Scientific Psychiatry:
A Novel Framework for Psychiatry Understood as a Branch of Medicine

by

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Abstract

This dissertation explores the connection between medicine and psychiatry and argues for a biomedical account of mental disorder. The work begins by employing cutting-edge evolutionary theory in order to develop an account of disease that is consistent with contemporary medical theory and practice. The proposed account understands diseases to be biological malfunctions that are deemed harmful or undesirable. This scientific account of disease is then used to devise a scientifically respectable account of mental disorder. This account sees mental disorders as malfunctions in either neural or cognitive mechanisms. The project concludes by considering the potential implications of accepting this account of mental disorder for both health and social policy.

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Chapter 1: Introduction

1.1. Introduction

In this dissertation I develop and discuss Scientific Psychiatry (hereafter, SP), an account that holds psychiatry is best understood as the branch of medicine tasked with understanding breakdowns in the human mind-brain. Such a view of psychiatry is often said to be committed to the "medical model" since it places emphasis on the idea that psychiatry ought to employ the theoretical and methodological commitments and reasoning strategies of medicine. Several medical model accounts of psychiatry exist (Guze, 1989, 1992; Andreasan, 1997; Kandel, 1999; Hohwy & Rosenberg, 2005; and Murphy, 2006). None of these accounts, however, strike me as theoretically satisfying, even though much of what they say seems correct.[1] It is for this reason that I propose SP as a better performing medical model account of psychiatry.

The general goal of developing the SP account is accomplished by fulfilling three sub-goals. The first sub-goal is devoted to developing a greater appreciation of the medical model that psychiatry is supposed to adhere to, a task that is accomplished by investigating actual medical practices in order to determine the theoretical and methodological commitments of medicine (chapter 2 & 3). The second sub-goal centers on interpreting psychiatry in terms of my proposed view of medicine, Partial-Objectivism[2], in order to develop the SP account. SP is, essentially, a description of what psychiatry is like if it adheres to the Partial-Objectivist medical model—i.e., takes on the theoretical and methodological commitments that are central to Partial-Objectivism (chapter 4). The third and final sub-goal focuses on explaining how SP is related to

[1] Not only do I think much of what they say is correct, much of the account I propose is informed by and consistent with these friendly medical model accounts.

[2] Partial-Objectivism holds that core medical practices are focused on the restoration, maintenance, and improvement of health, where health is understood as freedom from disease, and diseases are understood as partially-objective states that consist of both an objective biological component and a subjective evaluative component.

other mental health professions (chapter 5). By fulfilling these three sub-goals, I hope to meet the greater goal of providing a better performing, more developed medical model account of psychiatry. In the remainder of this chapter, I discuss in more detail the sub-goals and the motivation behind this project.

1.2. The goals of this project

Medical model accounts of psychiatry are the norm among theorists interested in psychiatry insofar as it has become commonly accepted that psychiatry, understood as a discipline, ought to follow and employ the theory and methods of medicine (Black, 2005 quoted in Murphy, 2006; Murphy, 2008).[3] According to this view, psychiatry is the branch of medicine tasked with understanding how the human mind-brain can breakdown and what can be done to fix it when this happens. Proof of psychiatry's commitment to the medical model is found in the fact that psychiatrists are trained as medical doctors, a process that insures that they are thoroughly steeped in medical theory and methodology. Further proof is found in the idea, prevalent among many theorists, that psychiatry should be theoretically grounded in science—especially the biological sciences—in the same way that other medical disciplines are (Boorse, 1977; Guze, 1989; Andreasen, 1997; Murphy, 2006). Arguing in this vein, Samuel Guze writes that "[p]sychiatry is a branch of medicine, which in turn is a form of applied biology. It follows, therefore, that biological science, broadly defined, is the foundation of medical science and hence of medical practice." (Guze, 1989, 319) Guze goes on to emphasis that psychiatry essentially involves biological theory when he notes "that what is called psychopathology is the manifestation of disordered processes in various brain systems that mediate psychological

[3] The medical model view of psychiatry does not endorse extending the domain of medicine to include psychiatry. Rather, it holds that psychiatry should be committed to the sorts of ideas, reasoning, and methodology embraced by medicine. The move is, if anything, to bring psychiatry within the domain of medicine.

functions." (1992, 317) Guze is not alone in his claim that the biological sciences are central to psychiatry. Nancy Andreassan also reminds us of this when she writes

> "[c]ontemporary psychiatry studies mental illnesses as diseases that manifest as mind and arise from brain. It is the discipline within cognitive neuroscience that integrates information from all these related disciplines in order to develop models that explain the cognitive dysfunctions of psychiatric patients based on knowledge of normal brain/mind function." (1997, 1568)

Such strong claims about psychiatry's grounding in the biological sciences and the sort of training that psychiatrists receive reveal psychiatry's tacit commitment to the medical model.

Even though there is widespread agreement that psychiatry *should* "adhere" to the medical model, there is disagreement among theorists about what such adherence entails theoretically, conceptually, and practically for psychiatry. The disagreement seems to stem from more general questions concerning the nature of medical theorizing and practice, questions that are central to how we understand the medical model. One area of dispute centers on the sort of biological theory that is most important and relevant to medicine. Some theorists view medicine as especially interested in understanding genetic level influences and causes of disease (Kandel, 1998) and, therefore, privilege genetics and molecular biology as the "grounding sciences". Others place emphasis upon the role that sociological factors play in diseases (Engel, 1977) thereby introducing a crucial role for the social sciences in medical theorizing. Still others argue that all aspects of biology may be relevant to understanding disease, hence, medicine is scientifically grounded in the biological sciences generally understood (Bernard, 1865). Another area of disagreement among theorists interested in medicine centers on the nature of disease with some holding that diseases are socially constructed states and others holding that they are objective biological states (Boorse, 1977; Engelhardt, 1974, 1976; Wakefield, 1992; Ereshefsky, 2009; Murphy, 2009).

The existence of such disagreements and disputes affect our understanding of psychiatry insofar as we cannot properly characterize and understand psychiatry as a branch of medicine if

we do not have an understanding of the theoretical and methodological commitments of medicine. Accordingly, the first step in developing a medical model account of psychiatry is to gain an appreciation of the nature of medicine, a task that is best accomplished by investigating the actual theoretical and methodological commitments of medical theorists and practitioners. This task constitutes the first goal of this work—to provide an account of medicine that is consistent with contemporary medical theorizing and practices. I take the account of medicine that I uncover and develop in chapters two and three—Partial-Objectivism—to provide the theoretical and methodological commitments of the medical model I employ to re-interpret psychiatry.

It should be pointed out that this work will have very little to say about the clinical aspect of medicine aside from a few remarks about diagnosis. I will not have much to say about clinical issues such as physician-patient interaction, the role of personal values in decision-making about care, etc. Instead, my focus will be on the research side of medicine since I am primarily interested in better understanding medicine's scientific sphere of concern—i.e., the sorts of "objects" in which medicine is theoretically interested.

The second sub-goal of this project is to develop and spell out the commitments of SP (chapter 4). In order to do this, more is required than simply holding that psychiatry is committed to employing the theoretical and methodological commitments of medicine since psychiatry appears to enjoy some unique features not found in other medical domains. Two factors that are likely to cause problems for my attempt to develop and explain SP are 1) assumptions about the nature of the mental that influence theories about mental health and disorder, and 2) the complexity and higher-level properties of the human mind-brain. The problems that arise as a result of these factors appear as inconsistencies in contemporary psychiatric theory (e.g., nosology) and practice (e.g., identification and diagnosis). In order to achieve the second sub-goal of my work, I will need to show that SP has the conceptual and theoretical tools necessary to

handle these problems. In the process of developing SP, I end up introducing an account of psychiatry that is revisionary in its understanding of mental disorders and, therefore, the proper domain of psychiatry. I take part of achieving the second sub-goal to entail showing that the revisionary aspects of SP should be accepted (i.e., that they are called for and justified) even if they conflict with our intuitions and contemporary practices. By taking this stance, I align myself with other medical model accounts (Guze, 1989, 1992; Andreasan, 1997; Kandel, 1999; Hohwy & Rosenberg, 2005; and Murphy, 2006).

It is important to understand that achieving the second sub-goal of this project does not entail offering a novel, fully fleshed out nosology, nor does it result in a theoretical reconsideration of all alleged mental disorders. Indeed, one should not expect this paper to function as a replacement for the DSM or any other catalogue of alleged mental disorders! Rather, this work is primarily meant to spell out the principles that psychiatry will need to adhere to if it aims to be a branch of Partial-Objectivist medicine. Whenever possible, I also try to draw out, and clarify, any theoretical upshot that results from psychiatry adhering to Partial-Objectivism. This upshot is usually presented in the form of further principles or commitments of SP. Along the way to achieving the second sub-goal, I make use of many examples to illustrate SP's commitments, principles, and structure. When using such examples, I try to draw from the most up-to-date empirical work available. I do not, however, aim to consider all, or even many, alleged mental disorders in terms of SP's commitments and principles. To do so would require considerably more scientific knowledge and time than currently available. Furthermore, given that the aim of the project is to lay the foundation and present a framework for SP, such extensive work seems unnecessary.

The third sub-goal centers on answering an objection to the SP account of psychiatry. The objection centers on the idea that SP forces a reconceptualization of mental health and mental disorder that is 1) inconsistent with how mental health professions in general understand mental

health, and 2) would have a negative impact on public policy. In responding to this objection, I describe how SP is related to non-medical, mental health professions and how accepting SP could positively influence public policy.

1.3. Motivating the project

A central task of any project is explaining why it is theoretically or practically valuable. Put another way, one must be able to make the case that one's project is worth spending time to consider. My work is not exempt from making such a case. Fortunately, there are at least four reasons that this project is worthwhile. First and foremost, the project is valuable because it provides a better performing medical model account of psychiatry, namely, Scientific Psychiatry. In providing this account, the work clarifies the commitments of the medical model account of psychiatry. This clarification is valuable since there is much uncertainty regarding the nature of medical model accounts even though most theorists seem to agree that some sort of medical view of psychiatry is correct. If most psychiatrists are committed to such a view of psychiatry, then it will be important to understand what it entails. Second, this project offers an account of medicine—Partial-Objectivism—that is theoretically superior to other accounts that have been employed as a means to develop medical model accounts of psychiatry. As a result, my interpretation of psychiatry in accordance with my view of medicine is theoretically superior even though it shares much in common with other medical model accounts. Third, my project moves beyond a simple characterization of psychiatry and attempts to understand how implementing such an understanding would potentially change the theoretical landscape of psychiatry and other disciplines allegedly interested in mental health and mental disorder. Fourth, the project as a whole is an exercise in applied philosophy of science and mind. As such, it illustrates the value of philosophy to other disciplines and its relevance outside of academic settings. Taken together,

these four reasons provide both motivation for, and justification of, the project of developing SP and spelling out its theoretical upshot.

1.4. Chapter Overviews

The dissertation proceeds as follows. In chapter two I discuss the nature of medicine. Getting a clear understanding of medicine is a prerequisite to understanding SP since SP holds that psychiatry is a branch of medicine. To gain a better appreciation of the nature of medicine, I consider the theoretical commitments of various medical practices. After noting that medicine may enjoy a very abstract unity, I suggest that core cases of medicine—i.e., practices that most theorists recognize as medical—seem to share several commitments. In particular, these practices seem to 1) aim to be grounded in scientific theory, and 2) understand diseases to be partially objective biological states since there is both an objective and a subjective component to diseases. The objective component is that the state must be a biologically abnormal bodily state, while the subjective component is that the state is deemed harmful or undesirable by a culture or society (Wakefield, 1992; Murphy, 2006). A large portion of this chapter is devoted to understanding and making sense of the notion of biological normalcy that is likely at play in medical theorizing about health and disease. My conclusion is that core medical practices are probably best described as being committed to partial-objectivism insofar as they are interested in the restoration, maintenance, and improvement of health, where health is understood as freedom from disease and diseases are understood as partially objective bodily states. I then go on to suggest that core medical practices have a purely objective component insofar as they are interested in better understanding the dysfunctional biological parts that give rise to clusters of signs and symptoms (i.e., biologically abnormal states) even if these dysfunctional parts have not been deemed harmful or undesirable. I label such states medical conditions (MCs) to emphasize their theoretical importance to medicine. Before concluding, I consider how medical theorists identify

and diagnose MCs and diseases. The upshot of this chapter is a description of the theoretical and methodological commitments of core medical practices. The principles and commitments that I identify in this chapter as central to medicine serve as theoretical constraints and guides when I develop the SP account in chapter four.

In chapter three I consider the sort of classes that are likely to be theoretically interesting and useful for medical theorists. I begin by proposing that certain classes are likely to be of theoretical interest to scientists (i.e., scientific kinds) because of the roles these classes play in scientific practices like explanation and inductive reasoning. This leads me to develop the P-scientific kind account, an account of scientific kinds that I argue outperforms other accounts of scientific kinds. I then note that medical theorists are likely to be interested in similar sorts of classes (i.e., P-scientific kinds) since medicine aims to be grounded in scientific theory. For clarity, I label the P-scientific kinds which are of interest to medical theorists 'medical kinds'. I then argue that medical conditions as understood per chapter 2 are the sort of classes that will be theoretically interesting and useful to medical researchers and physicians. I take this as evidence that medical conditions are the P-scientific kinds of interest to medical theorists and should, therefore, be identified with medical kinds. I then explain that the causal mechanism that unifies the members of a medical kind is the common dysfunctional biological part (i.e., the shared pathology). Before concluding, I discuss several causal factors that are relevant to typing MKs—namely, etiologies and pathologies—and consider the possibility of other classes besides medical kinds in which medical theorists might be interested.

In chapter four, I use the work from previous chapters to develop my account of psychiatry, scientific psychiatry (a.k.a. SP). The chapter begins by considering some of the causes that have led to the theoretically confused nature of contemporary psychiatry. I then consider the view of the mind this project assumes. Next, I describe the commitments of the medical model beginning with general commitments—i.e., commitments presumably shared by

all medically-based accounts of psychiatry—and then moving on to the more specific commitments of the version of the medical model I employ. These more specific commitments are derived from the preceding work on Partial-Objectivism and MCs. Section five explores psychiatry according to my version of the medical model—SP. Here, SP is described as a multi-level, inter-level discipline that is theoretically grounded in the mind-brain sciences and whose domain is the malfunctioning mind-brain. I also spend time in this section discussing the MCs and MKs of interest to SP—namely, mental medical conditions (MMCs) and mental medical kinds (MMKs)—and the sort of etiological factors that are uniquely relevant to MMCs. The sixth section considers the dual nosologies of interest to SP, while the seventh section considers how SP might respond to Szaszian "anti-psychiatry" challenges. After discussing which therapies are relevant to SP in section eight, I conclude by proposing, in general agreement with Andreasan (1997) and Murphy (2006), that SP is best thought of as clinical mind-brain science.

In chapter five I consider and respond to a potential objection to SP. The objection, which takes the form of a *reductio* argument, holds that SP is problematic since it requires that we radically reconceptualize how we think about mental health and mental disorder, and this reconceptualization would have negative reverberations that would extend into the realm of public policy. My response to this objection explains why this worry is unwarranted and misguided and ultimately fails to argue against acceptance of SP. In the process of responding, I discuss how SP is related to other mental health professions and why accepting SP will likely have a positive impact on public policy concerning mental health.

Chapter 2: The Nature of Medicine

2.1 Introduction

In this chapter I explore the nature of medicine in order to gain a better understanding of its theoretical and methodological commitments and the reasoning strategies employed by medical theorists.[4] I take the commitments and strategies that I uncover to constitute an account of medicine, Partial-Objectivism. Developing the Partial-Objectivist account of medicine is an important component of my project since understanding Scientific Psychiatry will require an understanding of the nature of medicine. In this and the next chapter, I offer a glimpse into the nature of medicine as I investigate and spell out these commitments and reasoning strategies.

The chapter proceeds as follows. I begin by noting that medicine is not a monolithic practice with a clear-cut, fixed domain or singular set of aims (section 2.3). I then argue that regardless of whether medicine enjoys a global, unified nature, core cases of medical practice—practices that most medical theorists recognize as medical—do appear to be interested in restoring, maintaining, and improving health where health is understood as *freedom from disease* (Boorse, 1977).[5] This more refined, but potentially less encompassing, notion of medicine invites multiple readings since there are several ways to understand disease: as socially constructed states, as objective biological states, or as partially-objective biological states. I argue that a large number of core cases of medicine are committed to a partially objective construal of disease; namely, they understand diseases as objective states of the body that are deemed harmful or undesirable by a society or culture (sections 2.4.). My argument that medicine is committed to partial-objectivism about disease involves showing that there is both an objective and a subjective

[4] "Medical theorists" is a technical term I use to refer to theorists that study or research biological malfunctions. It does not just refer to those theorists interested in biological malfunctions that have attended medical school.

[5] Boorse notes that it is "a traditional axiom of medicine that health is the absence of disease."(1977, 542)

component to the way that core medical practices think about disease. I spend much of the fourth section of this chapter discussing the objective and subjective components. The upshot of this work is the idea that medicine is properly characterized as a partial-objectivist endeavor insofar as core medical practices are focused on the restoration, maintenance, and improvement of health, where health is understood as freedom from disease, and diseases are understood as partially-objective states. Before moving on, I briefly note that there is a purely objective aspect to medicine which is focused on understanding dysfunctional biological parts (i.e., biologically abnormal states). I term these dysfunctional parts and the cluster of signs that they give rise to 'medical conditions' to emphasize that they are likely to be of interest to medical theorists even though they may not constitute diseases. Section 2.5 briefly considers three further aspects of medicine: 1) the nature of preventative medicine, 2) the identification of medical conditions, and 3) the diagnosis of diseases. The chapter concludes with a review of the partial objectivist view of medicine.

2.2. Characterizing medicine via *a posteriori* conceptual analysis

So what is the best way to characterize medicine? A common response is that medicine is the "science and art of healing" (AMA). Leaving aside concerns about the status of medicine as a science or art, this claim does seem to capture something intuitively central to medicine, namely, its emphasis on health. But is this emphasis on health a necessary and sufficient condition of medicine? That is to say, do only those endeavors that are interested in the health of individuals count as medical practices? What about palliative care, preventative care, and cosmetic surgery—practices that, at first blush at least, do not seem to deal with healing but comforting the dying, avoiding diseases and maintaining health, and altering the body for aesthetic reasons?[6] Should these practices count as medical? Conversely, should all practices

[6] Such alterations could be for non-aesthetic reasons if they are motivated by self-esteem issues.

interested in health be considered medical practices, even those that have no grounding in scientific theory? What about prayer, chiropractic care, acupuncture, or "quantum healing"[7], practices allegedly aimed at healing but which currently lack good scientific grounding? Answering these questions without a clear understanding of the notion of health is difficult. After all, if medicine is primarily interested in health, then it will certainly matter what the term 'health' means.

In an ideal world—or at the least, a world where all terms were rigidly definable and accompanying concepts were *a priori* analyzable—defining health would be a practice easily accomplished from the armchair. Our world is not ideal. It is very likely, therefore, that understanding the nature of medicine will require more than *a priori* conceptual analysis. Indeed, if there is a lesson to be learned from the past 75 years of philosophy, it is that we are only rarely able to provide *a priori*, clearly defined, non-porous definitions for concepts that refer to practices like medicine that have such long and varied histories. Attempts to better understand the discipline of medicine via *a priori* conceptual analysis are unlikely to get us very far since there is no obvious reason to think that the terms 'medicine' and 'health' are analytic or that their accompanying concepts are ones that can be *a priori* analayzed.[8] Given that *a priori* analysis is unlikely to prove fruitful, we ought to be wary of allowing intuitions about medicine and medical practice to play too much of a role in our attempt to understand medicine. After all, intuitions are often unstable, tend to vary among people, and may only play a "parameter setting" role by determining what things should count as core cases of the "kind" under investigation—i.e., cardiology and surgery better count as medicine, while it may be questionable whether cosmetic surgery and palliative care do (Murphy, 2006, 62).

[7] http://www.quantumhealingcenter.com/ (retrieved 7-28-2014)

[8] Though these two may come apart as Kripke (1972) has persuasively, in my mind at least, argued.

