examining the epistemological practices that guide psychiatric research and clinical treatment. The expression of neurobiological etiology which focuses on neurochemicals, brain regions, and genes fails to pay heed to the environmental, psychosocial, and other exogenous factors that play a role in psychiatric phenomena.

Psychiatric researchers have voiced concerns about the reductionist assumptions that play a role in many findings, worrying that this type of presentation will have troubling outcomes for clinical practice. As Thomas Fuchs has said

> The simple bottom-up explanation of mental disorders as products of genetic or neurophysiologic determinants is inadequate to the causal complexity involved. Instead we ought to develop etiologic models that are based on a *circular causality* between organism and environment, with the brain acting as a mediating entity. (Fuchs, 2005)

While I prefer the idea of co-constitution to the one of "circular causality" that Fuchs proposes here, this excerpt helpfully expresses the concern amongst members of the psychiatric community that the move toward neurobiological etiology fails to pay proper heed to the causal complexity of psychiatric disorders. In a response to Fuchs's piece philosopher Drew Leder has highlighted the need for "new ways of understanding the etiology and treatment" of psychiatric disorders. (Leder, 2005) The following section develops some philosophical ideas for rethinking psychiatric etiology and causality.

My case study of ADHD discusses the emergence of the catecholamine hypothesis and shows that this description of the disorder could not have been generated without the practice of psychostimulant medication. The evidence that the phenomenon of ADHD cannot be separated from the history of its treatment practices demonstrates an issue with the way neurobiological causality is currently taken to function. We cannot understand ADHD as being simply "caused" by a

catecholamine deficit if the main evidence that supports the claim is that psychostimulants are effective in treating ADHD. This is a matter of being ethically responsible for scientific epistemological practices. The boundaries enacted within experiments that generate evidence for the catecholamine hypothesis exclude the environmental, psychosocial and other exogenous correlates of the disorder, and can thus be challenged on these grounds. However, in order for this challenge to work, there must be a notion of causality that is sensitive to the complexity of psychiatric phenomena.

Various ADHD researchers have contended that non-pharmacological evidence-based treatments for ADHD are being overlooked in favor of drug treatments. (Pelham & Fabiano, 2008) (Gonon, Bezard, & Boraud, 2011) Arguments advanced by these researchers could be supported by focusing on the way that environmental, architectural and other psychosocial factors are being under-theorized in the course of experimental design. It is becoming increasingly common for scientists to voice concerns about the effect that the catecholamine hypothesis is having on treatment research and clinical practice. One research group has recently expressed dismay at the fact that the dopamine-deficit theory (essentially a sub-species of the catecholamine hypothesis) has misguided research to favor psychostimulant medication.

> Unfortunately, the dopamine-deficit theory of ADHD is so dominant that it discourages the human and financial investments needed to explore alternative theories. Moreover, whatever the hypotheses, neuroscientists should be more aware of the fact that neurobiological theories of ADHD do influence its social representation and, thus, its treatment. The main drawback of the dopamine-deficit theory is that it gives scientific credence to a view that favors psychostimulant medication over other medical, psychological and social approaches to ADHD treatment. (Gonon, 2009)

Exploring other possibilities for causality will help psychiatric researchers to become more attuned to alternative possibilities for measurement, thus allowing for the generation of different types of data about non-drug therapies. The research I have done not only foregrounds the fact that the catecholamine hypothesis relies on psychostimulants, it also proposes alternative experimental configurations that would be accountable to this situation. It thus provides the theoretical basis for further study in this area.

There are clear points at which the ethical context of the disorder is co-constituting with the data that is produced about the disorder, and this relationship has outcomes for etiological determination. Frequent treatment of ADHD with psychostimulants creates a context for the disorder where these same drugs are employed in the production of evidence supporting the catecholamine hypothesis. This hypothesis concomitantly justifies the treatment of ADHD with psychostimulants at the expense of other treatment practices, making the justification for psychostimulant treatment self-sealing. The current notion of causality in psychiatry creates a situation that allows researchers to exculpate themselves from the ethical consequences of that hypothesis. If ADHD is taken to be simply caused by a deficiency of the same chemicals that psychostimulants promote, then psychostimulants are the most reasonable treatment option. This story emerges because a notion of neurobiological causality exists that allows psychiatric researchers to produce etiological descriptions that pay no heed to the ethical commitments entangled with the practices of scientific measurement.

Making space for alternate theories of ADHD

In order to challenge the view of psychiatric disorders as "products of genetic or neurophysiologic determinants" we can begin by considering the notion of etiological determination that grounds this view. Insofar as neurobiological description has become the new standard for psychiatric etiology, psychiatrists are incentivized by their epistemic community to generate evidence that aligns with such a view. In this context, symptoms of psychiatric disorders are taken as being caused by the abnormal functioning of neurobiological (neurophysiological and genetic) systems. In this etiological context there is a unidirectional notion of causality. In short, the accepted view is that abnormal brain processes cause psychiatric symptoms. Aside from not paying heed to exogenous determinants of mental disorders, this view makes the further mistake of treating the patient's brain and the patient's symptoms as separate phenomena that can be clearly distinguished from one another. As Fuchs and Leder propose, new etiological models based on different notions of causality are needed.

Karen Barad's theory of Agential Realism proposes a notion of causality that does not consider a causal relation to be "a relation between distinct entities." According to Agential Realism the relata do not precede their relation. Instead, objects are disclosed insofar as boundaries are enacted within phenomena and the relation becomes fixed as part of scientific practice. Barad proposes that the notion of "intra-action" replace the standard notion of "interaction" so that the theorist can

consider all the processes occurring within an undifferentiated phenomenon. The concept of intra-action allows us to consider phenomena as unbounded and entangled outside the context of boundary enactments.

This stance can provide a new basis for understanding psychiatric etiology. This view allows us to recognize that the properties of psychiatric phenomena are made determinate by the experimental configurations employed to produce data about those phenomena. Insofar as neurobiological description is the target of psychiatric research, boundaries are enacted that produce particular data sets to the exclusion of others. Considering psychiatric etiology from this position allows theorists to understand how mental phenomena are constituted as neurobiological by the boundary enactments that take place in psychiatric research.

If we consider the phenomenon of a psychiatric disorder first as undifferentiated from the world, it will be much easier to properly theorize the multitudinous factors at play in its becoming. In particular, fully understanding the discursive materialization of ADHD requires us to consider classroom and work environments, psychosocial features, relationships between patients, teachers, clinicians, researchers, pharmaceutical companies, and the political economy etc. that are part of the phenomenon. Only once these features can be seen as a whole can the results of boundary enactments that produce psychiatric etiology be accounted for. The ontological consequences for the notion of causality produced within these practices can be effectively theorized by considering entangled ethical-epistemological engagements. As Barad says "the larger apparatus is causally

significant." By carefully considering the apparatuses of psychiatric research my dissertation attempts to provide an alternative account of etiological determination that is properly aware of the causal complexity of psychiatric phenomena.

The apparatus must be unearthed

I use the term "apparatus" as part of a philosophical tradition that is interested in examining the relationship between scientific data and experimental practices. The key figures I draw on to understand this relationship include Michel Foucault, Ian Hacking and Karen Barad. Each of these theorists employs an approach that draws out different insights about the meaning of data in relation to experimental configurations, and as such I discuss different features of psychiatric apparatuses at different moments in this section.

Michel Foucault uses the term '*dispositif*' (translated as 'apparatus') to develop a conceptual technique for understanding how science produces knowledge in certain ways. In particular Foucault thinks of the apparatus as an

> ensemble consisting of discourses, institutions, architectural forms, regulatory decisions, laws, administrative measures, scientific statements, philosophical, moral and philanthropic propositions–in short, the said as much as the unsaid. (Foucault, 1972b)

Foucault uses these concepts in many of his studies to help explain how a particular sub-population (such as psychiatric patients) comes to be categorized and normalized by the larger population. Foucault is chiefly concerned with the way scientific knowledge is produced by a population regarding a sub-population with the goal of treating or controlling the latter.