Granting my concerns about the *a priori* analysis of medicine via intuitions, it seems that developing an understanding of medicine will require the employment of a different methodology. In particular, it seems to require that we look to the world in order to gain an understanding of the nature of medicine. This is, in effect, a proposal that we engage in *a posteriori* conceptual analysis. This *a posteriori* strategy centers on looking at the sorts of things commonly considered medicine in order to determine what features, if any, these practices share.[9] A virtue of this approach is that it is the most theoretically neutral way to gain an understanding of medicine since the actual practice of medicine acts as a constraint on our claims.

Because this chapter is primarily interested in understanding the nature of core medical practices and not in mapping the conceptual domain of medicine in general, I will make some quick remarks to illustrate why I think that medicine is, at best, abstractly unified, and for all intents and purposes, best thought of as a hodge-podge of related, yet distinct practices with varying aims. These remarks on the non-monolithic nature of medicine are meant to assuage any fears that might arise about the value of the account of medicine that I propose (i.e., Partial-objectivism) since it may not properly characterize *all* alleged medical practices. Indeed, my point will be that Partial-objectivism—the view that medical practices aim to be grounded in scientific theory and understand diseases as partially-objective states—does seem to capture what is important about core medical practices even if it fails to properly characterize all alleged medical practices.

2.3. The abstract unity of medicine

So why deny that medicine is a monolithic discipline? The answer is straightforward: the evidence does not suggest that medicine is a discipline with a single purpose or end since the actual practices of medicine are quite varied with respect to their aims and motivations. Practices

[9] See Murphy 2006, Chapter 3 for discussion of a similar strategy.

that are commonly considered within the domain of medicine include both research and implementation practices. On the research side, there are attempts to develop and extend our theoretical understanding of disease states, as well as work aimed at the development and implementation of treatment strategies and interventions that can restore, maintain, and improve health. The implementation of the body of knowledge gained by such research constitutes the mass of much medical practice. It is this aspect of medicine that is embodied in the various physician-patient interactions. Of course, there are many alleged medical practices that have nothing to do with disease states and perhaps are not focused on health. In particular, palliative care and cosmetic surgical procedures are both considered medical practices but it is questionable whether they are primarily focused on disease states or that their aim is the restoration, maintenance, or improvement of health.[10] One could argue that such practices are essentially health related given a certain notion of health. For instance, if we think of health as somehow involving human well-being, then it may be possible to think of cosmetic surgery and palliative care as leading to increased health insofar as they increase well-being in some sense. I take it that the notion of health as well-being provides a sort of abstract unity to medicine since medicine in general seems to involve practices aimed at restoring, maintaining, and increasing human well-being.

The claim that all medical practices are interested in health understood as well-being, the abstract unity account, is weak on content until more is said about what we mean by 'well-being'. And, there are problems that follow any attempt to provide further content to this claim. Some attempts to give content will cause the range of the account to narrow. For instance, if we hold that the relevant sense of human well-being is biological well-being, then we will have trouble explaining how cosmetic surgery or palliative care are medical practices since they do not, at least not obviously, lead to biological well-being. Attempts to give content to the claim that cause the

[10] The American Society of Plastic Surgeons is a sub-society of the AMA.

account's range to narrow rob it of its intended goal—offering a unified account of medical practice. Other attempts to fill out the claim make it too broad by entailing that a number of practices that are commonly not considered medical would count as such. If, for instance, we follow the World Health Organization's lead and take health to be a state "of complete physical, mental and social well-being", then we will be forced to treat a number of practices aimed at reducing poverty, reducing gun violence, improving economies, etc. as medical given that these practices are crucial to maintaining, improving, and restoring social well-being (WHO, Preamble, 2006; Sen, 1985, 1993). While it seems quite clear that these practices belong in the domain of public health, it is far from clear that they do, or should, count as medical practices.[11] One possible move for the person sympathetic to the WHO's understanding of health would be to argue for an additional constraint on the abstract unity account that explains why the problematic cases that result from the WHO's notion of health—gun control, poverty reduction, etc.—do not actually count as medical. The problem with this move, however, is that it is unclear what this delineating constraint might be and if we could figure out what it is, then it would effectively replace the health as well-being account since it would, hypothetically at least, properly delineate the medical from the non-medical. A final strategy would be to simply abandon the task of filling out the content of the claim and accept it as it is, devoid of determinate content. This move is also unsatisfactory as it leaves us with an account that is uninformative since it is unclear what is meant by 'well-being'.

In the end, it is unclear how one should proceed with an abstract unity account of medicine. It seems that such an account faces deep problems that will need to be dealt with if one is committed to medicine as a monolithic practice and wants to advance an account of what unifies medicine. I offered the abstract unity account, which holds that medical practices are

[11] The WHO's notion of health as "physical, mental, and social well-being" seems to capture the notion central to Public Health. This suggests that the notion of health relevant to public health and medicine are likely to diverge even though there is certain to be some overlap—i.e., both public health and medicine appear to be interested in health understood as physical well-being.

those interested in restoring, maintaining, and improving health understood as 'well-being', only to suggest that there may be some very abstract way that all medicine is unified. I am not, however, deeply committed to this claim. Indeed, I am willing to leave the problems that besiege the abstract unity account as live problems since I am open to the idea that medicine is not a monolithic practice. In fact, I think it is important that we not allow any potential abstract unity to prevent us from developing accounts of the various ways that medicine may be characterized. Furthermore, once we give up the idea that what we say must range over all alleged medical practices, an interesting conception of medicine—one that focuses on the distinction between health and disease—emerges that seems to characterize a large number of core medical practices.

2.4. Medicine and health as freedom from disease

The more promising way to understand the claim that medicine is a discipline that is primarily interested in health is to think of health not as well-being but as freedom from disease, disorder, and injury (hereafter, I use 'disease' as a general term meant to encompass disorder and injury as well).[12] This idea seems to capture what is central to core medical practices. Indeed, the idea that medicine is interested in the restoration, maintenance, and improvement of health seems easily understandable if health is understood as the state of being free of disease: restoring health involves a return from a diseased state to a disease free state; maintenance of health involves maintaining a state of freedom from disease; the improvement of health involves increasing resistance to disease.

An important component of this account is that diseases should be understood as deviations from a normal state, while normal states should be thought of as healthy states.[13] The

[12] I have more to say about these various states when I discuss medical conditions and medical kinds in chapter 3.

[13] One may worry that the notion of health as freedom from disease obscures a useful distinction between normal states and healthy states. Following Boorse's (1977) work, the idea here is that freedom from disease only gets a person to normalcy while something more is required for being healthy. Hence, the potential states of a person are three:

role of normal states in this account raises several questions. First, how do we determine which states are normal? There are two relevant approaches to understanding the standards of normalcy, or norms, that play a role in determining what counts as a normal state: one is constructivist and holds that the relevant norms are determined by human (i.e., cultural or societal) values, the other is objectivist and holds that the relevant norms are natural—i.e., they are objective matters of fact that are discovered in nature and consistent with our best current science. The second question concerns whether all states that deviate from the relevant normal state are diseases or is something in addition to this deviation necessary for a state to count as a disease? These two questions leave us with three possibilities: 1) diseases are socially constructed states since they are deviations from a normal state that is socially or culturally determined, 2) diseases are pure objective states because they are deviations from a biologically normal state that is objective, or 3) diseases are partially objective states since they are states that have an objective component (i.e., they are biologically abnormal states) and a subjective component (i.e., they are states that are deemed harmful or undesirable by a society or culture). In the coming sections (sections 2.4.1- 2.4.5), I determine which notion of disease is employed in core medical practices.

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diseased, normal, or healthy. Such a tripartite distinction often rests upon a conception of positive health, a notion that ties degrees of health to optimal biological functioning of either the species or the individual (Boorse, 1977). In terms of cardiovascular health, the idea would be that the cardiovascular system can be diseased, free from disease (normal), or healthy (i.e., functioning optimally) with the understanding that a cardiovascular system that is normal insofar as it is not diseased is not the same as one that functions optimally—one is simply free from disease, the other performs its function exceptionally well. I do not dispute the usefulness of such a distinction. But, I also do not feel it necessary to employ it since accepting the distinction would not really affect the proposed analysis of the aims of many medical practices for we could merely reformulate my proposal to say that much of medicine is interested in the restoration, maintenance, and improvement of diseased states to normal *and/or* healthy states. The idea behind the fix is that diseased states involve a deviation from normalcy and medicine is interested in getting people *at least* to the norm.

In any case, it seems unnecessary to take a stance on the theoretical usefulness of this tripartite distinction. What is important for my proposed understanding of medicine is the idea that disease states can be, and are, distinguished from other states that are recognized as being normal. Whether we choose to think of these normal states as healthy states or some intermediate between diseased states and healthy states is an issue I wish to leave open. For ease of exposition, however, I will present the health as freedom from disease account as being committed to a simple binary distinction between health and disease, and, therefore, will treat normal states as though they are the healthy states. I don't expect anything of theoretical value to hinge on this conflation—it is only meant to illustrate that what is of central importance is that a distinction can be drawn between disease states and non-disease (i.e., normal or healthy) states.

I begin by considering the constructivist account of disease and suggest that this is not the notion of disease employed in most medical practices. Next, I turn my attention to the objectivist and partial-objectivist accounts of disease. I suggest that all objectivist accounts, partial-objectivism included, will hold that diseases are abnormal biological states. Given the centrality of biological normalcy to objectivist accounts of disease, I analyze several notions of biological function that could ground the notion of biological normalcy at play. I argue that one of these notions of biological function, and the account of biological normalcy that it gives rise to, likely plays a role in medical theorizing about disease since it is employed in the biological theory relevant to medicine. This suggests, in turn, that core medical practices are committed to an objectivist notion of disease insofar as they recognize that diseases are essentially abnormal biological states. I then explore whether the relevant objectivist notion should be construed as a purely-objectivist or a partially-objectivist account. My suggestion is that medicine is most likely committed to the partially-objectivist account of disease.

2.4.1. Health as freedom from disease: the constructivist model of disease

If diseases are understood as being deviations from socially constructed norms—if cultural or societal values determine which states are normal—then the accompanying conception of disease will be laden with social values since it is these values which determine the relevant socially constructed norms (Murphy, 2006, 2009; Ereshefsky, 2009) . There are several ways that the socially constructed view of disease is argued for. The first notes that disease and health are normative concepts and that the natural world is value neutral. Hence, disease and health cannot be objectively grounded so they must be subjectively grounded (Margolis, 1976). This position is beautifully simple. The problem, however, is that there are many attempts, though not all are successful or even very good, to provide a naturalized account of norms (e.g., Boorse, 1977; Millikan, 1984, 1989; Neander,1991, 1995; Allen & Bekoff, 1995; Griffiths, 2009). If such a project were to succeed, it would rob this subjectivist position of its necessary premise that

normative concepts cannot be objectively grounded (e.g., grounded in biological theory). In the next section of this paper, I argue that a particular account of natural norms does succeed thereby robbing this constructivist argument of its necessary premise.

The second argument in favor of constructivism holds that social values drive inquiry into diseases and, as a result, end up influencing how we understand disease. The general idea behind this position is captured well in Tristram Englehardt's work on the history of the "disease of masturbation" (1974, 1976). Engelhardt reminds us that during the 19th century it was widely held by "medical" theorists that masturbation was the causal mechanism responsible for a certain set of symptoms including, but not limited to: increased hand-size, insanity, infertility, stooped posture, and lowered intelligence. Englehardt argues that it was the social values of this period—especially in America but also England—that lead to the postulation of masturbation as the relevant causal mechanism of these symptoms. As an account of the history of the *idea* that masturbation is a disease, Englehardt's analysis seems correct. After all, it probably is the case that social values and human interest guide research and influence our understanding of medical conditions (Murphy, 2009). What Engelhardt fails to realize, however, is that this by itself doesn't argue for constructivism for even the objectivist can hold that human/societal values and interests play a role in guiding research. After all, we generally only research things we are interested in and there is no reason to think that our motivation for research would strip it of its objective standing. The relevant question has little, if anything, to do with the motivations for research and everything to do with whether the distinction between disease and health can be, and is, objectively grounded. What Engelhardt shows does not suggest that it cannot, nor that it is not. All it does is suggest that theorists can be wrong as they were in the 19th century when they claimed that masturbation was the causal mechanism responsible for the alleged syndrome (Murphy, 2009).

The above constructivist claim suggests a third strategy to argue for constructivism, one that is essentially a variant of the first argument. This third way holds that any notion of disease whatsoever is bound to be socially constructed since human interests are necessary to "negatively value" a state and to draw a distinction between disease states and healthy states one must judge one state negative with respect to the other (Murphy, 2006, 2009). The constructivist notes that there is nothing in nature that makes one of the states negative and the other positive—hence, the judgment that one is a disease state (negatively valued) and the other is a healthy state (not negatively valued) can only be explained by recognizing that human interests determine these judgments.[14] Even the idea that a state should be negatively valued—i.e., judged a disease state—because it hinders survival is dependent upon human interests in survival. If Mother Nature has an interest in our survival, argues the constructivist, it is an interest that has not yet been revealed. The problem with this argument is that it seems to miss the point and thereby fails to address the central question of whether we can provide an objective account of the distinction between those states that we value and those that we do not (i.e., between health and disease states) (Murphy, 2009). If we can give an account of the distinction between these states that does not depend on human interests, then we will have provided the required objectivity—we will have a means to objectively ground our account of disease. This raises the question, can we account for the relevant distinction in non-constructivist terms? The objectivist answers affirmatively and holds that the distinction between negatively valued and positively valued states tracks a distinction between biologically normal and biologically abnormal states. The most promising way to explain this distinction is via a naturalized notion of biological normalcy. I turn to objectivist accounts shortly. But first, a few more remarks on the constructivist project.

[14] It is worth pointing out that there is something correct about the criticism just considered—namely, there is nothing in nature that tags a state as negative as opposed to positive. This is, in fact, exactly the sort of reason that has led many theorists, myself included, to posit a two-factor account of disease.

Three points about constructivist accounts are worth expanding upon in order to clarify how they differ from objectivism. First, the relevant constructivist account is the one that denies that the distinction between disease and health can be objectively grounded through some form of natural norms (Murphy, 2009). Part of the reason that constructivism denies the possibility of objective grounding is because it places conceptual priority on the evaluative judgment of the state. The notion of conceptual priority, as described by Murphy, has to do with the idea that one aspect of a concept may be more central and identity conferring than some other aspect (2009). Accordingly, the constructivist holds that the evaluative judgment about a state is conceptually prior (more important to determining the status of the state as a disease) to whether the state is actually caused by a biological malfunction. Even though there may be a search for an underlying causal mechanism, says the constructivist, the failure to locate such a mechanism does not negate the conceptually prior judgment that there is a disease for what makes a state a disease for constructivism is that it is judged to be undesirable or abnormal—not that there is an actual biological breakdown or malfunction of some sort (Murphy, 2009). Objectivist accounts, on the other hand, take the existence of a deviation from a biological norm of some sort to enjoy conceptual priority over evaluative judgments derived from human interests when it comes to determining the status of a state as healthy or diseased. Accordingly, for the objectivist, it is a necessary, though perhaps not a sufficient, component of diseases that they be states that deviate from an objective biological norm.

Second, the claim that societal or human values and interests play a role in guiding research and theorizing does not entail constructivism about disease; rather, it only shows that such interests and values can influence theorizing and should be closely watched for. Indeed, it is likely that evaluative judgments often prompt the search for underlying causal mechanisms responsible for the allegedly undesirable or abnormal behavior. As the objectivist is certain to point out, contra constructivism, the failure to locate a causal mechanism responsible for the

relevant behavior is often taken as grounds to reject the claim that the state should count as a disease. This suggests, as Murphy (2009) argues, that evaluative judgments may enjoy "temporal priority" in the process of understanding disease states even though they do not enjoy conceptual priority. The temporal priority of these judgments in the discovery of disease states illustrates the fact that they may play a robust role as a heuristic device (Murphy, 2009). But, that medicine tends to be interested in diseases understood in some sort of objective manner suggests a theoretical emphasis placed upon understanding underlying causal mechanisms, specifically, malfunctioning mechanisms. *Prima facie* evidence for this claim is found in the overwhelming tendency of medical theorists to think of diseases in terms of Robert Koch's (1890) and Louis Pasteur's (1860) germ theory, Claude Bernard's theory of "*internal mileu*" disruption (1865, 1957), and a generalized deficiency theory (see Carter, 1977), theoretical perspectives that focus on understanding the underlying causal mechanisms of diseases. Furthermore, constructivist accounts appear incapable of accounting for the objectivity that seems central to medicine understood as a discipline based in biological theory. The connection between biological theory and objective accounts of disease will be explored in depth in the next section of this chapter.

The third point worth mentioning is that it could turn out that a constructivist notion of health and disease—one where human or social interests and values alone determine what counts as a disease—may have a role to play in some aspects of medicine. It isn't clear what that role might be, but there is no obvious *a priori* reason to think that no branch of medicine is interested in diseases understood in this constructivist manner. The upshot of this consideration is that we cannot rule out health understood as freedom from constructively determined disease as a potentially relevant notion of health on *a priori* or theoretical grounds alone. Nonetheless, we can recognize that even though conceptually and theoretically medicine *could* be interested in such a constructivist notion of disease, much of medicine and many medical practices that do seem interested in health understood as freedom from disease appear primarily, if not exclusively,

interested in freedom from disease where what counts as a disease is, at least partially, an objective matter of fact.

2.4.2. Health as freedom from disease: objectivist models of disease

In this next section, I consider two objectivist accounts of disease—pure-objectivism and partial-objectivism—and argue that medicine is, and should be, committed to the partial-objectivist view.[15] I begin by noting that both of these positions gain their status as objective because they take diseases to be, at least partially, abnormal biological states. Since biological abnormalcy is central to both of these accounts, I spend some time getting clear on the notion of biological normalcy that is likely implicit in medical theorizing about disease and health (section 2.4.1- 2.4.4). I then argue that actual medical practice and medical theorizing suggest that medicine is implicitly, if not explicitly, committed to a partial-objectivist view of disease.

Objectivist accounts of disease are objective in the sense that they understand diseases to be, at least partially, an objective "matter [about human biological states] to be determined by science" (Murphy, 2009). That objectivist accounts recognize that science has a guiding role to play in understanding disease suggests that these accounts ought to be developed in accordance with the principles of methodological naturalism. The upshot of taking on these principles is that theorists will be limited to employing only those causes, events, and properties posited by the relevant sciences when attempting to explain and understand the objective aspect of disease (Ruse, 2001). Given medicine's interest in understanding the human body and how it can "breakdown", it is reasonable to assume that the relevant science to consider when dealing with theoretical issues in medicine is biology.[16] Taking biology as the relevant science, it is likely that an objectivist account of disease, or the objective component of a partial-objectivist account, will

[15] The claim is that medicine is implicitly committed to this view because it is implicit in medical theorizing and reasoning.

[16] Or more precisely, certain sub-disciplines of biology.

be cashed out in terms of biological normalcy. Getting clear on exactly how we should understand the notion of "biological normalcy" at play is a matter dealt with in detail throughout the remainder of this section.[17] Nonetheless, the motivating thought behind objectivist accounts of disease should be clear: diseases are, at least partially, abnormal biological states that are objective matters of fact.

In order for biological normalcy to deliver its theoretical goods, there needs to be a way to draw a principled distinction between normal and abnormal biological states. One way theorists attempt to meet this challenge is to think about biological normalcy in terms of normal, or proper, biological functions. The idea here is that we can provide a principled distinction between normal and abnormal biological states by taking normal biological functioning to be the mark of biological normalcy. Malfunctioning biological parts, then, would count as abnormal biological states (see Figure 2.1). For this strategy of developing an objective account of biological normalcy to succeed, however, we will need an account of normal biological function that is objective—i.e., an account that sees the distinction between a normally functioning biological part and an abnormally functioning biological part as being a matter of fact that is discoverable by science.[18]

[17] See Wachbroit (1994) for more on the importance of "normalcy" in biology.