Foucault discusses the material configurations by which normalization and regularization are possible, but also conceives of a *discursive* component to the apparatus. When Foucault invokes "the said as much as the unsaid" he is asking us to think broadly about all of the practices involved with our knowledge regarding a particular phenomenon. This way of thinking allows us to theorize a phenomenon without the restraint of a view of science that takes scientific phenomena to be somehow separate from the practices of the scientists and other agents who are involved in the exercise of the apparatus.

As my ADHD case study demonstrates, the apparatus of ADHD is an ornate configuration consisting of the architecture of classrooms and other workspaces, the neuropsychological battery employed to confirm behavioral diagnosis of ADHD patients, the practices of psychostimulant medication, the expectations of parents, teachers and students in school contexts, and the genetic, animal modelling and neuroimaging studies used to produce neurobiological data regarding the disorder, etc. to name just a few components. It is only in recognizing all the material and discursive features of this apparatus that scientists can account for the meaning of data produced by the apparatus. In particular, it is important to think here about hyperactive behavior as being disruptive to the standard classroom architectural model. Insofar as students are often expected to work in ways that require silent and solitary activities, the ADHD child emerges as problematic to this context, and thus needs to be dealt with. This is part of the causal story of ADHD that is excised from

or under-theorized in psychiatric experiments that generate support for the catecholamine hypothesis.

It would be a mistake here to mention the panoptical architecture of the classroom and halt with an interpretation of classrooms according the function of disciplinary power. Although classroom architecture does mold and inscribe bodies in particular ways, it is the function of regulatory power that is most relevant to the context of ADHD. "Regulatory power" (also referred to as "bio-power") is theorized by Foucault to express the evolution of disciplinary power in the sense that it medicalizes abnormal behaviors and requires performance to align with certain standards. If ADHD emerged in a fully disciplinary context, ADHD patients would be removed from the normal population of school children. Instead, it has become a value within western education that children with special needs are integrated into mainstream classrooms and expected to perform at the requisite level. It is also in part because of this that the practice of psychostimulant medication has emerged as the most effective treatment for the disorder. It is both a cognitive enhancer and reduces hyperactive behavior, thus fulfilling both the disciplinary and regulatory needs of the classroom.

Hacking's book *Representing and Intervening* provides a detailed understanding of how scientific phenomena are *produced* in the context of experiments. (Hacking, 1983) Hacking observes that, with the exception of a very few phenomena, most of the processes studied by science require a set of instruments constructed by scientists in order generate data. Hacking is mainly

concerned with the *material* aspects of the apparatus. My examination of the galvanic skin conductance measurements of DPD patients as compared with healthy controls as they watch a clip from the film *Silence of the Lambs* generates some Hacking-style observations. Inducing DPD patients to enter a state of intense depersonalization in the laboratory requires careful contrivance. Hacking might urge us to ask questions about how the creation of a phenomenon in a laboratory context affects our understanding of it. What does it mean, for instance, that the subject is asked to sit perfectly still and to try not to blink as the film clip plays? This kind of measurement may not accurately reflect the phenomenon of DPD as it occurs outside a laboratory setting, simply because this setting produces the phenomenon in a specific way, thereby generating data that reflects warped expressions of the disorder. For instance, the fact that many DPD patients in that study were asked to carry out memory tasks while under the influence of drugs that are known to impede proper memory function may be producing data about the disorder that improperly characterizes the experience of temporal distortion that some DPD patients suffer from.

In the case of ADHD, Hacking's work on the apparatus helps the theorist to pay attention to the material context in which the phenomenon appears. Recognizing that ADHD emerges and is diagnosed mostly in classroom contexts reminds us that this is a disorder that is particularly relevant to the way that pedagogy and work are carried out. The materiality of the apparatus is configured according to the needs of the education system that trains students for work in

similar physical spaces e.g. cubicles in offices. The way that space is divided produces particular manifestations of the disorder that are not considered relevant to neurobiological research, and are thus not considered to be causally efficacious in the phenomenon of ADHD.

The term "apparatus" has been used by Barad in a way that moves beyond the conceptions developed by Foucault and Hacking. In Barad's view the primary ontological unit is not *object* but *phenomenon*. Through differential boundary enactments psychiatric phenomena are isolated from the world. Only by abandoning certain assumptions can a new philosophical position be developed that allows scientists to ensure that their work does not ignore important aspects of the phenomenon under investigation. Barad defines 'apparatus' in her book *Meeting the Universe Halfway* as an unbounded set of "material-discursive practices," in which boundaries are enacted to produce and measure phenomena. (Barad, 2007) The hyphenation of the words "material-discursive" draws attention to the idea that these two terms cannot be disentangled. Instead matter and meaning are co-constituting in Agential Realism, and this point of departure opens a new avenue for understanding psychiatric disorders.

Barad's suggestion that "Discursive practices and material phenomena do not stand in a relationship of externality to each other"(Barad, 2007) but rather intra-act so as to have outcomes in the way each appears to us in scientific inquiry helps us to escape theoretical systems that take either discourse or matter as primordial.

By properly considering the role of boundary enactments scientists can become accountable to the meaning of scientific data in the context in which it is generated. By focusing, for instance, on the fundamental role that pharmacological compounds play within the measurement of psychiatric phenomena one can make explicit the co-constituting relationship between neurobiological descriptions and drug treatments. The psychopharmacological interventions for ADHD are co-constituting with the neurobiological description of the disorder. When the boundaries between psychopharmacological intervention and neurobiological description are not enacted, the disorder cannot materialize in the catecholamine deficient way. For scientists to be ethically responsible in this situation they must make this state of affairs explicit when publishing the results of research. In this situation boundary enactments could be collaboratively theorized, critiqued, and altered in ways that produce new types of evidence. If such a practice were to become the norm of psychiatric research, the problems voiced by the psychiatrists in the first section could be addressed more effectively. Insofar as the health of an epistemic community means responding to the needs of its members, the new notion of causality I have presented produces a healthier reality for the psychiatric research community.

On Barad's view, scientists are required to consider their own role within the apparatus as well. Within ADHD research there is much to consider regarding the role of pharmaceutical companies and their financing of drug research. Currently, the main standard required of scientists is to reveal the sources of their funding in

the "conflict of interest" statement contained in research publications. Several studies have demonstrated that research funded by pharmaceutical companies is "strongly associated with pro-industry results." (Sismondo, 2008) (J. R. Lexchin, 2005) (J. Lexchin, Bero, Djulbegovic, & Clark, 2003) The question remains whether or not the statement of "conflict of interest" really constitutes an adequate foregrounding of boundaries. Some theorists have argued that research conducted in the context of a conflict of interest should simply be prohibited by regulatory bodies. (J. Lexchin & O'Donovan, 2010) However, the extreme cost of drug development is claimed to preclude the possibility of other sources of funding. One theorist has proposed that conflict of interest statements do little to preclude the possibility that data will be properly understood or interpreted, proposing instead that greater attention be paid to the actual experiments themselves. (Borgert, 2007a) Recognizing instead that "the complete removal of bias from scientific experiments is impossible" Borgert instead proposes transparency of the scientific process whereby scientists are required to make all data relevant to the experimental configuration available for review. (Borgert, 2007b) For Borgert relevant data includes "experimental design, data collection, criteria for interpretation, laboratory notebooks, raw data, and the statistics used." This view fits well with the idea that the apparatus must be fully unearthed in order for scientists to be responsible for the data that is produced within experiments, however some crucial features have been left out of Borgert's list. These include the philosophical and theoretical assumptions employed by scientists when designing experiments as well. Insofar as scientists

must be accountable to the boundaries enacted within research, it should be a matter of course that participation in the psychiatric epistemic community requires full inclusion and active theorizing of all personal theoretical commitments and assumptions.

The insights of Foucault, Hacking and Barad concerning the apparatus have guided my examination of ADHD and DPD at every turn. I have examined scientific experiments down to their finest detail. I have explored the naturalcultural contexts of both disorders in an attempt to sketch out a conceptual tool that can be usefully applied to scientific investigation. This conceptual tool consists first in detailed examination of boundary making practices with experimental configurations, second in the consideration of the meaning of data in relation to the apparatus, and third in the challenging of boundaries that are enacted uncritically to produce unhealthy realities.