[18] I use 'normal functions' and 'proper functions' interchangeably throughout. I also use 'proper function' and biological function' interchangeably since I take the proper or normal function of a part to be its biological function.

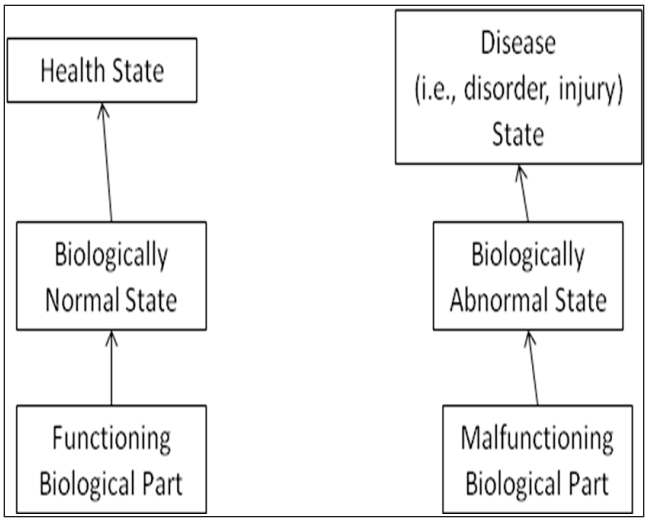

Figure 2.1. Figure illustrating the connection between functions and health & disease.

Naturalized accounts of biological function are the obvious place to look for such an account since they are committed to the relevant notion of objectivity in virtue of being committed to methodological naturalism. Accordingly, naturalistic accounts of biological function (should)

1. offer an account of normal biological function that relies on our best current scientific theory, and
2. understand the distinction between normal and abnormal biological function as a matter of fact.

Accounts of biological function that make good on (1) and (2) will count as objective in the sense relevant to this project since they will rely on natural norms—i.e., standards of normalcy that are grounded in matters of fact and "are determined by science"— to differentiate normal biological functioning from abnormal biological functioning.[19] A successful naturalistic account of biological function would provide the natural norms needed for distinguishing functions from dysfunctions (or malfunctions), and this distinction could then be used to differentiate normal

[19] Subjective interests may play a non-constitutive role in naturalistic accounts of function to the extent that such interests will determine what sorts of capacities we are interested in better understanding, but they ought not to determine what the actual function of the part is (see Boorse, 1977; Murphy, 2006; Davies, 2001).

from abnormal biological states. And, once we have a notion of biological normalcy that is objective—i.e., grounded in the natural norms of biological functioning—then we can use this to objectively ground, or partially-objectively ground, the distinction between disease and health.

Since both objectivist accounts imply that medical theorizing relies on a notion of biological normalcy that is grounded in a notion of biological function, I will begin by getting clear on the notion of biological function and biological normalcy such theorizing employs. To this end, the next section explores several naturalistic accounts of biological function. Discussion is limited to those accounts that could be employed in the biological sciences since these are the sorts of science that are likely to be relevant to, and employed in, medical theorizing.[20] There are two accounts of biological function that I consider: 1) the selected effects account and 2) the evolutionary systemic-capacity account. After explaining each account, I then assess whether they meet three constraints that a theory of biological function ought to meet if it is the sort that is involved in medical theorizing about disease:

1. **a practicality constraint**: the account needs to be practical—it should be capable of actually being employed by medical theorists.
2. **a differentiation constraint**: the account should provide natural norms for biological functions that can ground the distinction between biologically normal and abnormal states. The second aspect of this constraint is that the distinction based on these norms ought to respect core cases of disease and health—e.g., the norms ought not to allow core cases of disease like a broken leg or lung cancer to count as biologically normal states.
3. **a foundation constraint**: the account should be grounded in the appropriate biological theory. Following Winther (2006), Wouters (2005, 130; 2007), Griffiths (2009), and Ereshefsky (2009), I take the appropriate biological theory for medicine to be compositional, or functional, biology which studies the organized activity of the parts of biological mechanisms at various levels (e.g., the genetic, molecular, cellular, and systems level).[21]

[20] Though we may also need to think about humans as social creatures, I take it to be a central tenet of medicine that we think of humans as biological systems.

[21] So what sort of biological theory is medicine grounded in? Following Winther (2006), I take "canonical advanced textbooks" (i.e., those read by medical students), as well as actual research strategies, to be indicators of a discipline's basic theoretical commitments (479). Accordingly, if we wish to know what sort of theory medicine is grounded in, we ought to look to the textbooks and practices relevant to medicine. As numerous theorists have noted, when we do this, we will recognize that the biological theory of interest is functional biology (Winthers, 2006; Wouters, 2007; Griffiths,

I conclude by noting that the evolutionary-systemic-capacity account of biological functions is the one most likely involved in medical theorizing about disease given that it meets the three proposed constraints. Having secured an objective account of the distinction between normal and abnormal biological states, I then go on to consider whether medicine is committed to the pure objectivist view or the partial-objectivist view of disease.

2.4.2.1. The Biological function component: The modern history selected-effects accounts

One of the most commonly discussed naturalistic accounts of biological functioning is the selected-effects account (Millikan, 1984, 1989, 1989b; Neander, 1991, 1995; Wakefield, 1992; Godfrey-Smith, 1994; Allen & Bekoff, 1995). The selected-effects account understands an item's function to be best understood in terms of the purposes or goals for which the thing was designed. While a teleological approach like this seems appropriate for understanding the function of artifacts, many have questioned whether it should be employed as a means of understanding biological functions since it seems to require that we posit a supernatural agent responsible for designing parts with the purpose of performing specific functions (Davies, 2001; Allen & Bekoff, 1995). The worry is that because such agents are not admitted by scientific theory, any teleological approach to understanding biological function is destined to be anti-naturalistic and, therefore, scientifically questionable.

Ruth Millikan's (1984, 1989, 1989b) and Karen Neander's (1991, 1995) work on the selected-effects account argues that a teleological account of biological function that is scientifically respectable, and naturalistically consistent, is possible. Their general claim is that the proper—i.e., normal—function of a biological part[22] is whatever effect the part was naturally

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2009). Functional biology (a.k.a. compositional biology) seeks to understand how the parts of a biological system contribute to the system's overall capacity of surviving and reproducing.

[22] I use 'part' in the generic sense of "one piece of a greater whole". Mechanisms, systems, pieces, components, etc. can all be parts.

selected for. Accordingly, the function of the heart is to pump blood and not to make thumping sounds because the effect of hearts that was naturally selected for was the pumping of blood, not the thumping sound. Millikan's and Neander's accounts vary in fine detail but agree in most respects. In particular, they both hold that proper biological function is an objective matter of fact that depends on the evolutionary history of biological parts. The upshot of this move is that the evolutionary history of a part sets the natural norms for the part: a part *ought* to function in the way that led to it being naturally selected for. Having natural norms for a part allows us to objectively differentiate between properly and improperly functioning (i.e., dysfunctional) parts: a part is functioning normally or properly when it is playing the role—i.e., "exhibiting the effect"—it was naturally selected for, a part is malfunctioning whenever it fails to "exhibit the effect" for which it was naturally selected. A heart is properly functioning if it exhibits the effect (i.e., pumping blood) that it was naturally selected for; a heart is malfunctioning if it fails to exhibit this selected for effect. For the selected-effects accounts, the only relevant information when attempting to understand the normal function of a part is the effect in virtue of which the part was naturally selected. Accordingly, the account considers it nonsensical to speak of biological functions that were not selected for since it understands being selected for as a necessary condition of biological functions. Furthermore, because the selected effects approach limits the biological function of a part to the effect that the part was naturally selected for, if there is no effect in virtue of which a part was naturally selected for—e.g., if the part is a spandrel—then the part will not be attributed a biological function.

Given the tension discussed earlier between naturalism and teleological accounts, it may be worthwhile to note that the selected-effects account alleges to maintain its status as both teleological and naturalistic (for arguments contra this claim, see Davies, 2001). The account maintains its status as teleological since it understands the function of a biological part to be defined in terms of the purposes that the part was designed to fulfill. It manages to escape the

anti-naturalistic charge commonly leveled at teleological accounts by holding the mechanism of "design" to be natural selection. Accordingly, we can understand the function of a heart to be pumping blood because the heart was designed, through natural selection, to pump blood. Understood in conjunction, these two claims do seem to offer a naturalistic teleological account of biological function.

Since its inception, the selected-effects account has been reformulated to handle a number of shortcomings that issue from the underdetermined nature of the requirement that a part's proper function just is the function in virtue of which it was naturally selected for. In her earliest writings, Millikan held that the proper function of the part was the function that led to the part *originally* being selected for (1984). But there are problems with this strict understanding of natural selection as original function selection. To appreciate the problem, consider the following case. It is possible that the coloring of the plumage of a certain bird species was originally selected for in virtue of its ability to ward off a certain type of predator that is now extinct, but the coloring has since then been selected for —i.e., maintained in the bird population—because of the role it plays in attracting potential sexual mates. If we accept the proposal that the function of a part is whatever that part was originally selected for, then we will be forced to say that the biological function of the part—here, the plumage—is "defensive" as opposed to "mate attraction" even though mate attraction is the only function that the part plays in contemporary members of the species. Some have suggested that it is out of line with much biological reasoning to treat a function which a part is no longer selected in virtue of as the proper function of the biological part simply because it explains the part's origin (Godfrey-Smith, 1994; Griffiths, 2009). What seems more important for much of biology, at least according to Godfrey-Smith, is the function in virtue of which the part has most recently been selected for—i.e., the function in virtue of which the part has most recently been maintained in the relevant population (1994). This sort of reasoning led Millikan (1989) to amend the selected-effects account in such a way

that proper biological functioning could be understood as the function a part was originally selected for *or* the function the part was most recently selected for (maintained in virtue of). Indeed, in later work, Millikan holds that the relevant evolutionary history for determining the proper function of a biological part may very well be the part's recent history, perhaps a history as recent as yesterday, since a part may be selected for in virtue of very recent evolutionary pressures (1989; Godfrey-smith, 1994). This move suggests that two sorts of evolutionary history are relevant for Millikan's account when attempting to determine a part's proper function—modern selective history which focuses on the recent selective history of the part and ancient selective history which focuses on the origin of the part (Godfrey-Smith, 1994). By recognizing the theoretical value of these two sorts of history, it looks as though Millikan can skirt the problems that besiege her earliest formulations of the account by recognizing that parts may be selected for different functions throughout an organism's evolutionary history since different evolutionary pressures may exert themselves at different times.

But, here we have a classic case of robbing Peter to pay Paul for another problem seems to arise from this amendment. The problem is connected to Godfrey-Smith's charge that Millikan's amended selected-effects account falls short because it allows that the proper function of a part could be the function which was important in a part's ancient selective history—i.e., the function in virtue of which the part was originally selected for—*or* the function which was important in a part's modern selective history—i.e., the function in virtue of which the part was most recently selected for. To appreciate the problem, let us consider the possibility, as some have proposed, that that the feathers of birds were originally selected for in virtue of their ability to aid in thermoregulation but have most recently been selected for in virtue of their ability to aid in thermoregulation *and* make flight possible (Cowen & Lipps, 2000 ; Dimond et al., 2011). If we accept that proper function could be the function for which a part is maintained or the function for which a part was originally selected for, we seem required to posit that feathers have both

functions: thermoregulation and flight. This, of course, raises the issue of how we are to determine which of these functions is *the* proper function of the part. Of course, a proponent of Millikan's account could argue that both functions are proper functions since the part has been recently selected for in virtue of *both* functions. Notice, however, that what is doing the work in this sort of response is that both of the functions have been relevant to the part's *most recent* selective history. As Godfrey-Smith (1994) notes, this raises the question of whether ancient selective history is ever relevant to determining a part's biological function. The answer seems to be yes if the function that the part was originally selected for and a function that the part has recently been selected for are one and the same. It is more difficult to see, however, why we ought to count the function in virtue of which a part was originally selected for as a biological function of the part at present if the function for which the part was originally selected for has not been relevant to the part's recent selective history. Again, it is hard to see why we should think of "protection/defense" as a proper function of bird plumage if the plumage no longer functions to protect or defend the bird even if it was in virtue of this function that the part was originally selected for.

None of the above remarks are meant to suggest that understanding a part's ancient selective history is theoretically unimportant. Indeed, evolutionary biologists may be keen to garner a better understanding of why a part originated in the first place and such a project would hinge on understanding the part's ancient selective history. The point of these considerations is to suggest that when it comes to understanding the proper function of biological parts for much of biology, what seems to be important is why the part has been maintained and not necessarily why it originated. Peter Godfrey-Smith's (1994) version of the selected-effects account, the modern history account, pursues a strategy that recognizes the value of focusing solely on the recent, as opposed to ancient, selective history of parts.

Godfrey-Smith's modern history account holds that "functions are dispositions and powers which explain the recent maintenance of a trait in a selective context." (1994, 16) Accordingly, the only relevant selective history to consider when determining proper biological function would be recent selective history and the proper biological function of a part would be whatever function that part has been recently selected for—i.e., maintained in the current population in virtue of. The upshot of this is that the natural norms that govern a given part will be determined by the most recent selective history of the part—i.e., the part *ought* to function in the way that has most recently been responsible for it being selected for. Furthermore, the modern history account explicitly recognizes that a part may be maintained in virtue of more than just a single function; it could be that it is selected for because of several functions and all of these functions would qualify as proper, or biological, functions of the part. Returning to our earlier example, if feathers are maintained in a certain bird species because they aid in thermoregulation and because they aid in flight, then in this bird species, feathers have the proper functions of enabling flight *and* thermoregulation. If feathers in another bird species are maintained only in virtue of their flight enabling capacity but no longer in virtue of their role in thermoregulation, then in this bird species, the proper function of feathers is to enable flight but not thermoregulation even though the feathers may have been originally selected for their thermoregulation function.

The modern history account embodies the theoretical spirit of the naturalistic, teleological program insofar as it understands the proper function of biological parts to be intimately connected with the function the parts were "designed" via natural selection to perform. Furthermore, the account is able to embody this teleological spirit while simultaneously avoiding the problems that Millikan's teleological account encounters as a result of her tying proper functioning to ancient selective history.

2.4.2.1.a. The modern history account and the constraints

For the reasons just discussed, I take the modern history account of functions to be the version of the selected-effects account most likely to play a role in medical theorizing about disease. Indeed, theorists such as Jerome Wakefield (1992, 2011) seem to think that such an account of biological function actually *is* implicit in the notion of disease that is central to much of medicine.[23] To decide whether Wakefield's claims are warranted, we will need to assess whether the modern history account, what I take to be the best performing selected-effects account, satisfies the three constraints mentioned earlier.

Let me begin by noting that the account meets the first leg of the differentiation constraint: it is capable, in principle at least, of accounting for the distinction between normal and abnormal biological states in an objective manner. After all, the account holds that judgments about proper function are hostage to natural norms that issue from matters of fact concerning a part's recent selective history. And while it is less certain, it is likely that this account will also respect core cases since the function in virtue of which a part has been recently selected for is likely to be the sort of function whose disruption would lead to a negative outcome for the organism. Hence, disruptions to the functioning of these parts—i.e., malfunctions—constitute the sort of biological states that are likely to be of interest to objectivist medicine. Of course, before we can really get clear on whether or not the modern history account actually respects core cases, we will need to know about the recent selective history of the part under consideration. In particular, we will need to know what function the part has recently been selected for. Without this information, we will not know how to draw the relevant distinction between functioning and malfunctioning parts that grounds the distinction between biologically normal and abnormal states that is supposed to respect core cases of disease and health. Such concerns about what is

[23] Wakefield (1992) is especially interested in psychiatry.

required in order to determine the proper functioning of parts leads us to the practicality constraint.

A particular challenge for any selected-effects account, the modern history account included, is that it is incredibly difficult to determine why any given part was selected for, if it was selected for at all. To borrow Paul Griffiths'(2009) terminology, the "backward-looking" nature of this type of account requires us to work out the evolutionary function of a part before we can decide if there is a malfunction, a task that seems unlikely for most biological parts since determining design protocol would require us to correctly appreciate the exact environmental pressures that most recently lead to the selection of the part via a particular "fitness-enhancing trait". This feat is not easily accomplished given our inability to "view" our evolutionary past (see also Davies, 2001; Wouters, 2005; Griffiths, 2009).[24] In essence, the problem is as follows: focusing attention on selected-functions, regardless of whether we focus on the ancient or recent history of parts, requires that we take a particular stance on our evolutionary past even though we do not have reliable access to information about this past. Granted, we have bits and pieces of information in the form of fossil records and genetic sequences, but we probably do not have enough to warrant the speculative conclusions needed to support this sort of project.[25] If we do not know the function in virtue of which a part has been selected for, then we will not be able to determine its norms. This problem suggests that we cannot rely on the notion of norms this account offers since we have no way of being certain that we have actually determined the actual

[24] E.g., were feathers selected for because they allowed flight, or because they allowed for thermoregulation?

[25] This isn't to deny evolutionary theory. Rather, it is only to say that we ought to be wary of employing such an approach, especially if better performing accounts are on offer, since claims about health and disease often carry significant impact.

norms of the part and, therefore, will be unable to draw the relevant distinction between normal (i.e., functional) and abnormal (dysfunctional) states.[26]

The previous point suggests that even though the account meets the differentiation constraint in theory, it falls short because it is unlikely that the distinction can be employed in practice owing to the epistemic constraints of humans (i.e., the inability to know for certain about our evolutionary past). This fact, in turn, suggests that this notion of biological function is probably not what medical theorists actually rely on when reasoning about disease and health states. Put another way, the failure to satisfy the practicality constraint suggests that core medical practices probably do not ground the distinction between disease and health in a selected effects theory of biological function.

One may find the above considerations less threatening than I do. After all, much scientific work traffics in probabilities and uncertainties. Why should we think that the inability to know for certain about the selective history of biological parts should be grounds to reject the modern history account when we are so prone to uncertainties in science? This response is not without merit. The problem for the modern history account, however, is that even if we could somehow deal with the problem posed by the practicality constraint, the account would still seem unlikely to be the one employed by most medical theorists since it fails to meet the foundation constraint.

Concerning the foundation constraint, the modern history account of function appears to employ a notion of normal biological function that is only rarely, if ever, of interest to the biological sciences that are relevant to medicine. Indeed, the discussion of the normal functioning of parts in medicine appears rather insensitive to why and how parts evolved (Craver,

[26] Wakefield (2009) sometimes speaks as though claims about something being a dysfunction are actually just defeasible, speculative claims. The problem isn't that the claims are defeasible—indeed, in science most claims are. The problem is that the means to determine the probability of such claims is highly suspect given the speculative nature of evolutionary theorizing—i.e., theorizing about why certain traits evolved, not that they did evolve.

2001; Davies, 2001; Wouters, 2005; Griffiths, 2009; Ereshefsky, 2009). For this reason, it seems that the modern history account of function in particular, and selected-effects accounts in general, is grounded in a different sort of biological theory (i.e., historical evolutionary biology) than that employed in most medical practices (e.g., functional biology such as physiology, cellular and molecular biology, genetics, and anatomy) and, therefore, fails to meet the foundation constraint.

One may respond to the above discussion concerning the foundation constraint that evolutionary biology actually is relevant to medicine insofar as medical theorists are interested in understanding how biological parts contribute to the evolutionary fitness—i.e., [27]survival and reproduction—of individuals. Given that medicine actually is informed by evolutionary theorizing, the objection goes, we ought to be wary of assuming that the modern history account is not grounded in the right sort of biology. My response to this objection is to note that the issue here is not with the relevance of evolutionary theorizing *per se*; rather, following Griffiths (2009), the point is that a certain type of evolutionary theorizing, one that is committed to understanding the norms of biological functioning as being determined by the part's evolutionary history—both ancient and recent selective history—as the selected effects account does, is misguided. It is true that medicine appears to be interested in, or guided by, evolutionary biology insofar as it tries to understand how humans as biological systems can attain their highest level goals of individual survival and reproduction, goals proposed by evolutionary theory (Boorse, 1977; Murphy, 2006). The relevance of these goals to medical knowledge, however, does not require, nor even indicate, that medicine is interested in the sort of norms that the modern history account proposes (i.e., why parts were selected for); rather, it only suggests that there is an interest in understanding how biological parts contribute to the survival and reproduction of organisms at present, regardless of

[27] Medicine is probably most interested in how parts contribute to the evolutionary fitness of individuals—especially individual survival—and less interested in how they contribute to the biological fitness of groups—i.e., tribes, families, species. It is likely, however, that understanding biological function sometimes requires theorists to think in terms of the fitness of groups.

why the parts were selected for. The next theory of biological functioning that I discuss is explicitly evolutionary in this "forward-looking" sense (Griffiths, 2009).