By showing how non-material aspects of science have results for the conclusions produced by scientific research, Foucault's accounts of the apparatus are meant to make the reader aware of the discursive complexities of scientific endeavor. However becoming aware of the discursive complexity does not afford the theorist a complete tool for understanding the complex causality at play in psychiatric neurobiological etiology. Barad extends Foucault's work by moving beyond the concept of bio-power/regulatory power to understand the intra-action of apparatuses with other apparatuses. In her consideration of the piezoelectric transducer that is part of the apparatus of ultrasound imaging, Barad demonstrates

how the apparatus both allows phenomena under investigation to materialize in certain ways, but also materializes temporally within the practices which it helps to constitute. A complete understanding of the relationship between ADHD and psychostimulant medication must be aware of the ways in which the materiality of stimulants are constituted in the context of ADHD. Ultimately Barad updates Foucault's view by moving past the notion that it is only human bodies that are constituted by the exercise of apparatuses. In Agential Realism the "apparatuses are themselves material-discursive phenomena, materializing in intra-action with other material discursive apparatuses." (Barad, 2007) In the context of ADHD research, this helps to elucidate the alterations in legality and efficacy that psychostimulant compounds have undergone since they were first administered to children with behavior problems in 1937. In the next section, I discuss the alterations of legality of ADHD medications and their diversion to illicit use within student communities in the context of the disorder. I also consider how ADHD research has led to the recognition of psychostimulants as "cognitive enhancers" and how this new label has in turn altered the materiality of the phenomenon of ADHD.

Hacking's view of the apparatus, although by no means naïve in its realist leanings, attempts to restore confidence in the stability of scientific knowledge and practice. Hacking's restraint in conceiving of the apparatus as fixed laboratory instruments is problematic, however, in that this view fails to recognize that "apparatuses do not possess inherent outside boundaries limiting them to laboratory spaces or experimental practices." (Barad, 2007)

Barad's view moves beyond both Foucault's and Hacking's conceptions of the apparatus by avoiding the fast distinction between materiality and discursivity. Both accepting the social features of the apparatus but refusing to reduce experimental design to a purely social practice, Barad shows us that "apparatuses are neither neutral probes of the natural world nor social structures that deterministically impose some particular outcome." (Barad, 2007) This view allows us to accept the claims produced by scientists as valid, but this acceptance requires a much more detailed understanding of the meaning of experimental configurations. By foregrounding the boundaries enacted within experiments by scientists, patients, physical spaces and drugs, my analysis shows how psychiatric data is produced and psychiatric disorders discursively materialize as they do.

When scientists disagree about the conclusiveness of data, they often speak about the ways that experiments have been "biased" in favor of one conclusion or another. I have urged that the language of bias is becoming less helpful in scientific discourse. Every boundary enacted within an experimental configuration opens some possibilities and precludes others. There is no way to achieve a completely "objective" experiment, and this recognition is valuable in moments where scientists are critiquing experiments. When scientists suggest that an experiment is "biased" in favor of a particular outcome, they imply that there is some neutral position from which scientific data can be produced. Only by foregrounding the boundaries enacted within experiments can scientists become responsible for the data that is produced. My case studies provide numerous examples of how boundary making

practices can be foregrounded. If researchers can be brought into dialogue to discuss how boundary making practices produce scientific data, more collaborative and sensitive experiments can be designed. In the space of the Science and Justice Working Group I have brought researchers into conversation with one another in order to take part in such dialogues.

The "genealogical accounting of material-discursive practices" that Barad calls for and I employ allows philosophers and scientists to see how ADHD and DPD have emerged as diagnostic categories through the intra-action of human and non-human agencies. Greater awareness of the context in which measurements are situated allows scientists to resolve disputes more amicably by collaboratively generating new experimental configurations in which theoretical assumptions are foregrounded. While scientists themselves might not hold a position of naïve realism this view is sometimes built into experiments. If scientists do not explicitly account for the boundaries enacted by the apparatus their experiments can be misinterpreted as un-problematically measuring an objective feature of the world. Instead each experiment measures a phenomenon that is differentiated from the world according to a particular configuration. My analysis of experiments in ADHD and DPD research shows how these configurations have ethical consequences for the way that measurements are produced.

The undifferentiated and entangled process of becoming that is the world does not offer itself to science with necessary cleavages. In order to be studied by science a phenomenon must first be differentiated from other phenomena using

material-discursive boundaries. The apparatus is the means by which science differentiates the phenomenon of concern from the rest of the world. By theorizing the apparatus scientists can become explicitly aware of how boundary making practices situate the knowledge their experiments produce.

For example, my discussion of classroom architecture as a context of measurement for ADHD examines the way that the structure of the classroom is often uncritically excised from consideration in ADHD research. I contend that much of the dispute about relevant treatment programs for ADHD might be resolved by a more careful consideration of the classroom context. When drug treatments are compared with behavioral interventions for ADHD experimental designers rarely consider the possibility of altering the architecture of classrooms. Although studies have shown that replacing standard chairs with inflatable exercise balls for ADHD students greatly reduces the most visible signs of the disorder, this is not taken into consideration in large treatment comparison studies.

As I have mentioned previously in this chapter, it is also important to see drugs themselves as part of the apparatus of measurement for psychiatric disorders. As long as scientists think of drugs only as a treatment and not part of the phenomenon of psychiatric disorders they will continue to miss the significance of the entanglement of ethics and epistemology in psychiatric research. Drug treatment practices produce a form of knowledge that justifies and even encourages the further administration of drugs. Insofar as scientists continue to look to drugs as the single solution for ADHD and other disorders, they tacitly accept that the harm they cause

is unavoidable. Unless psychiatric researchers can become responsive to the way boundaries are enacted within experimental configurations, the meaning of scientific data will be understood with incomplete information, and healthier realities will be left unexplored.

Barad's observations about indeterminacy as opposed to uncertainty are valuable in the understanding of psychiatric research.[44] Viewing psychiatric disorders as indeterminate outside their requisite context of measurement allows us to rethink the notion of causality in psychiatry. If we recognize that ADHD becomes determinate as a catecholamine deficiency only in the experimental context that employs psychostimulants, then it becomes possible to see that the boundary enactments (which include ethical decisions) made by ADHD researchers and clinicians have elevated this description to the status of scientific knowledge, thus marking catecholamine deficiency as the "cause" of ADHD despite the fact that many other causal factors exist.

[44] The debate between Bohr and Heisenberg regarding the impossibility of simultaneous measurement of the position and momentum of the electron in quantum mechanical experiments is central to Barad's agential realist view. Whereas Heisenberg expressed his uncertainty principle as a failure of knowledge, i.e. we *cannot know* the value of these two variables simultaneously, Bohr interpreted this phenomenon as indeterminacy, i.e. that *the electron does not admit simultaneously of position and momentum*. Where Heisenberg saw only a failure of measurement practices, Bohr saw an ontological feature of the universe. Barad's view suggests that indeterminacy is also true at the macroscopic level. Indeterminacy at the macroscopic level is demonstrated in Astrid Schrader's paper on *Pfiesteria piscicida*, a dinoflagellate that does not simultaneously admit of species being and toxicity. (Schrader, 2010)

The materialization of ADHD

As Barad says, "bodies differentially materialize as particular patterns of the world as the result of specific cuts and reconfigurings that are enacted." (Barad, 2007) This sentence elegantly expresses the state of affairs concerning ADHD and the catecholamine hypothesis. The body of the ADHD patient is only capable of materializing as catecholamine-deficient in the context of a practice that administers catecholamine stimulating compounds. The catecholamine hypothesis of ADHD emerges because the world is configured in such a way as to produce the phenomenon of ADHD in relation to stimulant medication. We have come to know the ADHD brain as a particular material configuration within the world because boundaries have been enacted that differentiate the phenomenon in the requisite ways.