In conclusion, it seems clear that selected-effects accounts of biological function are of interest, and maybe even central, to those branches of biology that aim to understand why biological parts originated and have been maintained throughout an organism's history. But, these sorts of accounts are unlikely to characterize the notion of biological function that medicine employs (though maybe only implicitly) to objectively ground the distinction between health and disease since they fail to satisfy the practicality constraint and the foundation constraint that a theory of biological function needs to satisfy if it is to play the grounding role. We now move on to our other candidate account of biological function, the Evolutionary-systemic-capacity account.

2.4.2.2. The Biological function component: The evolutionary-systemic-capacity account

I now present the evolutionary-systemic-capacity account. I begin with a discussion of the systemic-capacity approach since this provides the theoretical foundation for the account I propose. I then turn my attention to several modifications that need to be made. In discussing these modifications, I develop the evolutionary-systemic-capacity account of biological functions, an account that borrows heavily from Paul Griffiths' account of biological function (2009). It is my claim that the evolutionary-systemic-capacity account of biological function is implicitly employed in medical theorizing about disease.

The systemic-capacity approach holds an item's function to be the capacity that the item contributes to bringing about another capacity of a system that contains the item as a part (Cummins, 1975). As Cummins notes, "[w]hen a capacity of a containing system is appropriately explained by analyzing it into a number of other capacities whose programmed exercise yields a manifestation of the analyzed capacity, the analyzing capacities emerge as functions." (1975,

765) Such "functional analyses" proceed as follows: first determine the systemic function of a part by choosing a capacity of interest that is produced by the containing system; then analyze the system into the capacities and parts that play a role in bringing about the capacity of interest. Finally, whatever capacity a part contributes to the overall capacity of interest will be attributed to that part as its function relative to the capacity of interest. More formally, the systemic-capacity approach takes the following form:

> "X functions as a # in S (or the function of X in S is to #) relative to an analytic account A of S's capacity to * just in case X is capable of #-ing in S and A appropriately and adequately accounts for S's capacity to * by, in part, appealing to the capacity of X to # in S." (Cummins, 1975, 762)

Accordingly, the function of a carburetor with respect to a car's capacity to locomote is to mix fuel and air as this is what the carburetor contributes to the car's capacity to locomote. The function of the kidneys with respect to a human's capacity to survive is to filter toxins from the blood as this is the activity of kidneys that contributes to a human's capacity to survive. For the remainder of the paper, I will use 'systemic-function' to refer to the function attributions that result from applications of the systemic-capacity approach.

A feature of the systemic-capacity approach that deserves mention is its' alleged inability to posit systemic-malfunctions or dysfunctions (Millikan, 1989b; Neander, 1995; Davies, 2001). The idea is that a part cannot systemically-malfunction since the part is allegedly identified solely by its contributed capacity (i.e., its systemic-function) and if it fails to contribute the capacity it will fail to be identifiable as something with *that* systemic-function. And, if it cannot be identified as having *that* systemic-function, it cannot be said to be a thing that is systemically-malfunctioning. The problem, in other words, is that a part cannot be identified if it doesn't perform its systemic-function; hence, it cannot malfunction. Several examples may prove useful in clarifying this alleged feature of the approach. If the systemic function of a carburetor is to mix fuel and air, and carburetors can only be identified by their systemic-functions, then a part

that occupies the same location in a car's engine but does not mix fuel and oxygen would not be identifiable as a *malfunctioning* carburetor since it will not be identifiable as a carburetor. In the biological realm, if the systemic-function of the heart is to pump blood, and hearts can only be identified in virtue of fulfilling that systemic-function, then a part that occupies the same location in a body as a heart but doesn't pump blood will not be identifiable as a *malfunctioning* heart since it will not be identifiable as a heart.

If it were the case that the systemic-capacity approach did not allow for the malfunctioning of parts, then it would be difficult to see why we should think that this approach could possibly be the sort that is employed in the biological sciences relevant to medicine. After all, much of medicine seems to be interested in understanding not only the functions of biological parts but also in understanding how such parts can malfunction and how we can get malfunctioning parts back to their functional state. The core idea of objectivism, recall, is that the health/disease distinction is grounded in the normal/abnormal biological state distinction that is grounded in the biological function/dysfunction distinction. A theory of biological function that doesn't respect the possibility of malfunctions (i.e., dysfunctions) is unlikely to be employed, either explicitly or implicitly, in medicine and, therefore, will be of little use to our project.

Of course, it could turn out that the systemic-capacity approach is capable of recognizing malfunctioning parts. If this were true, then we would have one less reason to question the claim that biological functions are a sort of systemic-function. Showing that this is possible would only seem to require showing that biological parts can be identified independently of their systemic-functions. That such parts can be identified in other ways has become widely accepted. The strongest case for this claim issues from the work of Carl Craver (2001; for earlier discussion of similar ideas see Armundson & Lauder, 1994). Craver argues that in many cases, especially those involving biological systems, parts can be identified not just in virtue of their systemic-functions but also in virtue of their organized location within their containing system. The

general idea behind Craver's proposal is that the functional analysis of a containing system that exhibits certain capacities can be thought of as a mechanistic analysis of a mechanism that gives rise to a certain phenomenon. To see how this move solves our problem, let us begin by recognizing that a containing system can be construed as a mechanism and the relevant containing system capacity can be understood as the phenomenon of interest. The mechanistic analysis involves decomposing the containing system (i.e., mechanism) into the component parts whose organized activity gives rise to the relevant containing system capacity (i.e., phenomenon of interest). The activities of each part that are picked out in a mechanistic analysis are taken as equivalent to the systemic-functions attributed to each part in a functional analysis. The value of thinking in terms of mechanisms is that it forces us to recognize that parts within a containing system often must be organized in a certain way in order to give rise to the relevant containing system capacity (Craver, 2001). Both carburetors and hearts need to occupy certain locations in their respective containing systems if they are to perform their systemic-functions of mixing air and pumping blood, respectively. A carburetor needs to be connected to the fuel line, have access to ambient air, and be connected to the manifold intake valve if it is to perform its' systemic-function of mixing fuel and air. A human heart needs to be roughly in the center of the chest and connected to four valves in a particular manner if it is to fulfill its systemic-function of pumping blood. That the parts of containing systems need to be spatio-temporally organized in a certain way affords us an alternative means to identify the parts of containing systems; namely, parts can be identified by their spatio-temporal locations within the organized systems (Craver, 2001). The upshot of Craver's proposal is that it is possible to identify the parts of containing systems even if they do not perform their systemic-functions, thereby making the attribution of systemic-malfunctions possible. This is a boon for the systemic-capacity approach since it removes one of the reasons to question its usefulness for those domains of inquiry—i.e., functional biology and medicine—that frequently posit and reason about malfunctioning parts.

Another feature of the systemic-capacity approach worth mention is the promiscuous or liberal manner in which it attributes systemic-functions (Millikan, 1989; Neander, 1991, 1995). Because the systemic-function of a part is always identified relative to the "the systemic capacity we wish to explain", single parts are capable of having multiple systemic-functions insofar as they may figure in more than one containing system or because the containing system of which they are parts may have more than a single capacity (Davies, 2001, 25). The systemic-function of the heart, for example, relative to the circulatory system's capacity to move blood and nutrients throughout the body is to pump blood, *and* the systemic-function of the heart relative to the physician-patient system's capacity to detect an irregular heartbeat is to thump. The fact that the systemic approach attributes so many functions to parts is often referred to as the problem of promiscuity or liberality (Davies, 2001; Wouters, 2005b). If the liberality charge holds, one may worry that we are then faced with the practical problem of determining which of the plethora of systemic-functions ought to be counted as biological functions.

The charge of liberalism is justified. After all, there is no shortage of potential capacities of biological systems that are subject to functional analysis—e.g., the capacity of the heart to stop unexpectedly, the capacity of the circulatory system to be damaged by a bullet, etc. What seems questionable, however, is whether promiscuity really is as deep a problem for an account of biological functions as some have supposed (esp. Millikan, 1989; Neander, 1991). After all, there is nothing inherently problematic about the systemic-capacity approach assigning multiple systemic-functions to biological parts since biologists need not consider every systemic-function attributed to a part. In fact, they can always simply ignore those systemic-functions that result from considering a part's contributing role to some systemic capacity that is not of interest. Returning to our previous example, biologists needn't recognize the 'thumping" of the heart to be a systemic-function of the heart unless they recognize the systemic capacity of detecting heart beat patterns as a systemic capacity of interest to biology. To the extent that this capacity is of

interest to biology, then they should accept this systemic-function of the heart as a relevant biological function of the heart (see Craver, 2001 for more on this point). Of course, one may argue that the above strategy doesn't really help to assuage concerns with the approaches ability to identify biological functions since we still do not have a way to identify which systemic-function(s) *should* count as a part's biological function(s). Indeed, this seems to be the real problem that issues from promiscuity—that the systemic-capacity approach does not offer any way to determine which systemic-functions *ought* to count as biological functions.

Luckily, the systemic-capacity approach can be made to handle this problem with a slight modification. The relevant modification holds that we treat the organism as a whole as a containing system that exhibits certain capacities that are to be functionally analyzed. In order to maintain naturalistic integrity, the modification restricts our focus to only those systemic capacities of organisms that are recognized by the biological sciences. Given the widespread acceptance of evolutionary theory, the modulation suggests that we take the systemic capacities of organisms as a whole to be survival and reproduction (Boorse, 1977; Wouters, 2007; Griffiths, 2009).[28] Whatever systemic-function a biological part contributes to the organism's ability to survive and reproduce will be identified as that part's biological function. Such an understanding affords us a means to determine the biological function of biological parts: it is the part's systemic-function relative to the organism's capacity to survive and reproduce. This, in turn, allows us to determine which systemic functions should count as biological functions.

Employing such a strategy as that just discussed essentially compels us to accept Paul Griffiths' (2009) account of biological functions, an account that I term the evolutionary-systemic-capacity account (hereafter, the E-systemic-capacity account).[29] Griffiths explicit

[28] There may be other higher-level goals of humans recognized by functional biology, but it is unclear what they might be.

[29] It should be noted that this account is intellectually indebted to that proposed by Christopher Boorse (1977) whose position I discuss in more detail in section 2.4.2.2.a.

proposal is that "[a]ll and only those parts and processes that contribute to the capacity of an organism for survival and reproduction, construed in terms of our current best theory of evolutionary dynamics, are aspects of its biological function" (2009, 18). Functions attributed to biological parts by this account are what I will call E-systemic-functions to mark that they are a special subset of systemic-functions. Accordingly, the E-systemic-function of the circulatory system is to distribute oxygen and nutrients throughout the body because this capacity of the circulatory system is what contributes to the systemic capacity of survival and reproduction. The E-systemic-function of the heart is to pump blood and not make a thumping sound since it is in virtue of the former that the heart contributes to the distribution of oxygen and nutrients throughout the body, which in turn contributes to the organism's systemic capacity of survival and reproduction. The upshot of employing the E-systemic-capacity account is that we have a way to sidestep the problem of promiscuity since we now have a principled means to distinguish those functions relevant to biology and, therefore, medicine, (i.e., the E-systemic-functions) from those that are not.

Since the notion of E-systemic-functions plays such a large role in this project, it is probably worthwhile to state explicitly what E-systemic functions are and to say a bit more about how functional biologists identify them. The E-systemic function of a biological part is the function that a part contributes to an organism's capacity to survive and reproduce. The E-systemic-function of the heart, for example, is to pump blood *in a way that is conducive to the organism's survival and reproduction*. A heart that pumps blood but does so in a way that is incapable of sustaining the organism's life would be an E-systemically-malfunctioning (i.e., abnormal) heart since it fails to fulfill its' E-systemic-function of pumping blood in a way that actually contributes to the organism's survival.

Identifying the E-systemic function(s) of biological parts is a task that falls to functional biologists, not medical theorists. How these theorists go about determining these functions is

sometimes easy and sometimes difficult.[30] Easy cases are those where there is more or less general consensus among theorists (esp. functional biologists) that a particular function is an E-systemic function of the biological part—i.e., that a given function of the part actually contributes to the organism's ability to survive and reproduce. Difficult cases are those where there is widespread disagreement concerning the part's E-systemic function(s)—i.e., where there is widespread disagreement as to whether a proposed E-systemic function actually contributes to

[30] In order to determine the E-systemic function of a part, it may sometimes prove necessary to employ the use of reference classes. Following Boorse (1977), I take the use of reference classes—subsets of an over-arching class—to be an important tool in understanding E-systemic functions since it is likely that some biological parts will have biological functions that vary with respect to the organism's age, sex, etc. For example, the testicles of pre-pubescent males are not malfunctioning because they fail to produce sperm, nor are the reproductive systems of post-menopausal women malfunctioning if they fail to menstruate. It is an altogether different situation, however, if the testicles of a twenty year old man fail to produce sperm or a twenty year old female does not menstruate. In these cases, it does seem that the biological parts are E-systemically malfunctioning. These points illustrate that we will sometimes need to know which reference class an individual falls into if we aim to determine whether a part is E-systemically malfunctioning or not. The necessity of the employment of reference classes serves to remind us that a single biological part may have different E-systemic functions for different reference classes—e.g., different E-systemic functions for the different sexes or at different points in the life-cycle.

Determining exactly which reference classes will need to be recognized goes beyond the scope of this paper. Nonetheless, there are some that are quite certain to prove useful, while others are certain to prove useless. Sex, pre/post-pubescence, and pre/post-menopausal are several reference classes that are likely to prove very useful for functional biologists interested in understanding the E-systemic functions of biological parts. It is less clear, however, that we should follow Boorse (1977) and think of age groups as reference classes for the purpose of determining E-systemic function. After all, it doesn't seem to matter whether a person is 10 years old or 90 years old, E-systemically malfunctioning hearts are E-systemically malfunctioning. It may very well turn out that E-systemically malfunctioning hearts in 90 year olds are much more common than they are in 10 year olds. And because of this, we may be more alarmed to find such a heart in a 10 year old than we would be to find it in a 90 year old. We might also think that the presence of this E-systemic malfunction in the 10 year old is an indicator of some other underlying E-systemically malfunctioning part which we might not imagine afflicts the 90 year old. Nonetheless, both people would be enjoying an E-systemic malfunction even though it is a fairly typical condition for the person that falls in the 90+ year old reference class but not for the person that falls in the 10-20 year old reference class. The point is that using age by itself as a reference class is unlikely to prove useful when attempting to determine the E-systemic function of biological parts. Whether or not age will prove useful as a reference class for other purposes such as risk assessment, diagnosis, etc. is a question I leave unaddressed.

As a matter of fairness, it is worth pointing out that Boorse (1977) probably focused on age as a reference class since he took the proper function of biological parts to be revealed by the species- typical function of the part—i.e., the biological function of the part is the function that the part makes to the organism's ability to survive and reproduce in *most* members of the reference class. If one accepts Boorse's notion of proper biological function, then age might seem more relevant to determining a part's proper function. After all, plaque-filled arteries, for instance, are very likely species typical—i.e., the statistical norm—for the 90+ year old reference class but not for the 10-20 year old reference class. Notice, however, that if we follow Boorse's line of reasoning, then it seems that we would need to claim that the contribution made by the plaque-filled arteries determines the biological function of arteries for the 90+ year old reference class. Accordingly, plaque-filled arteries that do a very poor job of aiding circulation would count as properly functioning arteries because this is the species-typical contribution of arteries for this reference class. This conclusion seems to offer a *reductio* of Boorse's position: plaque filled arteries that do a poor job of circulating blood are not properly functioning even if that is the statistical norm and any account that says they do is sure to be mistaken. In section 2.4.2.2.a I offer further reasons to question Boorse's position and to think that the E-systemic capacity account outperforms his species-typicality account of proper function.

organismic fitness.[31] Claims about the E-systemic function of a part, and any ensuing claims about the part's malfunctioning, are defeasible. The likely defeaters for such claims are future developments in evolutionary theory that give us reason to reconsider the part's actual contribution to individual survival and reproductive ability. Despite the defeasibility of these claims, once biologists *believe* that they understand a part's E-systemic function (i.e., once a function has been localized—i.e., attributed—to the biological part), they are then in a position to attempt to mechanistically decompose the responsible part, a process in which they attempt to understand how the components of the part work together, in an organized way, to give rise to the relevant E-systemic function (for more on the process of localization and decomposition see Bechtel & Richardson, 1993).[32] The upshot of the decomposition process is an understanding of the mechanistic nature of the properly functioning biological part.

Now let us briefly consider the notion of E-systemic malfunctions. A part counts as E-systemically malfunctioning whenever it fails to make the contribution to individual survival and reproductive ability that such parts actually do make. In some cases it may be relatively easy to identify a malfunctioning part and in others it may be more difficult. The ease with which such malfunctions are identified probably has to do with how well functional biology understands the alleged malfunctioning part—i.e., whether the part, when properly functioning, has been mechanistically decomposed. A developed understanding of the biology and function of the human heart, for example, make it relatively easy to tell when a particular human heart is malfunctioning. In other cases it may be difficult to determine with certainty whether a part is malfunctioning even when we have a developed mechanistic understanding of the part since it may be unclear whether the part is still making its normal contribution to evolutionary fitness.

[31] Of course, as I will mention shortly, even those cases where there is widespread consensus about a part's E-systemic function could turn out to be incorrect if the sort of theoretical reasoning that has led to the consensus view is incorrect.

[32] A possibility is that theorists will sometimes engage in mechanistic decomposition of a part prior to reaching agreement about the part's E-systemic function. Furthermore, it may be that information gained during the mechanistic decomposition leads theorists to revise prior claims about the part's E-systemic function.

The uncertainty regarding the part's contribution to evolutionary fitness is possibly due to genuine ontological vagueness with respect to the function being fulfilled—i.e., for some parts, at some times, it may simply be the case that there is no fact of the matter as to whether the part is functioning in such a way that it counts as still making its normal contribution to survival and reproduction. Such ontological vagueness with respect to malfunction is likely to be the exception, not the rule. Uncertainty resulting from epistemic limitations, including, but not limited to, failing to appreciate the mechanistic nature of the properly functioning part, is the more likely culprit responsible for the inability to identify malfunctioning parts. Fortunately, such limitations are not insurmountable and are likely to lessen as functional biology increases its understanding of the human biological system. Despite the difficulties that might arise as a result of genuine ontological vagueness and epistemic limitations, in most cases, it will be possible and probably quite easy to identify E-systemic malfunctions.

Another important component of identifying E-systemic malfunctions has to do with the fact that the attribution of a malfunction to a biological part requires that the failure of the part to perform its relevant E-systemic-function is not attributable to the failure of some other part's E-systemic-function. The failure of protein production in a cell, for instance, can be said to result from an E-systemic-malfunction in that cell's mRNA as long as we have no reason to think that any other E-systemic failure of some other part of the cell explains the failure of protein production. If, however, we found out that the cell does not have any ribosomes owing to some genetic mutation, then we would need to reconsider our claim that mRNA was malfunctioning since we now have another E-systemic-malfunction that explains the failure of protein production; namely, the E-systemic-malfunctioning DNA that is responsible for the absence of ribosomes. The point to appreciate is that determining whether a part is malfunctioning will often require us to explore why and how a part is malfunctioning. Consider a heart that fails to pump blood and is said to be E-systemic-malfunctioning. This heart has failed to perform its E-

systemic-function. Let us now imagine that upon closer inspection we find that the reason the heart cannot perform its E-systemic-function is because a number of veins have been severed and, therefore, are unable to perform their E-systemic-functions of bringing blood to the heart. It now becomes clear that the E-systemic-malfunction of the heart results from an E-systemic malfunction of the veins. Having such knowledge may prove useful in our attempt to restore proper E-systemic-functions to parts. In the above case, barring damage to the heart resulting from oxygen deprivation, restoring the veins such that they can perform their E-systemic-functions would cause the heart to return to performing its E-systemic-function. These considerations should serve to remind us that identifying E-systemic malfunctions is not always a simple task but it is one that can usually be accomplished with time and effort.

2.4.2.2.a. The E-systemic-capacity account and the constraints

Now that the E-systemic-capacity account has been introduced, we should consider how well it handles the three constraints. Let's begin with the foundation constraint. One feature of the E-systemic-capacity approach that suggests it is grounded in the right sort of biology is that it is developed by exploring how functional biology, the sort of biology relevant to medicine, reasons about biological functions (Wouters, 2005; Griffiths, 2009). The upshot of developing the account in this manner is that functional biology disciplines such as physiology, anatomy, molecular biology, cellular biology, and systems biology all end up being interested in E-systemic functions. Indeed, insofar as functional biology is interested in understanding how biological parts are organized and function in order to allow organisms to survive and reproduce, one might say that functional biology is committed to (though perhaps only implicitly) the E-systemic-capacity account of biological functions. The fact that the E-systemic-capacity account is developed in step with the reasoning strategies and practices of functional biology insures that it is grounded in the right sort of biological theory.

Two potential objections to the foundation constraint need to be addressed before moving on. The first concerns the evolutionary component of the E-systemic-capacity account and the second concerns the interest that biomedical theorists may have in parts that decrease survival and reproduction. One may question whether the evolutionary aspect of the E-systemic-capacity approach hinders its ability to satisfy the foundation constraint. After all, one of the reasons the selected-effects account was found lacking was because it was grounded in a sort of biological theory—"backward-looking" historical evolutionary biology—that I claimed was not relevant to most of medicine. Might this sort of fate also befall the E-systemic-capacity account in virtue of its reliance on evolutionary theory? My response is that the evolutionary component of the E-systemic-capacity account—namely, the dictum that we should focus on the systemic capacities of survival and reproduction—actually further suggests grounding in the right sort of biological theory. As mentioned earlier, the emphasis placed on understanding the organismic goals of survival and reproduction in light of our current understanding of evolutionary theory suggests that the account is best thought of as a "forward-looking" evolutionary account insofar as it focuses on the current and future prospects of organisms given evolutionary pressures as opposed to the "backward-looking" evolutionary approach which focuses on the historical pressures that lead to the selection of a certain part (Griffiths, 2009). As Griffiths notes, most of functional biology seems to be implicitly committed to such a "forward-looking" evolutionary approach at least to the extent that it is interested in understanding the contributions that biological parts make to organismic survival and reproduction (2009). Because the evolutionary component of the E-systemic-capacity account is also found within the sort of biological theory of relevance to medicine (i.e., functional biology), its appearance in the E-systemic-capacity account only further suggests that the account satisfies the foundation constraint.

The second objection notes that the E-systemic-capacity account faces the problem of accounting for the function of biological parts that do not contribute to survival and reproduction

(e.g., pathological states, etc.). This is a problem for the account because large areas of functional biology, especially aspects central to medicine, appear to be interested in understanding the function of the parts of pathological biological systems, parts that do not contribute to survival and reproduction.[33] A proper account of the sort of functions implicit in medical theorizing, the objection argues, will need to be able to account for these "pathological" functions of biological parts. This objection suggests that the E-systemic-capacity account of biological function cannot be the sort that medicine actually relies on since it is incapable of doing all the work that an account of functions needs to do.

The solution to this problem is quite simple for the claim of this project is not that there is just one type of function—E-systemic-functions—that properly describes the functions relevant to medical theorizing. Rather, the claim is that medical theorizing only relies on E-systemic functions to understand biological normalcy. But this does not deny that medicine sometimes employs other notions of function, perhaps the notion of systemic-function, if doing so could be useful. Instead, it only means that any other functions that may be attributed to a biological part will not be recognized as E-systemic-functions, and, therefore, would not be used to ground a distinction between normal and abnormal biological states. An example may prove useful. Let us consider a case where biologists attempt to understand the systemic-function of a malfunctioning dopamine system in depression. Like any attempt to determine a systemic-function, the first step is to identify the systemic-capacity of interest. In this case, the capacity of interest is the depressive state; hence, we may label the systemic-functions relative to this capacity depression-systemic-functions (or, d-systemic-functions). We then determine what capacity the dopamine system contributes to the depressive state and take this to be the d-systemic-function of the dopamine system. The upshot: an account of the d-systemic-function of

[33] Though it seems possible that some part could be biologically malfunctioning and also contributing to an organism's ability to survive and reproduce.

an E-systemic-malfunctioning part (i.e., the dopamine system in this hypothetical case is the E-systemic malfunctioning part since the dopamine system's E-systemic function is to regulate dopamine in a way that contributes to survival and reproduction and it fails to do this here).[34] In contrast to the claims of the second objection, it appears that there is no reason to think that acceptance of the E-systemic-capacity account of biological function precludes medical theorists from employing the systemic-capacity approach as a means to determine the various systemic-capacities of biological parts. Indeed, numerous capacity relative systemic-functions of biological parts may be of interest to functional biologists and medical theorists but these functions cannot play a role in grounding the distinction between health and disease because they do not constitute biological functions. E-systemic-functions do constitute biological functions and, therefore, are capable, and likely, to play a role in medical theorizing about disease.

Now let us consider the practicality constraint. The account meets this constraint insofar as there is no obvious reason to think that theorists cannot identify E-systemic functions and malfunctions. Indeed, as Griffiths (2009) and Wouters (2005) have recently pointed out, the sort of functions attributed to biological parts in functional biology tend to be those that would also count as E-systemic functions. The reason for this is not mere chance. Rather, the suggestion is that functional biologists are often interested in understanding the roles that various parts play in the survival and reproduction of organisms, and, as a result, they are often implicitly, if not explicitly, focused on identifying E-systemic functions (Griffiths, 2009). Again, this is what we should expect given that the E-systemic-capacity account was developed with the intent to capture the sort of reasoning strategies about biological functions employed in functional biology. Another boon for the practicality of this account is that it becomes quite easy to identify the E-systemic-functions of biological parts (and, as a result, to know when a part is malfunctioning)

[34] There are limits to this example; namely, depression in some instances may actually be indicative of a system that is E-systemically functioning. See Hagen, Edward H. "The functions of postpartum depression." *Evolution and Human Behavior* 20.5 (1999): 325-359.

insofar as these just are the functions attributed to biological parts in the textbooks of functional biology. That much of biology is already engaged in identifying E-systemic functions, even if they do not call them this, suggests that the account is practically applicable in the sense that medical theorists will be able to identify the E-systemic functions of parts. Indeed, for the most part, medical theorists can punt the work concerned with determining the E-systemic function of parts to functional biologists, while focusing their attention on how these parts can E-systemically malfunction, what they look like when they do, and how function can be restored.

The proposed division of intellectual labor between functional biologists and medical theorists may give rise to a worry about whether or not it is possible and practical for medical theorists to identify E-systemic malfunctions. As already noted, the identification of E-systemic malfunctions is possible because of the mechanistic decomposition of biological parts that functional biologists provide. These decompositions make it quite easy to appreciate when a part is failing to perform its E-systemic function since this sort of failure—E-systemic malfunctions—entails that the part enjoys a mechanistic breakdown. Accordingly, E-systemic malfunctions can be—in theory, at least—identified whenever we appreciate how a part normally works. Luckily for medicine, such mechanistic decompositions are available for many biological parts making the identification of E-systemic malfunctions a practical goal. I will have more to say about how medical theorists identify such malfunctions in a later section of this chapter (section 2.5.2).

Finally, we need to consider whether the account satisfies the differentiation constraint. The account appears to be able to handle this constraint. After all, normally functioning parts just are parts that perform their E-systemic-function, while abnormally functioning (i.e., dysfunctional) parts are those that do not. Furthermore, the account also allows that biological normalcy and biological abnormalcy will likely cover a range of states. To clarify this point, let us begin by noting that any part that is able to performs its' E-systemic-function is within the normal range—i.e., it counts as being in a normal biological state. Abnormal biological states are

those that result from parts that can no longer perform their E-systemic-function and this failure is not attributable to the malfunction of some other part of the containing system. In concrete terms, a heart is normal (i.e., functioning) insofar as it is able to fulfill its E-systemic-function (i.e., pump blood at a level that insures the organism's survival), while a heart that fails to fulfill this function (i.e., one that fails to pump blood at a level that insures the organism's survival) would count as abnormal (i.e., malfunctioning). The account also seems likely to satisfy the second aspect of the differentiation constraint: namely, that it will respect core cases of disease and health. This feature falls out of the fact that E-systemic-malfunctions will be attributed to parts that fail to make their contribution to organismic survival and reproduction—just the sort of states that are bound to count as core cases of disease. In conclusion, the E-systemic-capacity account satisfies the three constraints suggesting that it properly characterizes the notion of biological function that is likely involved in medical theorizing about disease.

Before concluding this section, a final objection needs to be considered. The objection notes that it may be better to think of the norms that govern proper biological function in terms of species-typicality, not E-selected functions as I have been arguing. This objection hinges on the idea, first proposed by Christopher Boorse (1977), that the proper function of a biological part is the function that the part normally plays in the members of a given reference class of a species, where "normally" is to be understood in the sense of statistically normal or species-typical—i.e., it is the contribution the part makes in most members of the reference class. The idea of the "specific reference class" is employed in order to account for the fact that the statistically normal function of a biological part may vary among certain subsets of the species—i.e., between the sexes, between pre- and post-pubescence, etc. That proper function just is statistically normal function entails that a part is properly functioning whenever it functions in a manner that is consistent with the statistical norm for the relevant reference class. A part is malfunctioning insofar as it fails to function in this "species-typical" manner. Accordingly, a heart that functions

in the way that most human hearts do is a properly functioning heart; one that does not function in this way is a dysfunctional heart.

Boorse's approach is interesting and it has intuitive appeal, but it appears to fall short insofar as it does not properly describe the notion of biological function employed in functional biology, the relevant biological science (Murphy, 2006, 2009; Ereshefsky, 2009). As a result, it is unlikely to be the notion of biological function implicitly employed in medicine. To see why it falls short one need only recognize that even if it was known that it was statistically normal in the human species for arteries to be plaque filled, this would not be reason for functional biologists to take plaque-filled arteries to constitute properly functioning arteries, nor would the proper function of the leg cease to be "providing locomotion" if every member of the human species happened to have broken legs simultaneously. Reliance on species typicality (i.e., statistical normalcy) as a means to determine the proper function of biological parts, however, would force us to concede that the proper functions of these parts, in these situations, ceases to be "allowing for blood circulation" and "providing locomotion". As others have noted, that functional biologists would be hesitant to concede that the imagined scenarios entail a change in the part's proper function suggests that functional biology is simply not interested in the species-typical function of parts, rather, it is interested in, as already noted, the E-systemic-functions of parts (Murphy, 2006, 2009; Ereshefsky, 2009).[35]

Importantly, none of the above is meant to suggest that species-typicality and statistical normalcy have no role to play in how theorists come to understand the proper functions of biological parts. In fact, it is probably the case that theorists often assume that the statistically normal function of a biological part *is* the part's biological function since these two are likely to dovetail in many cases—the function that a part contributes to an organism's ability to survive

[35] Another point in favor of this claim is that functional biology does not seem to make reference to species typical, or statistically normal, function when describing the functions of parts, a fact easily verified by perusing physiology texts.

and reproduce is likely to be the function that the part typically contributes to this goal in members of the species. But, the fact that statistical normalcy is treated as a heuristic to understand a part's biological function does not entail identity between the two. That theorists could, and have, held that properly functioning biological parts are not species typical—e.g., plaque-free arteries, non-decaying teeth, etc.—is further evidence that statistical normalcy should not be equated with biological normalcy. These remarks suggest that the species-typical notion of norms is not employed in functional biology and for this reason we ought to be wary of using it to ground our theory of biological function. In conclusion, this objection fails since the proposed notion of norms—species-typicality—is found to be theoretically suspect.

2.4.2.3.Objective accounts of disease: E-systemic –functions and biological normalcy

It may prove useful to recap the last section since I am claiming that the concept of biological normalcy that plays a role in medical theorizing about disease and health is grounded in the E-systemic-capacity account of biological function. The E-systemic-function of a part is the function that a part actually contributes to the organism's capacity to survive and reproduce. A part is in a biologically normal state if it is able to fulfill its E-systemic-function; the very same part would be in a biologically abnormal state if it were unable to fulfill its E-systemic function. For the remainder of this project I will use the terms 'functioning' and 'malfunctioning' as shorthand for 'E-systemically functioning' and 'E-systemically malfunctioning'.[36] Whenever I intend to refer to a different type of malfunction or function (e.g., 'modern-selective history function' or 'systemic-capacity function'), I will make this explicit.

The E-systemic-capacity account provides norms that govern biological functioning insofar as they dictate how a part ought to function if it is to be considered a normally functioning part. The E-systemic-function of a part sets the norm that governs the part. Human hearts *ought*

[36] It should be remembered that I am using 'malfunction' and 'dysfunction' interchangeably.

to pump blood in a way that allows humans to survive and reproduce because that is the function that hearts actually contribute to the capacity of humans to survive and reproduce. These norms are natural since they are objective matters of fact that are discoverable by science, a fact that follows from it being an objective matter of fact what function a part actually contributes to an organism's capacity for survival and reproduction. The E-systemic capacity account holds that functional biology, the biological science most relevant to medicine, tends to rely on a notion of biological function that is essentially the same as that proposed by the E-systemic-capacity account. Additionally, the account does not hold that only E-systemic-functions are ever of interest to functional biologists and medical theorists. Rather, the claim is much more modest in that it holds that E-systemic functions are the only sort of functions that constitute *biological functions*, at least insofar as functional biology is concerned.

Recall that the goal of this section (section 2.4) was to get clear on the notion of biological normalcy and biological function that are likely to play a role in medical theorizing about disease and health. I began the section by pointing out that any reasonable objective account of disease will hold that diseases are, at least partially, abnormal biological states. I then proposed that biological normalcy for medicine is best understood as being grounded in proper—i.e., normal—biological functioning and that the relevant notion of biological function for the biological sciences which medicine is theoretically grounded in is E-systemic-function. Along the way, I argued that the E-systemic–capacity account of biological function and the notion of biological normalcy that it grounds are implicit in medical theorizing about disease. The upshot of these considerations is that medicine does seem to be interested in a notion of disease that is, at least partially, objective since it takes being an abnormal biological state (i.e., being a biologically dysfunctional biological part) to be a necessary feature of diseases. Now, however, we need to consider which sort of objective account of disease medicine is actually committed to: the pure-objectivist account or the partial-objectivist account.

2.4.2.4.Objective accounts of disease: pure-objectivism vs. partial-objectivism

Up to this point, I have only tried to show that medicine is likely committed to an objectivist account of disease in the sense that it understands it to be a necessary feature of diseases that they are abnormal biological states. This much seems to be true—medicine does seem to see diseases as states that are necessarily biologically abnormal. What is less clear, however, is whether medicine takes all biologically abnormal states to be diseases. This issue pushes us to address the question of whether medicine is committed to a pure-objectivist view of disease or a partial-objectivist view of disease. The pure-objectivist account holds that the disease/health distinction involves nothing more than the normal/abnormal biological state description. That is to say, the pure-objectivist takes biologically normal states to be healthy states and biologically abnormal states to be disease states. The strength of this position is that it is purely objective; hence, it insures that medicine can be a purely objectively grounded discipline.

Interestingly, this approach has very few proponents, even among those who consider medicine to be an objectivist endeavor. The main reason for the poverty of support among objectivists is that the purely objective approach fails to recognize that even though we may be able to derive a notion of biological normalcy that doesn't smuggle in any unwanted human-centered evaluative judgments, it is less clear that we can, or should, offer a notion of disease that does not involve human interests. This concern is often taken to issue from the idea that certain obviously abnormal biological states are not deserving of the label 'disease' since the label tends to suggest that the state is bad or undesirable. A stock example is the gourmet lesion, a brain lesion that causes its bearer to develop gourmet tasting abilities (Regard & Landis, 1997; Murphy, 2006; Ereshefsky, 2009). The idea is that even though there is an obvious biological abnormality—namely, the brain lesion—the state of the bearer of this lesion does not constitute a

disease since there is nothing harmful or undesirable about acquiring gourmet tastes.[37] Given the E-systemic capacity account of biological normalcy, it is not exactly clear that the gourmet lesion would count as biologically abnormal if the only result were increased taste sensitivity unless this could be shown to interfere with the organism's evolutionary fitness. This does not mean that medical theorists will not be interested in the responsible brain lesion, but it would suggest that the brain lesion would not be, strictly speaking, biologically abnormal. It may be considered a statistically abnormal anatomical feature of the mind-brain, and theorists may be interested in understanding how this particular feature—i.e., the lesion—gives rise to gourmet tastes. Indeed, theorists may be interested in statistically abnormal states not because they are harmful or dangerous but because they pose a puzzle about how the human body works. These considerations are meant to illustrate the fact that tying biological normalcy to E-systemic function has the effect of insuring that the actual E-systemic malfunction of most biological parts will be harmful and/or dangerous insofar as they lower the individual survival component of evolutionary fitness. Accordingly, it seems that for most of medicine, pure objectivism holds true: biologically abnormal states are harmful and/or dangerous states insofar as they decrease individual survival, and therefore, constitute diseases.

But there is reason to be wary of accepting the pure-objectivist proposal. In particular, we should recognize that there may be cases where it is actually unclear that a deviation from biological normalcy deserves to be tagged as harmful or dangerous. These cases occur most prominently when we consider E-systemic malfunctions that interfere with reproductive systems and as we wonder into the psychological domain. With respect to the first sort of case, it is unclear that a man that receives a vasectomy or a woman who has a hysterectomy to insure she

[37] See Regard & Landis, 1997. An interesting possibility is that acquiring gourmet tastes could actually turn out to be harmful or undesirable if they interfered with the organism's ability to survive and reproduce. If, for instance, the sufferer's palate was so exquisite that they thought everything besides the best quality food imaginable was unbearable and, therefore, could not eat the food that was available to them, then the state that they enjoy would probably count as both biologically abnormal and harmful or undesirable. Actual cases of this condition do not seem to be so extreme.

cannot become pregnant should count as having diseases even though they do enjoy biologically abnormal states—i.e., they have biological parts that are malfunctioning. Notice, however, that a man who cannot have children not because of a vasectomy but because the *vas deferens* was accidentally disconnected or a woman who was born without a uterus probably should count as having diseases. The difference in these cases seems to be that the biologically abnormal state is undesirable in some of the cases, but not in others (i.e., the man who had the vasectomy or the woman who had a hysterectomy to insure she would not get pregnant welcomes the biologically abnormal state). As we will see later (chapter 4), these sorts of problems multiply when we turn our attention to the psychological domain. In order to handle these problematic cases, we may need to employ the partial objectivist view, a position that has been argued for most prominently by Wakefield (1992) and Murphy (2006).[38] In broad brush strokes, the Partial-objectivist view holds that the distinction between disease and health involves both an objective and a subjective component: first, the state must be found to be biologically abnormal, and second the state must be deemed harmful or undesirable.