Barad's view is best understood as a type of naturalism. (Rouse, 2004) Agential Realism shows us that the matter of bodies comes to be expressed in an ongoing performative process by which the universe is made intelligible to itself. The intra-actions of human and non-human agents become causal enactments through the process of boundary making and differential becoming. When we approach the phenomenon of ADHD and take the practice of psychostimulant medication as a practice of meaning-making, we can come to see how the drug treatment constitutes both an ethical and epistemological practice, and that these two expressions are inseparable from one another. The beneficial effect of psychostimulants for classroom and work contexts both supports the ethical

injunction to treat people with them and allows the brain of the ADHD patient to come to matter in the catecholamine-deficient way. To say simply that ADHD is caused by catecholamine deficiency does not tell the full story of the relationship.

To provide a more nuanced example, the view of psychostimulants as "cognitive enhancer" as opposed to "psychopharmacological cure" differentially determines the materiality of the disorder. Looking back at the recent history of ADHD we can see that a break has occurred between the clinical and etiological validity of the disorder. This break has occurred as a result of the debate around the function of psychostimulants on normal brains.

Very recent studies about psychopharmacological interventions have accepted that methylphenidate and dextroamphetamine are cognitive enhancers for humans. (Berridge & Devilbiss, 2011) The debate about how psychostimulants should be regarded is entangled with the materialization of the disorder as a catecholamine deficiency. Now that the "paradoxical effect" narrative has failed, and low dose psychostimulants are widely recognized to enhance cognitive capacities in all humans, ADHD has come to materialize in a new way. While the clinical validity has remained stable, the etiological validity is undergoing a shift. The language of "deficit" is no longer an effective description of ADHD symptoms, and so new discursive materializations of the disorder have emerged. ADHD researchers now point to the "dysregulation" rather than "deficit" of catecholamines in the pre-frontal cortices of ADHD patients as the cause of the disorder. To

understand why the recognition of psychostimulants as cognitive enhancers has changed the description of ADHD, some conceptual analysis is needed.

The notion of "cognitive capability" has traditionally been considered grounds on which to issue praise and blame. Those with greater cognitive ability tend to excel at school and work and are regarded as morally praiseworthy because of their achievements. If science now medicalizes shortcomings in cognitive capability and justifies the use of psychostimulants in order to alleviate these shortcomings, then the standard notion of praise and blame in this context is no longer available.

As a result, the language of deficit is no longer applicable in describing the causes of ADHD symptoms because it produces a slippery slope. If we draw a line in a continuum of cognitive capability and justify the psychostimulant medication for individuals on one side of the line but require those on the other side to complete their tasks without chemical support, we create inequalities in the potential for achievement. Those at the lowest non-medicalizable place on the continuum are at a distinct disadvantage in comparison with everyone else. If we link the measure of "cognitive deficit" to "catecholamine deficit" we essentially create an environment where larger and larger segments of the population will demand cognitive enhancements in order to remain competitive in school and work environments. If the psychiatric community alters its view of the disorder to be best described as a dysregulation of catecholamines, then the diagnosis is once again an all-or-nothing

phenomenon. No line in a continuum of cognitive capacity needs to be drawn when a "dysregulation" metaphor is employed.

The distinction between the metaphors of "deficit" and "dysregulation" may be given detail by drawing a comparison to metaphors of psychiatric disorder distinguished by Dan Stein. Although Stein distinguishes between five different metaphors, the two that are relevant to this discussion are the metaphors of "breakdown" and "imbalance." (Dan J. Stein, 2008) When psychostimulants were taken to have a paradoxical effect on ADHD brains, the disorder could legitimately be described as a deficiency of catecholamines. Now that psychostimulants are regarded as a cognitive enhancement, the disorder of ADHD expresses itself as a dysregulation rather than a deficit. The new determination of ADHD as dysregulation rather than deficit shows how the materiality of ADHD has been altered by the discursive features of drug definition.

Whereas we might consider the story of "deficit" to be a description of an imbalance of neurotransmitters in ADHD, the "dysregulation" story could be more closely aligned with the language of "breakdown." Whereas imbalance and deficit admit of degree, dysregulation and breakdown suggests an objective standard or norm has been shown to be violated. Thus the new language of dysregulation, because it invokes an absolute norm and not just a pragmatically noted deficit, allows psychiatrists to draw a principled distinction between those diagnosed with ADHD and those who are legally denied the right to use stimulants as cognitive

enhancers. In short, the dysregulation story allows psychiatrists to affirm the ADHD patient's *need* for cognitive enhancers as compared with the non-ADHD population.

The idea that ADHD discursively materializes in its current form only in the context of psychostimulant medications rejects the idea that the disorder and the drug treatment can be seen as ontologically separable. A philosopher who adheres to the ontology of separability might object that the materialization of ADHD through the administration of psychostimulants is merely an epistemological issue. Although psychostimulant medication allows us to know ADHD in this way, such a person might argue, ADHD brains would be catecholamine deficient whether we knew it or not. To take this position regarding ADHD would be to misunderstand several features of the phenomenon. First of all, it requires a misunderstanding of the temporality of ADHD by ignoring its history. If it were possible to remove psychostimulant compounds from the history of the disorder, it is not even clear that there would be such a disorder. The grounds on which the disorder becomes a legitimate diagnostic category of the name ADHD include over seven decades of psychostimulant research. Those that would adhere to a story of uncertainty, attempting to consider ADHD outside the context of psychostimulants and modern classrooms and workspaces, would essentially preclude the emergence of the disorder thereby fundamentally misunderstanding the phenomenon. The notion of indeterminacy makes a complete understanding of phenomenon in all its complexity possible. In order to make decisions that lead to healthy realities researchers,

clinicians and patients must understand what possibilities are generated by the relationship of the disorder to pharmacological treatment.

Insofar as the scientific practices that have generated the reality of psychiatric disorders have taken place in repeated contexts they open some futures and foreclose others. The continued treatment of ADHD by psychostimulant medication is an example of this. Because the neurobiological picture of the disorder has emerged in the context of psychostimulants (indeed, I contend, the context of psychostimulants is the core feature of that picture) some possibilities for treatment are closed off while other ones emerge.

The practices of psychiatric researchers that employ pharmacological interventions in order to produce neurobiological data are inseparable from the ethical practices they promote and engage in. Measuring ADHD by intervening with drugs supports and encourages the increased practice of drug treatment. The possibilities for future action are constrained by the knowledge making practices we engage in, which in turn emerge from our current possibilities for action. The ethical practice of drug treatment for ADHD provides its own epistemological justification, thus prolonging itself. Importantly, agency is not foreclosed in this context, but requires the foregrounding of boundary enactments within the apparatus and response-ability to them in order to re-open new possibilities. Just as anti-inevitability is expressed in the work of Nietzsche and Foucault, Barad's view shows us that new configurations are always available to us once existing structures are critiqued and destabilized.

Barad has shown us that "we are accountable for and to the exclusions that we participate in enacting." (Barad, 2007) Therefore, by "reconfiguring material-discursive apparatuses of bodily production, including the boundary articulations and exclusions that are marked by those practices" scientists and patients can alter the possibilities for ADHD research and treatment. (Barad, 2007)

What this means for psychiatric clinicians and researchers is that each time they carry out an experiment or treatment they are responsible for their decisions in configuring the apparatus. Good science here means not only being aware of and actively accounting for the role of boundary enactments within experiments, but actively pursuing imaginative boundary alterations. The inflatable exercise ball experiment is an example of what I have in mind here.[45]

The configuration of experimental apparatuses has important consequences for the identity of psychiatric patients. Barad says "determinately bounded and

[45] If drug studies were carried out in contexts in which architectural boundaries were also challenged, the possibilities for non-drug treatment of ADHD would be expanded. Disputes between psychiatrists regarding the appropriateness of psychopharmacological intervention such as the ones I bring up in my case study might be more effectively resolved if the language of boundary enactment could be added to psychiatric discourse. The study that focuses on the restructuring of classroom architecture in order to alleviate ADHD symptoms is relevant here. By replacing the standard foot-footed classroom chair with an inflatable exercise ball for the ADHD students, thus allowing them to release kinetic energy in a way that is considered appropriate for classroom behavior, this study enacts different boundaries within the phenomenon of ADHD. This type of material-discursive reconfiguration is promising but not well studied. The effects of psychostimulants on children are so dramatic as to eclipse the perceived necessity for such structural alterations of the classroom. The fact that psychostimulants are recognized to have several dangerous side effects for children has been offset by the view that these compounds are simply replacing a deficit of chemicals inside the ADHD brain. The picture of psychiatric disorders as occurring within the body of the patient rather than as emerging from the complete phenomenon of the patient within his or her context is one consequence of the catecholamine hypothesis and of the move towards neurobiological psychiatry in general.

propertied human subjects do not exist prior to their involvement in naturalcultural practices." (Barad, 2007) Insofar as the ADHD patient has his or her identity defined by the practices of categorization carried out in psychiatric research, their subject is being defined by their involvement within science. Recognizing first that the practices of science are contingent upon a multitude of factors that could have been otherwise, it is possible for patients to take part in the constitution of their subject by scientific research. Only by understanding the phenomenon of psychiatric diagnoses in all their complexity can those categorized by psychiatric diagnoses make fully independent decisions about how they know themselves and what practices of treatment they choose to engage in or submit to.

Part II: Active Genealogy

In this section I lay out a plan for an attitude I call "active genealogy" and distinguish it from other interpretations of the practice of genealogy. At the high level, my view is that the genealogist is justified in taking a proactive stance toward the formations he or she chooses to investigate. Because the entanglement of ethics and epistemology is often made covert in the course of scientific inquiry, it is up to the genealogist not only to uncover this entanglement, but to actively envision new possibilities that can produce healthier realities.

"Without granting as true the fictions of logic, without measuring reality against the purely invented world of the unconditional and self-identical, without a continual falsification of the world by means of numbers, mankind could not live." (Nietzsche, 1990)

Nietzsche's work lays bare the relationship between our drives as living things and our practices of knowledge production. From this perspective, the practice of science is the prolonged adaptation of a certain species-being. Nietzsche affirms "health" above all other values as a core guiding feature of his thinking. Insofar as Nietzsche also sees the practices of knowledge production as emerging from the drives of living things, he recognizes the root of the sciences as connected to the wills of its practitioners. Thus when we study psychology and psychiatry, which emerge from the desire to understand the thoughts, drives and behaviors of humans, we need to pay careful attention to the ways in which our forms of life inform our knowledge.

Carrying on the practice of genealogy that begins with Nietzsche and is developed by Foucault, my purpose has been to express the ways in which the wills of individuals and groups produce the experience of the subject.[46] In this spirit, this dissertation has endeavored to show how our pharmacological practices have shaped and produced the reality of psychiatric disorders. In particular, it has examined the measurement practices that produce data in psychiatric research, and

[46] Here I hold with Nietzsche that the subject is actually an amalgamation of drives. The fact that we refer to ourselves as a unity is an artifact of our language.

considered how these practices play a part in the production of reality and individual experience.

"How is explanation to be at all possible when we first turn everything into a picture? – our picture! It is enough to view science as an attempt to humanize things as faithfully as possible. Cause and effect: there is probably never such a duality." (Nietzsche, 2001)

Nietzsche recognizes that our logical practices emerge out of our attempt at ever more detailed descriptions. When it comes to the sciences of psychiatry and psychopharmacology, we are faced with the additional problem that the target of our knowledge is ourselves, and therefore we are faced with two mirrors when we consider questions in these areas. The form of life which medicalizes abnormal mental states makes its needs apparent within its practices of measurement and knowledge production.

The notion that the veil of nature can be removed or dissolved with science merely reminds us that what we are looking into when we practice knowledge production is actually ourselves. The notions of cause and effect and the logics that we supply for the constraint and description of reality by means of number must be held to the light and recognized as emergences of will.

Nietzsche affirmed a future where "artistic energies and the practical wisdom of life join with scientific thought so that a higher organic system will develop." (Nietzsche, 2001) It is one goal of this dissertation to explore the ways in which the notions of health might be reevaluated within the medical sciences so that new ways

of thinking and acting can develop. Above all this new practice requires the recognition of the scientist within his or her own apparatus of measurement. By seeing the instability of our thinking and ways of existing we can hopefully be open to other practices that promote the health of diverse forms of life. As Ian Hacking has so forcefully reminded us, we are constantly creating new people and new realities whenever we generate scientific data. Our expressions of knowledge are also expressions of our own will, and they take part in the shaping of experiences. Insofar as the targets of psychiatric knowledge have their subjects formed in part by the production of knowledge, these subjects must also take part in the creation of themselves. It is through the recognition of our art in science, and the practical features and consequences of knowledge production that a new organic system can emerge. This is because "artistic energies" and "practical wisdom of life" are invited into scientific thought by the practices of creativity and critique.

It is not possible to say exactly what a system that hybridizes art, practical wisdom and science will look like, but it is worth noting that the publication of the new edition of *The Diagnostic and Statistical Manual of Mental Disorders* is being generated using the input of the patients whom it categorizes. Those who have been categorized are now taking part in the process of categorization, thus taking a hand in the production of their own identities. This affirms the role of the individuals who are the objects of knowledge as producers of that knowledge. If the practice of psychiatric research can be so opened that patients, clinicians and researchers can act

collaboratively to generate new meanings for scientific data, then the wills that take part in these practices can be more fully expressed.

The recognition that the practices of science are intricately bound up with the creation of the human subject is central to this dissertation. Because each individual who is associated with a psychiatric diagnostic category is bound exist in relation to it, it is not only the right but the responsibility of that individual to interpret that category for him or herself. The practice of knowledge production within the formation of psychiatry does not lie solely with researchers, clinicians or patients. Instead, my case studies have endeavored to show how each individual within these groups acts to alter the logic of the discipline in order to be able to live with it.

The information I have brought together regarding the diagnostic categories of ADHD and DPD is meant for several purposes. First it offers a critique of the conclusions drawn by medical science in order to demonstrate the contingency of these conclusions. Second it highlights points of potential destabilization of these conclusions, should these conclusions come to be more widely considered unhealthy. Third it suggests alternative possibilities and directions that might be taken up in the production of healthier realities.

Nietzsche emphasizes the importance of names, noting that "what things are called is unspeakably more important than what they are." (Nietzsche, 2001) Insofar as the practice of generating and distributing psychiatric diagnoses is a convention of naming, it is important to be delicately aware of the duty each person has to

actively create the categories that are applied to them (either by themselves or by others.)

Nietzsche's injunction to give style to one's character, to be bound but also perfected by one's own law can be understood in the context of psychiatry as an injunction to take hold of the production of psychiatric diagnoses. Diagnoses are actively produced by communities of researchers, clinicians and patients in order to respond to a need. Whether we characterize this need as a relief from suffering, as a normalization of abnormal thoughts and behaviors, or as a response to the demands of a political economy, recognition of our active creation of these categories helps us to keep in mind the role of interests driving the production of knowledge.

In the recognition and elucidation of the entanglement of ethics and epistemology in psychiatry this dissertation aims to open possibilities for the alteration of scientific practice. By examining the places in which the logic of psychiatric disorders is constituted by those who care about those disorders, the case studies I have written carry out the Nietzschean project of creating a new kind of science.