While it is seems that most medical practices need not accept the partial-objectivist view, some may need to in order to account for biologically abnormal states or potentially abnormal states that are not deemed diseases by contemporary medicine since they are not taken to be harmful, dangerous, or undesirable. At this point, the proponent of pure-objectivism may respond by pointing out that the judgment that biologically abnormal states are not diseases because they are not harmful or dangerous hinges on a suspect intuition that diseases must be harmful or dangerous. Yes, the proponent admits, it seems odd to call such non-harmful/non-dangerous states diseases but many things in science have seemed strange but upon reflection and time have come to be recognized not only as theoretically reasonable but quite normal and obvious. The germ theory of disease, for instance, was originally thought to be radical and misguided but with

[38] They call it the Hybrid view.

time it has become a core component of medical theory (Richmond, 1954). I am sympathetic with the pure-objectivist's response insofar as it asks us to be careful to not allow our intuitions unnecessary theoretical weight when making judgments about disease and health. But just because we need to be careful about relying too heavily on intuitions does not mean they have no role to play. As already noted, the role of intuitions in this project has to do with judgments about core cases that need to be respected—i.e., intuitions help us determine which cases should count as core cases. In the cases I've previously discussed, it does not seem like intuitions are being given unnecessary theoretical weight since they are merely picking out certain states (e.g., men after vasectomy and women after hysterectomy or tubal ligation) as healthy since the people who enjoy them are not considered to be in a harmful or dangerous state. This reasoning about what counts as health and disease is just what the partial-objectivist account proposes: that our judgments about disease are influenced by our understanding of what counts as a harmful or dangerous state.

In addition to allowing theorists to respect intuitions about certain potentially abnormal biological states, partial-objectivism seems to enjoy widespread acceptance because it lets us maintain a distinction between judgments about biological normalcy and judgments about health and disease. The possibility of maintaining this distinction follows from partial-objectivism's insistence that determining disease status is a two stage process: stage one work involves determining whether or not a part is malfunctioning while stage two work concerns the evaluative judgment as to whether or not the malfunction is harmful, undesirable, etc. (Wakefield, 1992; Murphy, 2006; Ereshefsky, 2009). Being able to maintain the distinction between biological normalcy and health and disease as partial objectivism's two-stage process lets us do is valuable for research purposes since it allows theorists to develop an understanding of how the human body can breakdown or deviate from biological normalcy without simultaneously requiring such breakdowns to be construed as diseases. A further value issuing from the space between

recognition of biological abnormalcy and disease is that it allows us the possibility to treat findings about biological normalcy and abnormalcy as input for a number of different evaluative systems (see Murphy, 2006). Medicine which seems to have a special interest in biologically abnormal states that are judged to be harmful or undesirable may only be one of a number of evaluative systems that take biologically abnormal states as input. The importance of "first stage" work hinges on the fact that it acts as a filter designed to siphon off those cases that should not be offered as input to "stage two", evaluative work (Murphy, 2006). Applied to medicine, this sort of reasoning suggests that stage one work identifies those states that are potential diseases—i.e., the dysfunctional biological parts—from those that are not since diseases are simply biologically abnormal states that are judged to be harmful or undesirable (see Figure 2.2).

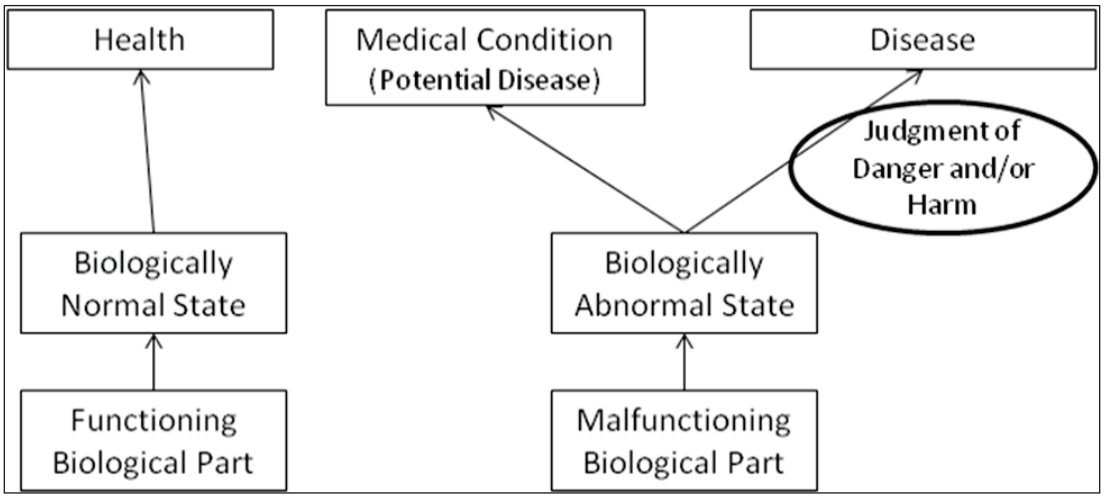

Figure 2.2. Figure representing the connection between biological function, biological normalcy, and the medical condition/disease distinction.

In this section, I have tried to show that even if much of medicine need not be committed to partial-objectivism about disease, there is good reason to think that core medical practices are committed to such a view since it allows them to handle difficult cases and helps them maintain a clear distinction between the research and therapeutic domains. Accordingly, it seems that medicine may be best characterized as a partially-objectivist endeavor, a view that takes medicine (i.e., core medical practices) to be primarily interested in the restoration, maintenance, and

improvement of health where health is understood as freedom from partially-objective diseases and partially objective diseases are understood to be biologically abnormal states that are deemed either harmful or undesirable by the relevant society or community.

Before concluding this section on the partial-objectivist account of disease, I want to say something about the purely objective component of medicine—i.e., the stage one work. "Medical condition" (hereafter, MC) is a term that I use to refer to the purely objective component of disease: the dysfunctional biological part, which is identified as the abnormal biological state, and the cluster of signs that accompanies this dysfunctional part.[39] I introduce the terminology of MCs to emphasis the potential value of these biologically abnormal states in medical theorizing even though they are not recognized as, or deemed to be, diseases. The value of MCs lies in the fact that they are likely to be of interest to medical theorists since they are *potential* disease states. Accordingly, a MC is a potential disease that becomes a disease when it is deemed harmful or undesirable by the relevant (i.e., appropriate) social structure. What it means for a social structure to be 'relevant' or 'appropriate' is an issue that is beyond the scope of this paper. Fortunately, this is an issue I need not settle since the Partial-objectivism that I believe medicine is committed to allows theorists such as myself to remain agnostic about second stage concerns—i.e., about which states are, or should be, considered harmful or undesirable—while allowing us to investigate first stage issues; namely, explore whether certain biological parts are malfunctioning and, thereby, constitute MCs. That the partial-objectivist view allows one to proceed with first stage work independent of second stage concerns is important since my aim in later portions of this project will be to show that we can make sense of psychiatry as a discipline

[39] When it comes to the nature of MCs, two possibilities present themselves: 1) that MCs are an all or nothing affair such that they are either always present or not present in any given individual, and 2) that MCs enjoy ontological vagueness such that there are some individuals such that there is no fact of the matter whether the MC is present. Notice that this second possibility does not hinge upon our inability to know whether a MC is present; rather, it has to do with the possibility that for some MCs, there is simply no fact of the matter whether or not the MC is present. Understood in terms of dysfunction, an ontologically vague MC is one where there is no fact of the matter whether the biological part is malfunctioning or not. Whether or not any MCs actually enjoy ontological vagueness is beside the point since I am merely canvassing possibilities.

focused on mental medical conditions (MMCs)—i.e., malfunctioning parts of the mind-brain—even if we wish to remain agnostic on which of these conditions psychiatry should recognize as harmful or undesirable.

2.5. Further Features of Medicine

So far I have primarily been concerned with clarifying the notion of disease in which medical theorists are likely interested in order to make sense of my claim about the nature of medicine. Now, I'd like to take the time to explore three further features of medicine: 1) the preventative and maintenance role of medicine, 2) the reasoning strategy(s), and difficulties, involved in identifying MCs, and 3) the reasoning strategy and process of diagnosis. For ease of exposition, I will only discuss these three aspects of medicine as they relate to MCs, though one should keep in mind that the following will also be relevant to understanding how medical theorists reason about diseases since all diseases are MCs. The exploration of these components of medicine should help to further clarify its theoretical commitments, a task that will help us to develop and understand the theoretical commitments of psychiatry understood as a branch of medicine in later chapters.

2.5.1. Prevention and Maintenance

One of the central claims of Partial-Objectivism is that medicine is primarily focused on the restoration, maintenance, and improvement of health where health is understood as freedom from partially objective disease. In its attempt to improve and maintain health, medicine is tasked with preventing disease. As a result of this aspect of medicine, it becomes quite important for medical theorists to not just understand MCs but to also appreciate how MCs are acquired and how they develop. Knowledge about the genesis and course of MCs is necessary for medical theorists interested in all aspects of medicine since such knowledge offers a more complete picture of the MC as an object of investigation, a picture that can be exploited for intervention

purposes. After all, if we want to restore someone to health (i.e., a disease free state) we will need to know what the next "step" of the disease (i.e., MC) is in order to know how to best intervene. Alternatively, if we want to prevent a person from acquiring a disease, we will need to know how the harmful or undesirable MC originates and how it is likely to progress in order to have the best chances of preventing the disease from occurring. Taken together, these points highlight the fact that medicine may often busy itself researching how MCs develop and intervening in situations where a disease (i.e., MC) has not yet been realized. Accordingly, theorists may focus on signs and symptoms that indicate a "disease process"—i.e., a process that ultimately leads to a disease state—is underway.[40] The upshot of focusing on these signs and symptoms as opposed to just those that indicate that the disease is already realized is that there will likely be greater opportunities for intervention, at least in many cases. Partial-Objectivism's recognition that medicine is interested in the prevention of disease and the improvement of health is a reminder that the actual practices of medicine, especially as it is embodied in physician-patient interactions, is likely to extend well beyond the range of simply treating people with diseases.

2.5.2. Identifying MCs

That medical theorists are primarily tasked with understanding MCs presses us to address the issue of how theorists identify MCs. After all, in some cases it may be difficult to determine when a part is failing to perform its E-systemic-function since it will not always be easy to tell if a part is no longer contributing to an organism's capacity to survive and reproduce. Just because it will not always be easy to determine this does not mean it will be impossible. Indeed, indications that a biological part is no longer performing its E-systemic-function—and that a MC is present—are often expressed as biological symptoms understood as "abnormal behavior".[41]

[40] Murphy (2009) discusses the actuarial model of disease, a view that highlights the importance of understanding risks factors associated with diseases.

[41] The sort of normalcy that I have in mind here when I talk about "abnormal behavior" is statistical normalcy. Of course, it could turn out that statistical normalcy does not track biological normalcy and *vice versa*. In such cases, we would simply defer to biological normalcy as defined by E-systemic function.

Such behavior can be, and often is, taken as an indication—i.e., *defeasible evidence*—that a part is malfunctioning. In these cases, medical theorists appear to allow subjective judgments concerning behavior to enjoy temporal, but not conceptual priority, in the process of identifying MCs (Murphy, 2009). Of course, medical theorists need not, in principle, employ any subjectively normative considerations in order to determine whether a state constitutes a MC since the identification of a MC hinges on whether or not a biological part is malfunctioning. But just because such considerations are not necessary *in principle* does not mean they will not play a role in the actual practice of identification. As Murphy (2009) considers, the identification process might, for instance, start with a judgment that a person's behavior is abnormal—the subjective judgment— and the assumption that such abnormal behavior is *prima facie* reason to think that there is a malfunctioning biological part (i.e., MC) responsible for the abnormal behavior.[42] Whether or not a MC is actually present ultimately depends on whether or not a biological part is malfunctioning and this is a fact that the medical theorists will need to respect in their identification process. That the status of the state as a MC hinges on the existence of a malfunctioning biological part that is responsible for the subjectively judged "abnormal behavior" is what insures that objective judgments about proper biological functioning enjoy conceptual priority in this identification process.[43]

The importance of identifying breakdowns in biological parts as a means of identifying MCs issues from the fact that abnormal behavior is rarely, if ever, sufficient by itself to confirm

[42] Note that 'abnormal' need not mean harmful or undesirable since we are just discussing how MCs, not diseases, might be identified.

[43] An example may prove useful in explaining the identification process just considered. Let us imagine a medical theorist (i.e., medical practitioner) who sees a patient that is having serious trouble moving around the room. Let us also imagine that the theorist judges that the patient's behavior is abnormal given that properly functioning bodies can normally locomote well. The theorist might take this alleged abnormal behavior as grounds to look for a malfunctioning part that is responsible for the inability to walk—i.e., the abnormal behavior. If the theorist's investigation revealed that the person has a broken a leg, then she would have identified a MC (because the patient has a biological part that is malfunctioning) and that this MC is responsible for the abnormal behavior. If, however, she investigated and found that the patient was merely acting as though they could not walk—i.e., that there was no malfunctioning biological part responsible for the abnormal behavior—then she would not identify the patient's state as a MC.

an E-systemic malfunction. Such behavior is insufficient for two reasons: 1) not all E-systemic malfunctions give rise to obvious abnormal behavior, and 2) not all obvious abnormal behavior is an indication of a malfunctioning biological part. Regarding 1, it isn't always the case that a MC will give rise to symptoms (i.e., behavior) that indicate that the relevant part is no longer making its normal contribution to the organism's survival or ability to reproduce. And in these cases, we simply may not recognize that a MC is present. But this is sometimes the case with medicine—we do not realize that a MC is present until symptoms indicating a threat to the organism's survival arise that alert us to this fact. Regarding 2, the abnormal behavior, understood as statistically abnormal, which is often taken as *defeasible evidence* of a MC (i.e., a malfunctioning part) need not track biologically abnormal behavior—i.e., there may be instances where behavior that is statistically abnormal is, nonetheless, biologically normal in the sense that it is not caused by a malfunctioning biological part.[44] Furthermore, even when biologically abnormal behavior does track statistically abnormal behavior (i.e., when behavior that is statistically abnormal is usually caused by a malfunctioning biological part), the abnormal behavior need not *necessarily* result from a malfunctioning biological part since it could result from a simple performance error. For our purposes, performance errors, by definition, do not result from malfunctioning parts. An example may help to clarify this distinction. A person may appear unable to see and we may assume that this "abnormal behavior" results from a malfunction in the person's visual system only to later learn that the person did not understand English and, therefore, could not appreciate the prompting commands to move. In this case, the failure to behave normally was not the result of a malfunctioning biological part; rather, it was the result of a performance error.[45] If there were no way to distinguish between "abnormal behavior" resulting from a performance error and

[44] Having an odd gait may be considered statistically abnormal behavior but it need not be biologically abnormal behavior; rather, it may simply be an alternate way of walking that is within the normal (biologically normal) range of human behavior.

[45] This is a performance error that results from a competence error since the person was not competent in the appropriate medium of communication.

"abnormal behavior" resulting from a malfunctioning part, medical theorists would be in trouble. Luckily, there is a way to determine which is the culprit, a performance error or a malfunctioning part. The process to do this involves employing the information supplied by functional biologists about the mechanistic nature of the biological part assumed to be responsible for the abnormal behavior in order to determine whether the part enjoys a mechanistic breakdown (Murphy, 2006; Craver, 2007). If the part does enjoy such a breakdown, then this is confirmation that the "abnormal behavior" results from a malfunctioning biological part, not a performance error. The upshot is that the mechanistic understanding of biological parts becomes a central tool to identify malfunctions since the actual identification of a malfunction hinges on showing that the responsible part is a mechanistically broken-down part.[46]

There are likely to be some cases where it proves difficult, if not impossible, to determine whether abnormal behavior results from a malfunction or a performance error. Two cases come to mind: 1) when a biological part's E-systemic function is not yet known, and 2) when a known E-systemic function (i.e., a function that currently accepted evolutionary theory suggests is necessary for individual survival and reproduction) cannot be localized and/or mechanistically decomposed (Murphy, 2006). In the former case, it will be impossible to know when the part is malfunctioning since there is no appreciation of the part's normal (i.e., E-systemic) function. In the latter case, it will be impossible to determine if the lack of the proposed E-systemic function results from a malfunctioning part or a simple performance error since such a determination requires us to know whether the relevant part is malfunctioning or not and we can only know this if we can localize and mechanistically decompose the function in a biological part. This is not to say that all such cases are irreversible dead ends, only that solving these difficult cases will require a more developed appreciation of the relevant functions and parts. With time, researchers

[46]The point to appreciate is that understanding the function of a part is only one piece of the task at hand for we will also need to understand the mechanism that gives rise to this function in order to distinguish between those cases in which the failure to function results from a malfunctioning biological part (and, therefore constitutes a MC) and those cases in which the failure is the result of a performance error.

will often be able to localize and mechanistically decompose known functions in specific biological parts.

In addition to the above points about how theorists identify MCs, it also important to point out that medical theorists are likely willing to hypothesize that a MC is responsible for some suite of "abnormal behavior" even if they have not yet identified the relevant malfunctioning biological part. In these cases, the claim, or judgment, that there is a MC present is more akin to a theoretical promissory note that there is, in fact, a biological part that is malfunctioning. Future findings that there is no malfunctioning biological part should lead theorists to reassess their claim that a MC is responsible for the state—i.e., their diagnosis that the person enjoys a MC.[47] The process of relying on a subjective judgment that some state is odd or abnormal as grounds to posit a MC seems reasonable as long as theorists 1) make clear that the MC is merely hypothesized, and 2) make clear that the actual identification of the state as a MC depends on identifying the malfunctioning biological part. The objectivity of MCs is maintained in this process since the final arbiter of MC status is whether or not there is a malfunctioning part, where forward-looking evolutionary theory determines what counts as functioning and the part is located within the body.[48]

The above points are meant to illustrate how medical theorists likely reason about and identify MCs. And, while it may sometimes be difficult to identify MCs, it is certainly not impossible in many cases, especially when we have a developed appreciation of the biological part understood as a mechanism. With these remarks out of the way, I now turn my attention to diagnosis.

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[47] In many cases, there is likely to be a considerable time lag between the positing of a MC and the time when the relevant malfunctioning biological part is identified or found to not exist. This lapse in time could result from difficulties in locating the dysfunctional part because of a poor understanding of the system or because evolutionary theory has not developed to the point that we understand whether or not the proposed dysfunction is disadvantageous. Because there could be such a long time lag, it will be important for theorists to make clear when a MC is merely hypothesized as opposed to already localized, and, therefore, discovered.

[48] A malfunctioning part also appears to be the final arbiter in Murphy's (2006) account.

2.5.3. Diagnosis

Unlike the problem of researchers attempting to identify malfunctioning biological parts in order to determine whether or not a MC is present, diagnosis has to do with "clinical expert's opinions as to whether some disorder [or disease or MC] is present in a particular patient." (Kraemer, S8, 2007) Accordingly, diagnosis is not concerned with the nature of MCs as it assumes, in principle, their reality.[49] Diagnostic criteria are the signs and symptoms associated with a MC that clinicians take to be indicative of the presence of the MC. Epistemological questions arise for the practice of diagnosis and the use of diagnostic criteria since we are confronted with the question of how we know when a MC or disease is present in an individual (Kraemer, 2007). These questions often reduce to further questions about the reliability and validity of diagnostic criteria (Kramer et al., 2004).

Theorists interested in diagnosis often focus on reliability and validity as two measures of the value of a diagnostic criterion/criteria. The reliability of the criterion/criteria is measured by how likely the same criteria are to deliver an identical independent diagnosis on the same patient. To measure this, theorists often employ the test-retest method which involves making a diagnosis using the given criteria (test) and then employing the same criteria once again as a means of diagnosing (the retest) (Kraemer, 2007; Kraemer et al., 2004). When the retest delivers the same diagnosis as the test, the criteria are deemed reliable.[50] The validity of diagnostic criterion/criteria has to do with whether the criterion/criteria pick out all and only instances of the relevant diagnosis. If the criteria only diagnoses those people that have MC X as having it and diagnoses all those that don't have MC X as not having it, then the criteria are taken to be valid.

[49] Diagnosis may concern the output of stage one or stage two since we may wish to be able to determine—i.e., diagnosis—when a MC is present even if this condition is not deemed harmful or undesirable (i.e., even if it is not taken to be a disease) (Murphy, 2006).

[50] This is sometimes referred to as inter-rater reliability. It scores whether different diagnosticians would give the same diagnosis if employing the same diagnostic criteria.

Perfectly valid criteria are notoriously hard to find for most MCs. This could follow from the ontologically vague nature of MCs—i.e., there could be genuine borderline cases of the MC that are not the result of epistemic shortcomings—but in most cases it probably follows from the difficulty of determining when a part is malfunctioning. In these cases, the vagueness of the diagnosis is epistemic in nature—our inability to diagnose a MC for certain does not issue from the ontologically vague nature of the MC but from our inability to determine whether the MC is present—i.e., the failure to diagnose is our "epistemic" fault.