My interpretation of Nietzsche's work is very much aligned with Foucault's, although I tend to take a somewhat more optimistic stance on the ability of individuals to create themselves in the face of power relations. A major component of the case studies that form the core of this work is the practice of genealogy. As

part of my work as a fellow of the Science and Justice Training Program, I have sometimes described my attitude towards this technique as "active genealogy." With this term I am not trying to differentiate my practice of genealogy from that of Nietzsche or Foucault, but to distinguish it from an interpretation of genealogy developed by Nikolas Rose. (Rose, 2007) In particular I want to emphasize a certain feature of the technique used by Nietzsche and Foucault that attempts to actively produce new realities. Rose consigns the practice of genealogy to what he calls "cartography," which loses the generative and active features of Nietzsche and Foucault's techniques. Foucault deftly expresses the purpose of "active genealogy" when he proclaims that "knowledge is not made for understanding, it is made for cutting." (Foucault 1971) I take this statement to be an expression of the view that active genealogy must always be employed with a disruption in mind. It can also be interpreted as the practice of producing and affirming new boundaries within the continuous phenomenon that is our reality.

Rose astutely characterizes a chief purpose of genealogy when he says "genealogies seek to destabilize a present that has forgotten its contingency, a moment that, thinking itself timeless, has forgotten the time-bound questions that gave rise to its beliefs and practices." (Rose, 2007) Active genealogy makes alternate futures possible by disrupting the knowledge practices that produce the reality of the present. Rose's suggestion that we should abandon genealogy in favor of a more conservative "cartography" is anathema to active genealogy. The idea that, instead of collapsing the undesirable features of reality, we should merely create a "map

showing the range of paths not yet taken that may lead to different potential futures" (Rose, 2007) gives the impression that our future is merely chosen from an array of fixed possibilities. While science holds things temporarily fixed for the purposes of stable measurement, it would be a mistake to reify this fixedness and think of reality as a room full of furniture. This is exactly the view of psychiatric diagnostic practice that my work attempts to avoid.

Rather than accept genealogy as a practice of cartography I prefer to think of it as the first step in an existential process. The way that psychiatric disorders manifest within the world is tied, at least in part, to the will of those who produce their realities. As John Law says "the world is not a structural something we can map with our social science charts." (Law, 2004) I prefer to follow Law and think of the world as a maelstrom that we are riding on and through, and that is also flowing through us. By recognizing our reality as a confluence of winds, unstable and constantly altering one another, we are capable of generating a science that is sensitive to the flux of reality.

On Law's view we can recognize that the paths we are currently capable of predicting with a map are but the most banal of futures, precisely because they are predictable. By taking up the notion of the maelstrom we can recognize that the future is the action of the multitudinous forces swirling amongst us, and the shape we make with our bodies in their unfixed becoming is part of the flow.

To accept Law's suggestion to follow the Red Queen from Alice in Wonderland and try to think six impossible things before breakfast is to move the

mind towards the right attitude for active genealogy. The purpose of active genealogy is to create unimaginable futures rather than just consider potential ones. Rose's claim that "our future will emerge from the intersection of a number of contingent pathways that, as they intertwine, might create something new" (Rose, 2007) demonstrates his implicit belief that the world's capacity for creating new realities is something that should be observed by humans rather than taken up as a project. Foucault's suggestion that "chance is not simply the drawing of lots, but raising the stakes in every attempt to master chance through the will to power" (Foucault, Rabinow, Rose, & Foucault, 2003) is more closely aligned with active genealogy. Like Nietzsche, Foucault never explicitly states what the future will or should be like. Nietzsche says at the end of the second treatise of *The Genealogy of Morality* "there is only one thing fitting for me, to be silent: otherwise I would be laying a hand on that which only a younger one is free to choose, a 'more future one.'"[47] (Nietzsche, 1998)

Foucault was criticized for lacking a positive political stance about what should be done with systems of governmentality, etc. that he critiqued. I interpret his book *Fearless Speech* as an attempt to explain the purpose of genealogy as a form of narrative that opens unimagined futures. (Foucault, 2001) The point of active genealogy is to see how the movements of people and things gone before participate

[47] It is not clear whether Nietzsche's references to a single person capable of affecting a revaluation of all values was more a function of his era's obsession with "great men" or a kind of attempt to inspire the reader into action.

in the currently occurring reality. Through this practice we see how current formations[48] can be disrupted, and in what ways new formations can be produced.

In generating a genealogy of the measurement practices for Depersonalization Disorder, I have worked to lay bare the difficulties of measuring a disorder that has no outward behavioral symptoms. The possibility that patients suffering from this disorder might not receive adequate care or treatment because of these difficulties should be marked as an issue for consideration. Because the current reigning logic of psychiatry requires a description of psychiatric disorders in terms of neurobiological processes, it has been difficult for researchers and clinicians to establish the validity of the category, and to obtain funding to carry out further research. The battle to retain the diagnostic category of DPD in the DSM – 5 was hard fought. If an individual suffering from this disorder was forced to obtain a different diagnosis in order to receive treatment covered by insurance, this would no doubt proliferate their suffering. Without the diagnostic category of DPD, the treatment guidelines for patients of this disorder would be altered, and the insurance practices surrounding care would have been made more difficult. While my work was not directly involved with the debate surrounding the status of DPD in the current DSM, the genealogy of the disorder I have created can be used in future debates surrounding other disorders that share similar hardships to this diagnosis.

[48] I mean the word "formation" more in the sense of birds in flight than rock formations.

Some philosophers have been inclined to draw a distinction between Foucault's earlier work including genealogies such as *Discipline and Punish* (Foucault, 1977) and *Madness and Civilization*, (Foucault, 1965) and his later work such as *The Hermeneutics of the Subject* (Foucault, 2005) and *Care of the Self* (Foucault, 1988) which focus more explicitly on the creation of the individual in relation to cultural practices of antiquity. (Koopman, forthcoming) While this distinction might help to divide some themes in Foucault's work, Colin Koopman has usefully suggested a more cohesive way to interpret Foucault. Koopman suggests that while the "anti-inevitability thesis" is well understood in Foucault scholarship it is frequently emphasized over what he considers to be the more important "composition thesis." The anti-inevitability thesis is described as the view that "whatever we take to be inevitable about ourselves is in actual fact the process of contingent historical accretion." (Koopman, forthcoming) The composition thesis is created for the purposes of helping people recognize that our possibilities for changing ourselves exist amongst our abilities to alter the processes at play in the generation of the subject. What is usually termed "Foucault's ethics" (i.e. his later work in which he examines the creation of the subject in antiquity and modernity) is perfectly consistent with his earlier genealogical work, in which he helps to foreground the processes by which the subject is constituted.

Foucault is thus, Koopman notes, always oriented towards past and future in his work, helping to uncover the possible moments for action in the present. Insofar as my case studies examine specific diagnostic categories and the current state of

research that produces and stabilizes them, I have tried to show both how these categories have arrived at their current state, and how those concerned with these categories might destabilize and reinterpret them. For instance, the research that I have brought together on ADHD uncovers the way that the neurobiological description of ADHD has been generated. Lest people come to accept that the description of ADHD determines their possible actions, I have shown that this description is contingent on particular practices of measurement and thus the recommended drug treatment for the disorder should never become an unquestioned requirement.

Similarly it is important for DPD patients to recognize that the lack of drug treatment for the disorder does not mean that they are untreatable, or that they ought to accept a diagnosis of anxiety or depression. While the lack of a drug treatment presents certain obstacles for patients diagnosed with DPD, this does not mean that patients with this disorder must suffer without respite. Instead, patients of DPD must participate in the generation of new therapeutic approaches. By understanding how the practices of medical research take part in the construction of individual realities, patients are able to take a more active role in relation to their diagnosis.

Another feature I have foregrounded in the production of psychiatric diagnostic categories is the issue of legality and illegality for various drugs that play a role. The fact that amphetamine use remains legally justified at all is in large part due to its use as an ADHD treatment. What this has meant for those suffering from

ADHD is that their disorder and the drug used to treat it are made ethically permissible by its high cultural value. Those who suffer from ADHD are the beneficiary of scientific narrative that expresses the disorder in terms of a genetic and neurobiological deficit. Through this narrative, ADHD sufferers are exculpated from any ethical shortcoming, and allowed to see themselves as innocent individuals who have psychostimulant deficient brains.

The reality of the disorder is bound up with the form of knowledge production that measures the phenomenon through the legal administration of drugs. While ADHD enjoys the status of a very stable disorder within the psychiatric community, the psychostimulant intervention has come under repeated attack partly as a result of the frequent diversion of psychostimulants to illicit use. Dr. Allen Francis, the physician who presided over the publication of the DSM-IV, recently noted that "30% of college students use stimulants to do better at school." (Francis 2010) Because many of these stimulants are provided by ADHD patients to their peers, the illicit use of stimulants in academic environments is connected to the phenomenon of ADHD.

Depersonalization Disorder (DPD) on the other hand, is still moving towards widespread recognition amongst the psychiatric community. (Sierra 2010) One reason for this is that many psychiatrists do not see the need for a diagnosis that has no clearly effective psychopharmacological treatments. One recent MD said that "diagnosis (of DPD) does not seem to be particularly useful for patients, as depersonalization symptoms are extremely resistant to both medications and

psychotherapy." (Reeves 2010) Because of this fact, DPD researchers have had to go to great lengths to find neurological evidence for the disorder, including the recent experiments with galvanic skin response to frightening stimuli. (Simeon, Geisbrecht, et al. 2010)

While DPD has no clearly effective pharmacological treatments, it can be induced by illicit drug use. A full chapter of the recently published *Depersonalization: A New Look at a Neglected Syndrome* is dedicated to "Drug-Induced Depersonalization Disorder." (Sierra 2010) This chapter contains a list of such culprits as cannabis, MDMA, LSD and Ketamine, each of which has been linked to drug-induced DPD. Each of these is classified as a "schedule I controlled substance" by the DEA, meaning that they have no accepted medical use. The very title of this book bears witness to the central issue surrounding DPD; the community who works on the disorder has trouble convincing the mainstream psychiatric community to diagnose it. Because it is very difficult to study the effects of illicit drugs on brains in any systematic way, the objectivity of DPD is not helped by its relationship to drugs. Also, because the disorder is sometimes induced through illicit drug use, a stigma regarding the drugs in relation to the meaning of the disorder persists. Understanding how psychopharmacological practices participate in the creation of the reality of psychiatric disorders is the first step in disrupting these realities for the creation of new ones. Alternative therapies for ADHD and DPD will not be the beneficiary of dedicated research and funding until the pharmacological therapies are de-prioritized.

By recognizing how Foucault's work functions not only to show the non-inevitability of these diagnostic categories, but also to allow the subject to compose or reconstitute themselves in the face of their diagnosis, my case studies open new ways for patients and clinicians to look at themselves in relation to medical science. Because drugs play a large part in our current scientific understanding of psychiatric disorders, it is tempting to think that who we are as individuals is best understood as a neurochemical state. When the philosophical question of personal identity is posed in this context, the neurobiological understanding of psychiatric disorders pushes us towards a materialist view. By recognizing the contingency of our scientific practices, those suffering from psychiatric disorders are freed to generate new interpretations of the diagnostic categories that are ascribed to them.

This is particularly important for the DPD patient, who is not likely to ever find relief from drug treatments. (Sierra, 2009) (Simeon, 2011) Because as much promise comes from psychoanalytic clinical practices as from psychopharmacological ones, sufferers of DPD must be allowed to seek whatever treatment program presents the healthiest possibilities for them. (Guralnik & Simeon, 2010) Interpretations of DPD that situate the patient in complex socio-cultural contexts and help them to recognize these situations might be allowed to flourish if drug treatments are deprioritized. The dominance of psychopharmacologically informed neurobiological perspectives hinders this possibility by dominating the landscape of treatment. Because insurance providers are more likely to cover drug treatments than psychoanalytic treatments, this state of

affairs has real consequences for the DPD patient. By foregrounding the epistemological practices that generate the current understanding of psychiatric disorders, I have aligned myself with the Foucaultian tradition of showing the non-inevitability of our current thinking on psychiatric disorders. By uncovering this situation my case studies open space for new and healthier identities to be composed and to flourish.

Ian Hacking's term "dynamic nominalism" is designed to express the process by which categories of people change historically within our naming conventions. In Hacking's view the practice of classification occurs amongst "the people classified, the experts who classify, study and help them, the institutions within which experts and their subjects interact, and through which authorities control." (Hacking, 2007) Hacking's work draws on Nietzsche and Foucault to describe the shifting process by which categories of people are developed. Hacking also calls attention to that section of *The Gay Science* in which Nietzsche underscores the powerful importance of names. While Hacking eschews "linguistic idealism," he uses Nietzsche and Foucault to make the point that there is an "evolving body of knowledge about the people in question –both expert knowledge and popular science" that alters the way that people come to understand themselves. (Hacking, 2007) Well known for his description of a "looping effect" by which the known (i.e. the subjects of a certain psychiatric classification) come to behave in the way that the knowers (psychiatrists)

know them, Hacking also notes that the different behaviors of labeled individuals also alter the meaning of each category. (Hacking, 1995)

Hacking's work has been influential on my case studies from the outset. Hacking's book on Multiple Personality Disorder *Rewriting the Soul* develops a compelling account of how a new self-rating scale (the Dissociative Experiences Scale) generates mathematical stability for emerging psychiatric diagnostic categories. The development of the Cambridge Depersonalization Scale (CDS) for DPD was an important moment in the history of the disorder, and understanding it in light of the Dissociative Experiences Scale for Multiple Personality Disorder (MPD) helps to demonstrate how a small community of clinician/researchers could generate a validated statistical tool for the measurement of psychiatric disorders. Hacking's critical stance is perhaps difficult to fully elucidate, but is nevertheless incisive in its products.[49]

Hacking's work demonstrates how those investigating psychiatric disorders can avoid the red herring of becoming caught in the question "Is it real?" While I am interested in the experience of these disorders, and sometimes refer to "the reality" of ADHD or DPD, I do not focus on whether any psychiatric disorder should be considered *real* or *not real*. Instead, my case studies focus on the stabilization and validity of diagnostic categories, and the experience of being diagnosed or categorized with a psychiatric disorder. In my exploration of DPD I go deeply into the history of the disorder and look at the ways that all the different measurement

[49] I have heard Hacking remark that those who defend the diagnosis of MPD accuse him of attacking the validity of the category, and those who deny its existence take him to be defending its reality.

techniques used to produce data about the disorder connect with and support one another. The generation of the CDS, the division of the disorder into symptom clusters, the exploration of the phenomenological stability of the disorder through history and the neurobiological measurement of the disorder all form an interlocking framework by which the etiological and clinical validities of the disorder are established. Hacking teaches his readers to look at scientific and cultural practices in order to see the emergence of psychiatric disorders in full context. Developing new ways of looking at ADHD and DPD extends this line of inquiry.

By following Hacking in this process my research has remained focused on what is most relevant in a philosophical understanding of psychiatric disorders; what our psychiatric knowledge can tell us about ourselves as individuals. Although the existentialist features of Foucault and Nietzsche's work are not foregrounded in Hacking's writings, his work helps us to see the ways that practices of naming and categorization in conjunction with scientific practices produce the individual reality we each experience.

One of the most important concepts employed in the generation of my case studies is that of entanglement. The recognition that the features of our reality cannot be understood in isolation grounds both my understanding of the entanglement of materiality and discursivity, and of the entanglement of epistemology and ethics. The relationship between ethics and epistemology has been discussed by feminist science studies scholars such as Donna Haraway, Sandra

Harding and Helen Longino, but Barad has developed a unique interpretation that makes use of the concept of entanglement. Where debates about the theory ladenness of scientific observations have taken place in post-Kuhnian philosophy of science, feminist theorists often begin by questioning of the value of objectivity as ontological separability. The recognition that objectivity requires not separability but responsibility to the epistemological practices that produce data is of central importance to this view. Haraway's call for a new kind of seeing that takes account of the situatedness of the viewer, Harding's "strong objectivity" that calls for the inclusion of the views of marginalized communities, and Longino's observations about the generative role of background beliefs and assumptions in scientific theories each delineate in different ways the need for new epistemologies that are responsive to the role observer within the experiment. (Haraway, 1991) (Harding, 1991) (Longino, 1990) Barad's description of ethics and epistemology as entangled elegantly expresses the need for scientists to be response-able for their roles in the process of data production by showing that neither ethics nor epistemology can be taken as first philosophy. Instead, each enactment in the production of scientific data must be carefully considered and critiqued as both inherently epistemological and ethical.