It should go without saying that understanding the nature of individual MCs is likely to be relevant to diagnosing those MCs. After all, getting a clearer understanding of a MC—e.g., its potential causes (i.e., etiology), its course, how it affects other biological parts of the organism, etc.—will likely help us appreciate the signs and symptoms that accompany the dysfunctional biological part (i.e., MC). And these signs and symptoms can be—indeed, they often are—employed as diagnostic criteria since they have diagnostic value: they indicate—to some degree—that the biological part is malfunctioning (Kraemer et al., 2004). Determining how reliable and/or valid a given sign/symptom is of a certain MC is likely to become clearer as more research is done. Fever by itself, for example, is neither a reliable, nor a valid diagnostic criterion of the flu. Presence of the flu virus in one's body, on the other hand, is both a highly reliable and valid diagnostic criterion of the flu. But, we only came to appreciate that presence of the flu virus is a highly reliable and valid indicator of the flu when biological theory had advanced to the point that we could 1) identify the flu virus in the body, and 2) had theory that proposed invasive organisms as necessary etiological factors of certain MCs.

The "gold standard" diagnostic criterion is the malfunctioning biological part itself because it is a criterion that is absolutely reliable and valid since it is identified as the MC. Unfortunately, the dysfunctional part cannot be relied upon as a diagnostic criterion in all situations since:

1) it may sometimes be difficult to appreciate when a part is actually malfunctioning and this ignorance may issue from either a) the ontological vagueness of the MC or b) our incomplete knowledge of the nature of the biological part;

2) given that medicine is tasked with preventing diseases and maintaining health, theorists will often want to be able to diagnosis when a process is underway that will lead to a MC and in these cases, the signs and symptoms that indicate a process is underway that will eventually lead to a malfunctioning biological part serve as ideal diagnostic criteria, especially given that there is no malfunctioning part that could be called upon for diagnostic purposes.

A third instance when the malfunctioning part may not constitute a practical diagnostic criterion is when the costs to identify the malfunctioning part are prohibitive (e.g., fMRI, PET, etc.). Accordingly, though the dysfunctional biological part is the ideal diagnostic criterion, there are likely to be some cases where we cannot rely on this as our diagnostic criterion. Therefore, we would do well to have other diagnostic criteria at hand, even if they prove less reliable and valid than the malfunctioning biological part. In any case, the point to appreciate is that diagnosis will probably improve as stage one work improves since we are likely to better understand the accompanying sign/symptom profiles that can be understood as indicators of the malfunctioning part, or the process that is leading towards the part's functional breakdown, as we gain a better understanding of the nature of these MCs.[51]

Given our epistemic constraints and the possibility of genuine ontological vagueness, it is worth considering the possibility of dimensional and categorical approaches to diagnosis. As Kraemer et al. note, the question of category vs. dimension (or spectrum) is primarily a question about diagnosis since all MCs are both categorical and dimensional in the sense that "[the MC or disease] is either present or not (categorical), but when the disorder [or MC or disease] is present, patients may vary with respect to age-of-onset, severity, symptomology, impairment, resistance to

[51] It is worth noting that the "art" aspect of medicine may have to do with the ability of medical practitioners to diagnose diseases. Indeed, this "art" may be something that is more or less improved by clinical expertise. Of course, whether seasoned physicians are better at diagnosis because they have better "implicit" methods of diagnosing or because they simply are more familiar with diagnostic criteria remains unclear. For more on this, see Thornton (2006).

treatment and a variety of other disorder characteristics (dimensional)." (18, 2004) When it comes to diagnosis, we can understand the distinction as follows:

> "A categorical approach to a diagnosis results in labeling each subject as either having (D+) or not having (D_) a disorder [or MC]…. A dimensional approach results in labeling each subject with an ordinal score (D), with higher scores a stronger indicator of the presence of the disorder." (Kraemer et al., 18, 2004)[52]

Following the suggestions of Kraemer et al. (2004) and Kraemer (2007), I think diagnosis ought to be two-pronged: it ought to have a categorical component and a dimensional component. The upshot of this would be diagnostic criteria that track dimensionality and category. Such a strategy could take the category component to mark the distinction between clear diagnosis of having MC X and not-having MC X—i.e., one is diagnosed as having MC X when one clearly has the malfunctioning part associated with MC X—and the dimensional component to track the range of states between a diagnosis of having MC X and a diagnosis of not-having MC X.[53] The range that gets reflected in the dimensional component, or along the spectrum, may reflect our inability to diagnose with certainty when a MC is present since we may not know that the relevant part is actually malfunctioning or it may reflect the ontological vagueness of the MC. It may also be useful to have a dimensional component for MCs that can be diagnosed with certainty since physicians may wish to intervene on the processes that lead to MCs before the MC itself has been actualized. Such pre-emptive intervention would seem to require a dimensional component that tracks when a breakdown in a certain biological part is underway and how close to completion—i.e., actual breakdown—the process is. How such a dimensional component is determined and

[52] Kraemer et al. (2004) seem to employ two notions of dimensionality. The first notion seems to see dimensionality as being related to variants of the disease that are related to age of onset, severity, etc., while the second notion seems to see dimensionality as indicating degree of presence of the disease (i.e., closer to, or further from, being clearly identified as having, or not having, the disease). I am more interested in the second notion of dimensionality since it is better suited to track the range between the clear cases and having a score that locates someone in this range will likely prove useful for intervention purposes. A final worry about Kraemer et al.'s (2004) work is that the categorical approach may not always work since there could be cases of genuine ontological vagueness. Of course, in these cases, we can simply impose an arbitrary cutoff to make the categorical distinction clear.

[53] Knowledge about the mechanistic nature of MCs would function as a sort of epistemic anchor to tie our diagnosis to insofar as it would allow us to develop both the categorical and the dimensional scales.

scored, as well as where on the dimensional spectrum theorists decide intervention is called for, is an issue that is beyond the scope of this paper. I am merely discussing the possibility of a categorical-dimensional approach to diagnosis as a potential strategy to deal with the many problems that are likely to arise in diagnosis owing to the epistemic constraints of theorists and physicians and the potential ontological vagueness of some MCs. [54] Whether such an approach is actually well-suited for diagnostic purposes remains to be seen.

Up to now, I have mainly discussed diagnosis and MCs. I have done this because I wanted to address the epistemological issues concerning diagnosis separately from the epistemological issues that may arise concerning the status of MCs as "harmful" or "undesirable" conditions. My aim was to make sure that the issues with diagnosis were presented as clearly as possible. Now, however, I want to quickly note how diagnosis is related to diseases. The most obvious connection has to do with the fact that diagnosis will be most relevant in those cases where we are dealing with diseases since these are the sorts of MCs that most clinicians and medical practitioners will be interested in identifying (i.e., diagnosing) in individuals. After all, these are the MCs that have been deemed harmful or undesirable and are, therefore, likely to be sought out since diagnosis is a necessary prerequisite to treatment interventions. That diagnosis is mainly centered on diseases is evidenced by the fact that most medical diagnostic resources—e.g., the ICD 10, the DSM IV—tend to focus on MCs that are already deemed harmful or undesirable. Regardless of the fact that medical theorists may be primarily interested in the ability to diagnose diseases, they are likely to remain interested in identifying and better understanding all MCs given the fact that all MCs are potential diseases. With these remarks on the further features of medicine completed, I will now conclude this chapter with a brief summary

[54] It is interesting to note that Kraemer et al. recognize a "fundamental equivalence" of these approaches since any dimensional approach can be assigned an arbitrary cut-off for category's sake and *vice versa* (Kraemer et al, 2004).

of the theoretical commitments of the Partial-Objectivist view that I believe properly characterizes medicine.

2.6. Conclusion and summary

In this chapter I have argued that medicine is properly characterized as a partial-objectivist endeavor. I have suggested that these practices are best characterized as being interested in the restoration, maintenance, and improvement of health where health is understood as freedom from partially-objective disease and partially-objective diseases are understood as medical conditions that are deemed harmful or undesirable. I also noted that medicine is probably not a monolithic practice and, therefore, no single account of medicine—aside from the very abstract and vague "health as 'well-being' account"—is likely to properly characterize all medical practice. Nonetheless, much medical practice does seem to be properly characterized as partially-objective in the manner that I have suggested. I have argued that the objectivist component of medicine is found in the role that biological normalcy plays in medical theorizing about health and diseases and that the relevant notion of biological normalcy is the one grounded in biological functioning understood as E-systemic functioning. The subjective component is found in the role that evaluative judgments play in determining which medical conditions actually constitute diseases.

It is also important to reiterate that the Partial-Objectivist view of medicine does not hold that medicine is only interested in treating patients that have acquired dysfunctional biological parts (i.e., diseases). After all, it will often be the case that medical interventions will be called for in order to insure that a patient does not acquire a disease.[55] In order to determine if a patient is headed towards a disease state (i.e., acquiring a dysfunctional part), however, it will be

[55] Such interventions fall within the purview of medical practices that are interested in the prevention and maintenance of health.

necessary to know what it is for the part to be dysfunctional. This is the sense in which medicine is primarily interested in health understood as freedom from disease: it is in terms of diseases that medical theorists develop a sense of how biological parts can malfunction or be abnormal and knowing this is just as important for the preventative and maintenance practices of medicine as it for restorative practices. Accordingly, there need not be an actual dysfunctional part in order for medical intervention to be called for; rather, there only needs to be an indication that one is headed towards acquiring a dysfunctional part. Signs indicating movement towards a dysfunctional biological part (i.e., abnormal biological state) are essentially calls to intervention.

The preceding work suggests a number of theoretical commitments and reasoning strategies central to the Partial-Objectivist view of medicine. They are, in no particular order:

- Medicine as a whole is globally unified insofar as it is interested in the restoration, maintenance, and improvement of health, where health is understood as "well-being". This claim is weak on content since the relevant notion of well-being is vague and uninformative.
- Medicine aims to be scientifically grounded, namely, it aims to be grounded in functional biology.
- Medicine—i.e., core medical practices—is best characterized as a Partial-Objectivist endeavor since it appears to be primarily interested in health understood as freedom from disease where diseases are understood as biologically abnormal states deemed to be harmful or undesirable.
- Biologically abnormal states are states that involve an E-systemically malfunctioning biological part.
- An E-systemically malfunctioning part is a part that fails to make its normal contribution to the evolutionary fitness of an organism—i.e., an organism's ability to survive and reproduce.
- Medicine is focused on individual evolutionary fitness.
- Medical condition (MC) is the term used to refer to the purely objective component of diseases—i.e., the biologically abnormal state.
- Diseases are MCs that are deemed harmful or undesirable by the relevant/appropriate group, society, or community.
- Medicine has both a research and therapeutic/intervention component.

- The research component is focused on understanding the origin, course and nature of MCs.
- The therapeutic component tends to focus on diseases.

With these final remarks out of the way, I will now proceed to the next chapter where I say more about the sorts of phenomena (i.e., medical conditions) that are likely to be of theoretical interest to medicine.

Chapter 3: Medical Conditions & Medical Kinds

3.1. Introduction

This chapter adds to my characterization of Partial-Objectivism by giving a more developed account of the objects of interest to medicine, medical conditions (MCs). The general idea motivating the chapter is an assumption on the part of Partial-Objectivism that medicine is primarily interested in classes of objects that are scientifically interesting and useful—i.e., classes that support a certain type of generalization, enjoy causal unity, and figure in scientific explanations[56]—since it aims to be scientifically grounded.[57] I term such classes 'scientific kinds'. Accordingly, Partial-Objectivism is committed to the idea that MCs should constitute scientific kinds. Understanding what this claim amounts to and what it tells us about MCs requires getting clear on the nature of scientific kinds. For this reason, I spend considerable time developing the notion of scientific kinds. Once this has been done, I turn my attention to clarifying and explaining some unique features of the scientific kinds of medicine, medical kinds (hereafter, MKs).

In addition to providing a more nuanced account of MCs, this chapter proposes the theoretical guidelines for developing a classification system, or taxonomy, of MKs. It is a common idea among philosophers that good scientific classification systems are those which include classes that are scientifically interesting and useful—i.e., classes that constitute scientific kinds.[58] Accordingly, any discipline that aims to be scientific and is engaged in the practice of

[56] What it means for a class to be scientifically interesting and useful is discussed at length in section 3.3.

[57] As a reminder, the claim that medicine aims to be scientifically grounded means that it aims to be informed by science—i.e., biological theory—a claim evidenced by medicine's commitment to, at the least, a partial-objectivist notion of disease. By making this claim I am not suggesting that non-scientific features are completely absent from the domain of medicine. After all, components of the therapeutic aspect of medicine such as diagnosis and treatment are probably greatly influenced by tacit knowledge, patient values, etc.

[58] See Khalidi, 1993; Boyd, 1991, 1999, 2010; Collier, 1996; Kornblith, 1993; Lange, 2006; Nickel, 2010; Bird & Tobin, 2012.

classifying objects ought to be interested in identifying, and constructing taxonomies of, scientific kinds. Given its aim to be scientifically grounded and its interest in nosology (i.e., disease classification), medicine seems committed to the practice of focusing on identifying conditions that constitute MKs and restricting the classes that figure in its taxonomy(s) to these sorts of kinds.[59]

The chapter, which consists of two parts, proceeds as follows. The first part deals with scientific kinds in general. It begins (section 3.2) with a discussion of the pragmatic methodology for understanding scientific phenomena developed by Sandra Mitchell (1997, 2000). I employ this methodology in order to develop an account of scientific kinds (section 3.3). Employing the pragmatic methodology entails that I develop my account by looking to scientific practice as a means to determine what sort of classes are actually scientifically interesting and useful. The upshot of this investigation is the proposal that scientists are particularly interested in classes that support certain types of generalizations (namely, P-laws) and that figure in scientific explanations. I then suggest that these classes are able to play these roles because they enjoy causal unity. I term the classes that possess these three features—supporting P-laws, figuring in scientific explanations, and enjoying causal unity—P-scientific kinds (pragmatic scientific kinds) to highlight the fact that they are principally picked out by the role they play in scientific practice. I then go on to consider how P-scientific kinds may be related to natural kinds (section 3.4). I note that P-scientific kinds are theoretically very similar to the HPC (homeostatic property cluster) kinds proposed by Richard Boyd. The P-scientific kind account outperforms the HPC kind account, I argue, because it offers a more nuanced understanding of the ways that scientific kinds vary and it does not employ the metaphysically loaded terminology of natural kinds.

[59] That medicine does aim to be grounded in science and is keen to develop a nosology goes all the way back to ancient Greek and Indian medicine. Modern proponents of this view include Francesco Redi (1626-1697), Thomas Sydenham (1624-1689), Robert Hooke (1635-1703), Xavier Bichat (1771-1802), Karl Ludwig (1816-1895), Rudolf Virchow (1821-1902), Claude Bernard (1813-1878), Ignaz Semmelweiss (1818-1865), Carl Mayrhofer (1837-1882), Louis Pasteur (1822-1895), Robert Koch (1843-1910), and Friedrich Loeffler (1852-1915).

In the second part of the chapter, I turn my attention to the P-scientific kinds of interest to medicine, medical kinds (MKs). I argue that the classes that constitute MCs also constitute MKs (section 3.5.1). This leads me to identify MCs as MKs. Noting that there may be special, scientifically interesting features of MKs, I consider the role etiological and pathophysiological factors play in typing (section 3.5.2). After suggesting it is useful to type MKs by both pathophysiology and etiology, I consider how this is relevant to classification. The upshot of these considerations is that a medical taxonomy is likely to be comprised of many nested MKs where a nested MK is one that contains several etiology-specific sub-classes that also constitute MKs. I present hepatitis as a case study to illustrate the roles etiological and pathophsyiological factors play in typing MKs and how these factors are relevant to developing a medical taxonomy. The chapter concludes with a consideration of the possibility of non-MKs that are of interest and usefulness to medical researchers (section 3.6).

3.2 The Pragmatic Methodology

Sandra Mitchell's work on the pragmatic approach to laws of nature provides the methodological basis for my account of scientific kinds (1997, 2000, 2002, 2003). Mitchell's work in this area is an application of a general pragmatic approach that can be employed as a means to understand scientific phenomena—e.g., models, explanations, classes of interest, etc. The general principle behind her approach is that philosophical attempts to understand scientific phenomena ought to be informed by how these phenomena are actually used by scientists.[60] She suggests that we ought to be wary of philosophical strategies that offer accounts of scientific phenomena that do not respect how scientists actually think about, reason about, or employ these phenomena (e.g., "normative accounts"). The problem with "normative accounts", Mitchell notes, is that they are liable to cover over and obfuscate important aspects of the phenomena they

[60] Marc Lange (2000) employs a similar approach to understanding laws in that he places much emphasis upon the role that laws of nature play in the various sciences.

aim to illuminate since they do not pay attention to how these phenomena are actually employed by scientists.[61] By placing emphasis on the role phenomena play in science, the pragmatic approach binds the value of an account of a given phenomenon with how well it helps us understand actual scientific practice.

Given Partial-Objectivism's commitment to methodological naturalism, I am interested in accounts of scientific phenomena that are consistent with, and informed by, scientific practice. For this reason, I am sympathetic to Mitchell's pragmatic methodology as a means to understand scientific phenomena including, but not limited to, what I am calling scientific kinds. In order to get a clearer understanding of the pragmatic approach and how it differs from normative approaches, we will consider Mitchell's work on laws of nature.

3.2.1 Laws of nature: the two approaches applied

A motivation behind philosophical accounts of laws is to better understand the generalizations about causal patterns that are of interest to scientists. It is commonly recognized by philosophers that scientists are interested in generalizations that can be employed for the purposes of explanation, prediction, and intervention (Mitchell, 2000, 2002; Carroll, 2012). It is also often assumed that the generalizations that are employed by scientists for these purposes constitute laws of nature while those that are not are merely accidental generalizations (Lange, 2000; Mitchell, 2000). An example of an accidental generalization is *all students in the class have 5 dollars in their pocket* when all of the students in the class actually do have 5 dollars in their pocket. An example of a law of nature is that *no object can exceed the speed of light*.[62] Philosophers have spent substantial effort trying to explain why some generalizations constitute laws of nature but others do not. For many, a successful account of laws is one that satisfactorily

[61] The concept of "normative approaches" is due to Sandra Mitchell (1997, 2000).

[62] This is a law that follows from Einstein's Theory of Relativity.

explains this fact. In the following sections, I consider two approaches to understanding laws—the normative approach and the pragmatic approach—that center on offering such an explanation.

3.2.1.a The normative approach to laws

Normative approaches (Dretske, 1977; Armstrong, 1983; Lewis, 1994; Ayer, 1998) to laws take the dichotomy between generalizations that constitute laws of nature and generalizations that are merely accidental to be primitive and in need of explanation (Mitchell, 2000). Given the acceptance of this strict dichotomy, these approaches attempt to account for the distinction by holding that there are certain necessary and sufficient conditions on laws. These conditions are often expressed as a definition of the laws of nature. As Mitchell notes (2000), this definition or set of normative constraints is then used as a means to determine which generalizations from which sciences count as laws. This move is in line with the general strategy of normative approaches: begin with a norm or definition of the relevant scientific phenomena—laws, explanations, etc.—and use this as the "benchmark" for determining when and where the phenomenon occurs (Mitchell, 2000, 244). If the proposed normative constraints take laws of nature to be logically contingent (i.e., having empirical content), physically necessary (being absolutely stable), true, universal generalizations (holding across all space and time), then only those scientific generalizations which have these features will count as laws. If a generalization does not meet these constraints, then it is not a law but an "accidentally true generalization". As a rule of thumb, all normative accounts seem to take the above four features as constitutive of laws (Mitchell, 2000). The value of normative approaches is that they offer rigid criteria that set clear boundary conditions on the type of generalizations that count as laws. If we know that laws must meet the previously mentioned constraints, then we have necessary and sufficient conditions for laws of nature. Such a feature of laws promises ease of identification.