The first half of this chapter that discusses the new notion of causation I defend, and the possibility of considering psychiatric phenomena as indeterminate outside the context of measurement draw on Barad's insights.

In his book *After Method* John Law encourages philosophers and social scientists to consider "the world as an unformed but generative flux of forces and relations that work to produce particular realities." (Law, 2004) If we see the universe as indeterminate outside our practices of measurement and knowledge production, then more detail must be provided regarding how things come to be determined in our experience.

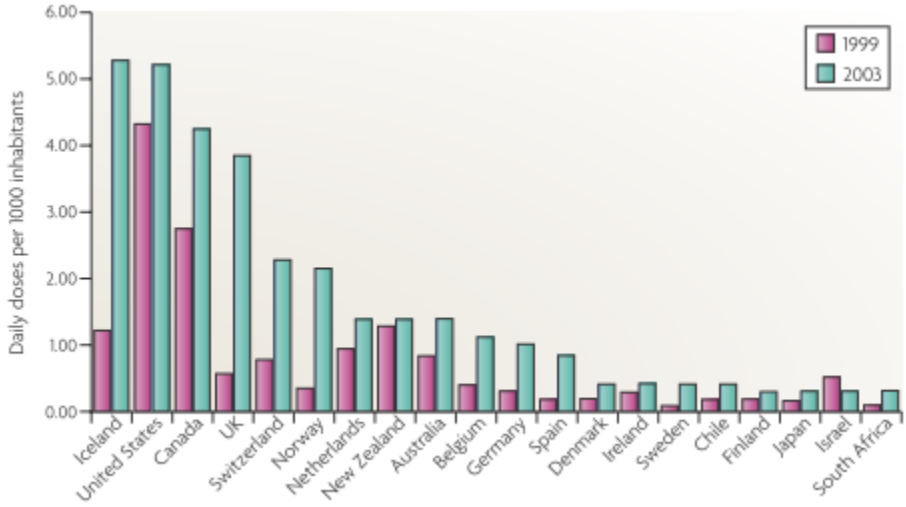
My view of the practices of knowledge production contains the idea that our habits of reasoning form a shifting stability that produces the experience of determinacy. I have sometimes referred to this view as "logical determinism."

As logical determinism pertains to psychiatric phenomena I would suggest that seemingly determinate neurobiological descriptions of psychiatric disorders would not be possible outside a logical context that contains the medical model of psychiatric disorders and the practices of psychiatric research and treatment. The phrase "logically determined" contains two claims. First, a logically determined phenomenon is "indeterminate" outside a particular context of measurement. The word "indeterminate" means that it has no fixed ontological status. Second, phenomena are made determinate through an amalgamation of reasoning practices referred to here as a "logic" or "logical formation" or "strategic logic." Here, a "logic" is comprised of the entire set of material-discursive practices that are employed for the production of knowledge concerning any phenomenon that can be investigated by science. It is perhaps here that my view aligns most deeply with Nietzsche's. I believe that the experience of an individual can be described as a sort

of self-governed fatalism, realizing fully the contradictions at play in such a view. However, it is only in rejecting such logical habits as the law of bivalence that new realities can be produced.

Appendix 1

Figure 1



Appendix 2

DSM-IV Criteria for ADHD (APA, 2000)
I. Either A or B:

A. Six or more of the following symptoms of inattention have been present for at least 6 months to a point that is disruptive and inappropriate for developmental level:

Inattention

1. Often does not give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
2. Often has trouble keeping attention on tasks or play activities.
3. Often does not seem to listen when spoken to directly.
4. Often does not follow instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions).
5. Often has trouble organizing activities.
6. Often avoids, dislikes, or doesn't want to do things that take a lot of mental effort for a long period of time (such as schoolwork or homework).
7. Often loses things needed for tasks and activities (e.g. toys, school assignments, pencils, books, or tools).
8. Is often easily distracted.
9. Is often forgetful in daily activities.

B. Six or more of the following symptoms of hyperactivity-impulsivity have been present for at least 6 months to an extent that is disruptive and inappropriate for developmental level:

Hyperactivity

1. Often fidgets with hands or feet or squirms in seat.
2. Often gets up from seat when remaining in seat is expected.
3. Often runs about or climbs when and where it is not appropriate (adolescents or adults may feel very restless).
4. Often has trouble playing or enjoying leisure activities quietly.
5. Is often "on the go" or often acts as if "driven by a motor".
6. Often talks excessively.

Impulsivity

1. Often blurts out answers before questions have been finished.
2. Often has trouble waiting one's turn.
3. Often interrupts or intrudes on others (e.g., butts into conversations or games).

II. Some symptoms that cause impairment were present before age 7 years.

III. Some impairment from the symptoms is present in two or more settings (e.g. at school/work and at home).

IV. There must be clear evidence of significant impairment in social, school, or work functioning.

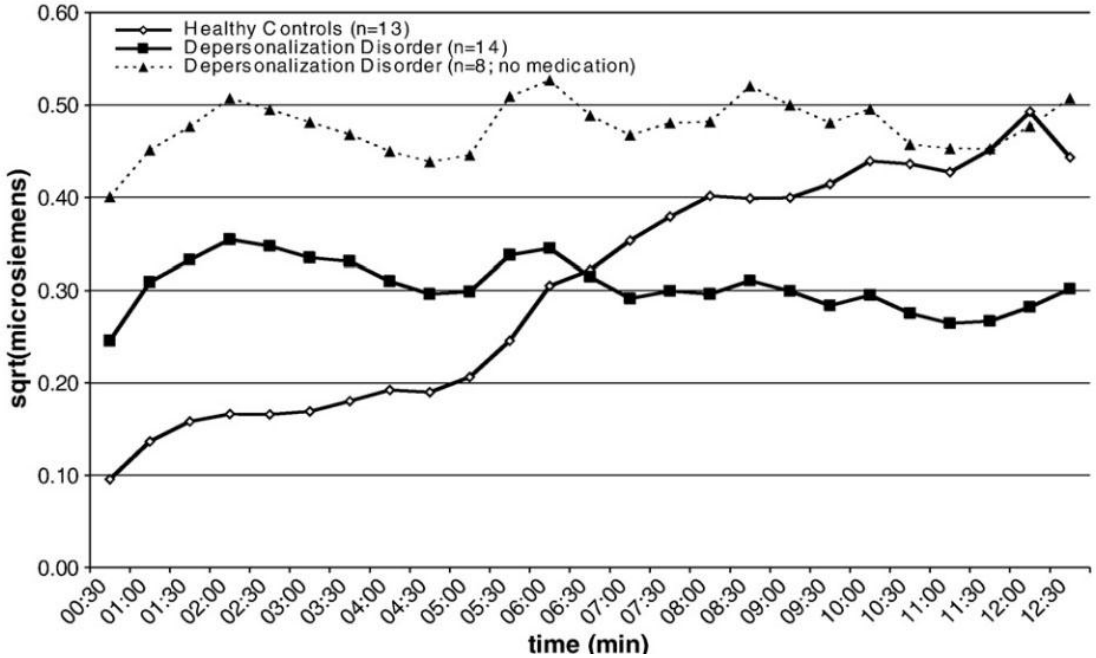
V. The symptoms do not happen only during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder. The symptoms are not better accounted for by another mental disorder (e.g. Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

Based on these criteria, three types of ADHD are identified:

1. ADHD, *Combined Type*: if both criteria 1A and 1B are met for the past 6 months
2. ADHD, *Predominantly Inattentive Type*: if criterion 1A is met but criterion 1B is not met for the past six months
3. ADHD, *Predominantly Hyperactive-Impulsive Type*: if Criterion 1B is met but Criterion 1A is not met for the past six months.

Appendix 3

Skin Conductance readings for DPD patients vs. healthy controls



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