But normative approaches are not without their problems. After all, they seem committed to the existence of a single set of criteria that properly distinguishes the generalizations of interest to scientists from those that are not of interest even though there is no obvious set of criteria that can actually account for this distinction in the requisite manner. That is just to say, a normative approach may impose constraints on laws—i.e., it may impose necessary and sufficient conditions that generalizations must meet if they are to count as laws—but it is unlikely that these constraints will pick out any and all generalizations about causal patterns that are employed by scientists for explanation, prediction, and intervention (Mitchell, 2000; Lange, 2000). A closely related worry is that it is likely to turn out that some generalizations are of interest to scientists but do not meet the stringent conditions the normative approaches place on laws. Given the strict dichotomy proposed by normative approaches, these scientifically useful "non-law" generalizations would be relegated to the accidental generalization category. As Mitchell notes, lumping all "non-law" generalizations into a single category is problematic for it potentially obscures a better understanding of the way in which these generalizations differ and may be of interest to scientists even though they do not meet the proposed norms (2000, 2002). These considerations about the shortcomings of normative accounts of laws point to a deep problem with the normative approach in general: employing it is liable to hinder our understanding of scientific practices since it need not pay attention to how the relevant phenomena are employed by scientists when determining normative constraints, a key ingredient if we hope to understand the role these phenomena play in scientific practice (Mitchell, 2000, 2002).

The proponent of the normative approach may attempt to deflate the worry that their method hinders our understanding of scientific practice by claiming that not all generalizations of interest to scientists need to be considered laws. It is no serious problem then, they may reason, that their approach does not properly differentiate between all scientifically interesting

generalizations and non-interesting generalizations. If this is their response, however, then they will need to say something more about how we distinguish the accidental generalizations that are of interest to scientists—i.e., those accidental generalizations that they count as "non-laws" that are employed by scientists for the purposes of explanation, prediction, and intervention—from those that are not of interest (Mitchell, 2000). If they can do this, then the norm driven distinction between laws of nature and accidental generalizations they propose seems less theoretically interesting since it fails to capture the perhaps more important distinction between generalizations of interest to scientists (i.e., scientifically useful generalizations) and those that are not. If they do not say something more about the scientifically interesting generalizations that they relegate to the accidental generalization category, then they seem subject to the charge of having failed to address the deeper issue of what distinguishes generalizations of interest to scientists from those that are not of interest (Mitchell, 2000).

Again, the proponent of the normative approach may have a response in the form of "biting the bullet" and simply holding that giving an account of laws is enough even if it cannot or does not account for the differing sorts of accidental generalizations—i.e., those that are of interest to scientists vs. those that are not. These theorists are not out of the theoretical woods, however, for most normative accounts of laws are plagued by the even deeper problem that no generalizations meet the constraints that they require of laws. This problem is most obvious in accounts that see the distinction between laws and accidental generalizations hinging on a distinction between these generalizations being either physically necessary or contingent (Mitchell, 2000). Consider the fact that it is now widely accepted that no logically contingent (i.e., having empirical content) generalizations are physically necessary since all causal relations in the world depend on certain prior conditions holding (Cartwright, 1999; Lange, 2000, 2005; Mitchell, 2000, 2002). If we accept the normative constraint on laws concerning physical necessity, then this fact about widespread physical contingency would seem to suggest that there

are no laws of nature and that law talk in the sciences is, at best, metaphorical (Mitchell, 2000, 2002). As a result, all generalizations would seem to collapse into the class of accidental generalizations forcing us to recognize that no generalizations about causal relations constitute laws of nature. And, while we might be willing to accept that this is the case (i.e., that there are no laws of nature because no generalizations enjoy physical necessity), treating all generalizations as simply accidental threatens to obscure useful distinctions that hold between the various generalizations in which scientists seem to be interested (Mitchell, 2002). After all, not all generalizations are as contingent as others and this is a fact that is likely to be of interest to scientists since it may suggest something about the domain that a particular generalization ranges over (Mitchell, 2000, 2002).[63] The problems that besiege normative approaches to laws suggest that alternative approaches may be better performing when it comes to understanding the sorts of generalizations that play a role in scientific practices such as explanation, prediction, and intervention.

3.2.1.b The pragmatic approach to laws

An alternative to the normative approach is the pragmatic approach which proceeds by identifying the role that the relevant phenomena are supposed to play in science and then looks to the actual practices of science in order to better understand the phenomena that actually do play these roles.[64] When it comes to laws, the pragmatic approach starts by noting that laws are generalizations that represent causal patterns that are useful and of interest to scientists. This raises the question, what kind of generalizations are useful and of interest to scientists? The pragmatic approach seeks an answer by looking to the sorts of generalizations that scientists are actually interested in. Its finding—scientists are interested in generalizations that represent causal

[63] This idea is explained in greater detail in the next section when I discuss the connection between stability and causal complexity.

[64] One can also take a paradigmatic approach but this inherits the problems of the normative approach since one will need to start with an idea about which phenomena should count as paradigmatic. See Mitchell (1997).

patterns that are useful for explaining, predicting, and intervening in the world (Mitchell, 2000, 248-249). Furthermore, the approach recognizes that the generalizations—i.e., laws—of interest to the various sciences are likely to differ with respect to stability and strength (Mitchell, 2000, 2002).

That generalizations differ with respect to degree of stability has to do with the fact that generalizations enjoy differing degrees of physical contingency. As already noted, the contingency of a generalization is tied to the nature of the prior conditions that the generalization depends on in order to hold (see Figure 3.1).[65] That no objects can travel faster than the speed of light is dependent on certain prior conditions holding in the earliest moments of our universe's history. If things in these early moments had been different, then some objects might be able to exceed the speed of light. The generalization having to do with gravitational force on Earth depends on the core of the Earth being iron and not lead. If the core were made of lead, the gravitational force on Earth would be roughly four times what it is now. The biological generalization concerning sex ratios in a population is dependent on prior conditions on Earth that lead to this ratio being naturally selected for—i.e., conditions were such that organisms that had this sex ratio were favored to those that did not have this ratio and as a result this ratio has become stable in the population (Fisher, 1958). Gresham's law of economics which has to do with bad money driving out good is dependent on human psychology, a trait that is highly dependent on prior evolutionary conditions. The point to appreciate about all of these examples is that the generalizations could have failed to hold if prior conditions had been different. It is in this sense that all of these generalizations—i.e., laws—are physically contingent. That some rely on a higher number of prior conditions holding suggests that some are "more" contingent than others. Biological laws are generally highly contingent in that they depend on prior conditions being held constant for at least 9 billion years since our Earth is only 4 billion years old and the

[65] Several of the following examples are from Mitchell, 2000.

universe is estimated to be around 13 billion years old (Mitchell, 2002). Many laws of physics are much less contingent insofar as they may only depend on prior conditions being held constant for the universe's first few moments. But they are all contingent and, therefore, less than fully stable.

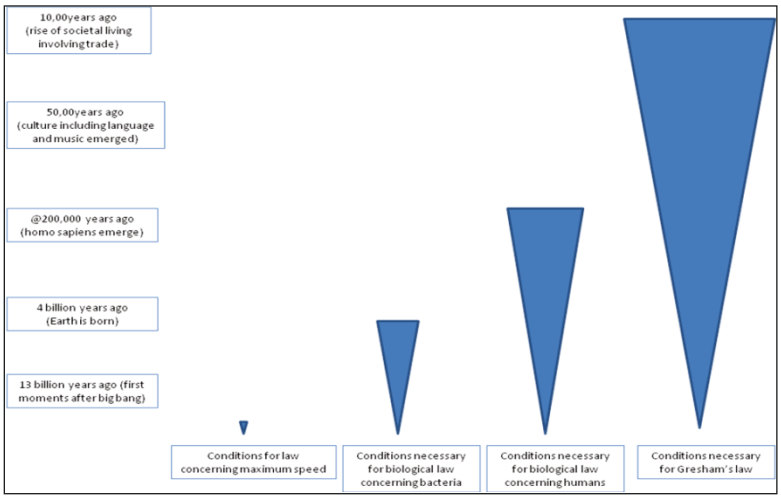

Figure 3.1. Representation of the physical contingency of laws from various sciences. The blue areas represent the "number" of prior conditions that need to hold in order for the generalization to remain true. Greater blue area means the generalizations are more physically contingent—i.e., more prior conditions must hold.

The pragmatic approach also recognizes that laws, understood as the generalizations of interest to scientists, differ with respect to strength (Mitchell, 2000, 2002). Differing degrees of strength refers to the fact that antecedent conditions named in a generalization give rise to consequent conditions with differing degrees of certainty. Consider the following generalization: *all objects on earth are subject to gravitational force X*. This generalization can be spelled out in the conditional: *If object Y is on earth, then object Y is subject to gravitational force X*. This generalization is relatively strong since it holds for all objects on earth—the probability that an object on earth will be subject to gravitational force X is 1. Other generalizations of interest to scientists hold with a lower degree of strength. Many biological generalizations tend to be

probabilistic. Consider the biological generalization *humans have one heart and two kidneys*. We can see that the strength of this generalization is weaker than that concerning gravity by rephrasing it in its conditional form: *If something is a human, then it is highly likely to have a heart and two kidneys*.[66] Now, while it is highly likely that anything that is a human will have a heart and two kidneys, this is not always the case—some humans are born with only one kidney and do not cease to be humans as a result. High likelihood suggests high probability but it doesn't entail absolute strength; accordingly, it would seem reasonable to say that this generalization enjoys strength of less than 1.

Of course, it may be possible to restate any generalization from any scientific discipline in such a way that it enjoys full strength.[67] But the point to appreciate, at least in line with the pragmatic approach, is that doing so wouldn't help us better understand scientific practice for it seems to be a part of the reasoning process of scientists to employ generalizations that have varying degrees of strength (see Mitchell, 2002). To restate the generalizations—i.e., laws—that scientists employ in order to ensure absolute strength is the sort of practice typical of normative approaches. For the pragmatist, however, the need to try to make the data—i.e., the facts about scientific generalizations—fit the theory—i.e., the normative constraint of absolute strength—does not arise since the facts are taken to be theoretically prior to the fulfillment of normative criteria.

[66] It could be argued that the law should be held as embodying an analytic truth about humans which would mean that this conditional holds necessarily. The problem with this move, however, is that science seems to be interested in discovering generalizations, not devising analytic definitions. For this reason, it seems truer to practice to think of the generalization concerning humans and hearts and kidneys as being a generalization that enjoys a certain likelihood of holding true—i.e., less than full strength.

[67] Attempts to do this often involve the addition of a *ceteris paribus* clause stating that "all things (i.e., surrounding conditions) need to be kept equal".

	Physics	Chemistry	Biology	Economics
Stability of generalizations	Highly Stable	Highly Stable	Less stable	Unstable
Strength of generalizations	Strongest	Strong	Moderately strong	Weak
Complexity of causal patterns of interest	Simple	Fairly simple	Relatively complex	Highly complex
Complexity of objects of interest	Simple	Fairly simple	Relatively complex	Highly complex
Dependency on prior conditions	Dependent on very few prior conditions	Dependent on a small number of prior conditions	Dependent on many prior conditions	Dependent on lots of prior conditions

Figure 3.2. Table of the stability and strength of generalizations, and the complexity of causal patterns and objects of interest, for several sciences.

A take home message of the previous sections is that the pragmatic approach recognizes the specific sciences to be interested in generalizations (i.e., laws) that enjoy varying degrees of stability and strength (see Table 3.1). Another take home message, one that is subtler and lurking in the background of Mitchell's work (2000, 2002, 2003), is that the stability and strength of a science's generalizations are probably related to the complexity of the causal patterns of interest to that science and this complexity is, at least partially, a function of the compositional complexity[68] of the objects in which that science is interested. Several examples may help clarify this point. Physics, which traffics in highly stable and strong generalizations, appears to be interested in simple causal patterns in virtue of its interest in compositionally simple objects such as fundamental particles.[69] Biology, which traffics in less stable and weaker generalizations than physics, is interested in fairly complex causal patterns given its focus on objects that tend to be compositionally complex—e.g., cells have many unique parts that work together in an organized manner, organisms have many parts that are made up of many parts that work together, etc.

[68] An object is compositionally complex when it has multiple parts that are organized in a certain manner. Accordingly, a cell is a compositionally complex object; a pile of sand is not, it is a mere aggregate. See Mitchell, 2002; Craver, 2007, esp. Chpt. 5.

[69] Generalizations about the gravitational force exerted on Earth represent the fairly simple pattern of causal interactions between gravitational fields. Note that the complexity of a given set of causes and facts does not translate into ease of understanding. Giving a full explanation of Earth's gravitational force has proven to be an incredibly difficult task even though the sorts of casual interactions involved are probably fairly simple insofar as this phenomenon can largely be explained as a result of interacting gravitational fields.

Economics, with its extremely weak and highly unstable generalizations, is interested in highly complex causal patterns because of its interest in highly complex objects like social structures and humans understood as decision-makers. These examples are meant to illustrate that there is a relationship between the strength and stability of a science's generalizations and the complexity of the causal patterns of interest to that science (i.e., the complexity of the objects of interest). I now want to explore how these elements might be related. I begin by offering a possible explanation of the relationship between stability and complexity and then turn my attention to the relationship between strength and complexity.

A possible explanation for the relationship between stability and complexity is that the necessary prior conditions that determine the stability of the generalizations of interest to a science also determine the complexity of the objects of interest (see figure 3.2).[70] The truth of this claim requires that objects whose existence depends on lots of prior conditions holding tend to be more compositionally complex. That this is often the case seems to be true. The existence of fundamental particles, which are compositionally simple and have been around since the first few moments of the universe, is dependent on very few prior conditions holding. The existence of a more complex object like a neural cell, however, is much more dependent on prior conditions holding—if there were no Earth, then it is possible there would be no neural cells. Finally, the existence of highly complex objects like social structures are dependent on an even greater number of prior conditions holding, in particular, everything up until about 50,000 years ago. If these conditions didn't hold, it is possible that there would be no social structures and that the generalizations of interest to economics would not hold. These three examples provide support for the claim that complexity and stability are related by their mutual dependence on prior conditions (see figure 3.3).

[70] Mitchell (2002, pp. 335, 340, 342-343) hints at this connection.

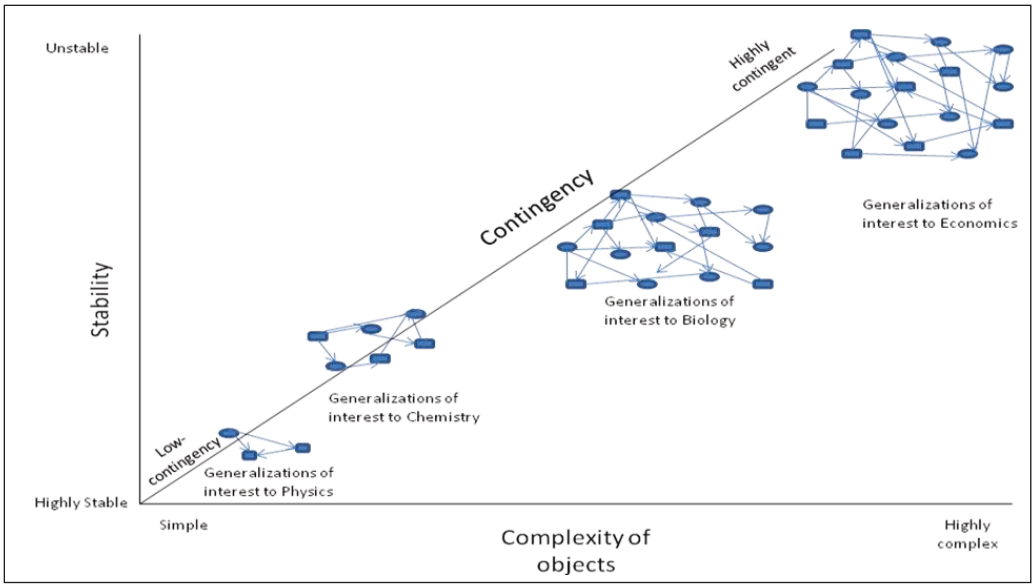

Figure 3.3. Figure representing the relationship between complexity, stability and contingency (i.e., prior conditions). The blue oval and square figures represent causal patterns with more complex patterns being represented by more interacting ovals and squares. The relationship between complexity of objects and causal patterns and prior conditions is positive—dependence on more prior conditions means more complex objects—i.e., more complex causal patterns. The relationship between stability and prior conditions is negative—dependence on more prior conditions means less stable generalizations. The relationship between stability and complexity is also negative—more complex objects means less stable generalizations.

It is worth pointing out that the existence of some complex objects may not depend on many prior conditions holding. Accordingly, the relationship between stability and complexity is likely to break down in some cases. Regardless of this possibility, the relationship appears to hold in many cases suggesting that there is a connection between the causal complexity of a science's domain of interest and the stability enjoyed by that science's generalizations (see Mitchell, 2002).

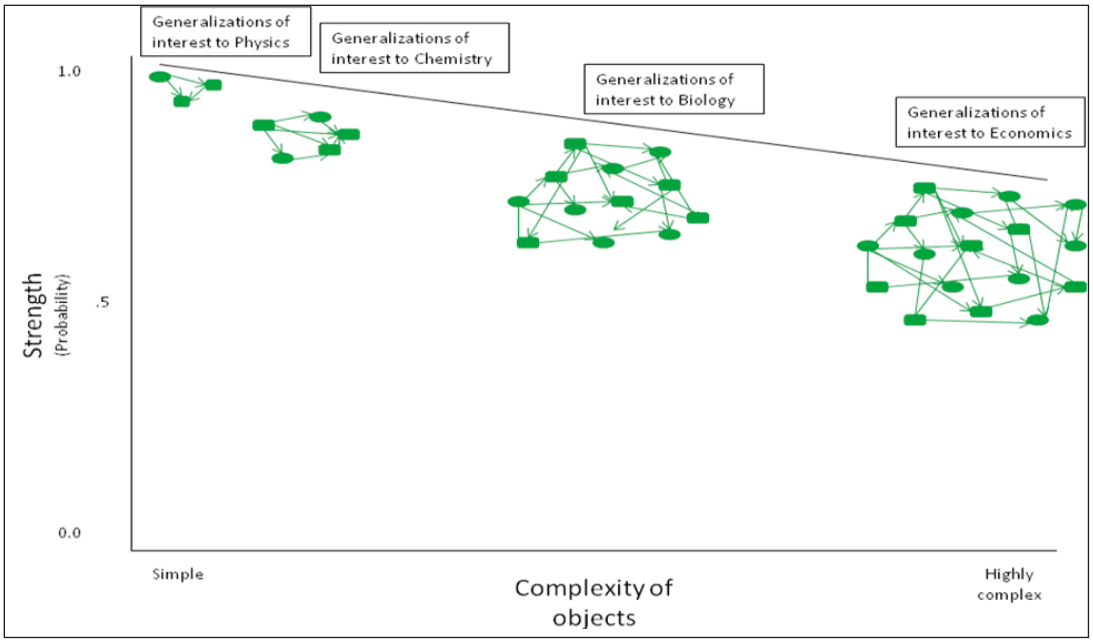

Figure 3.4. Figure representing the relationship between complexity and strength. The green ovals and squares represent components of objects. Accordingly, more interconnected ovals and boxes represent more complex objects. Complexity of objects positively correlates with complexity of causal patterns. The relationship between strength of generalizations and complexity of objects is negative—as the complexity of a science's objects increases, the strength of its generalizations decreases.

The complexity of the objects of interest to a science and the strength of the generalizations in which that science is interested also appear to be connected (see Figure 3.4).[71] To understand this connection, we need to introduce the concept of causal unity. A class enjoys causal unity, or is causally unified, when its members possess a common causal structure. The degree of causal unity a class enjoys depends on how much variation there is in the causal structures of the class's members.[72] The members of highly causally unified classes do not vary much, while the members of weakly causally unified classes tend to vary widely (see Figure 3.5).[73]

[71] The seed of this idea is present in Mitchell (2002).

[72] Variation in causal structure presents itself as variation in properties such that the greater the variation among class members, the greater the difference in the properties they possess.

[73] This is a definitional relation insofar as high causal unity just means little variation and weak causal unity just means much variation.

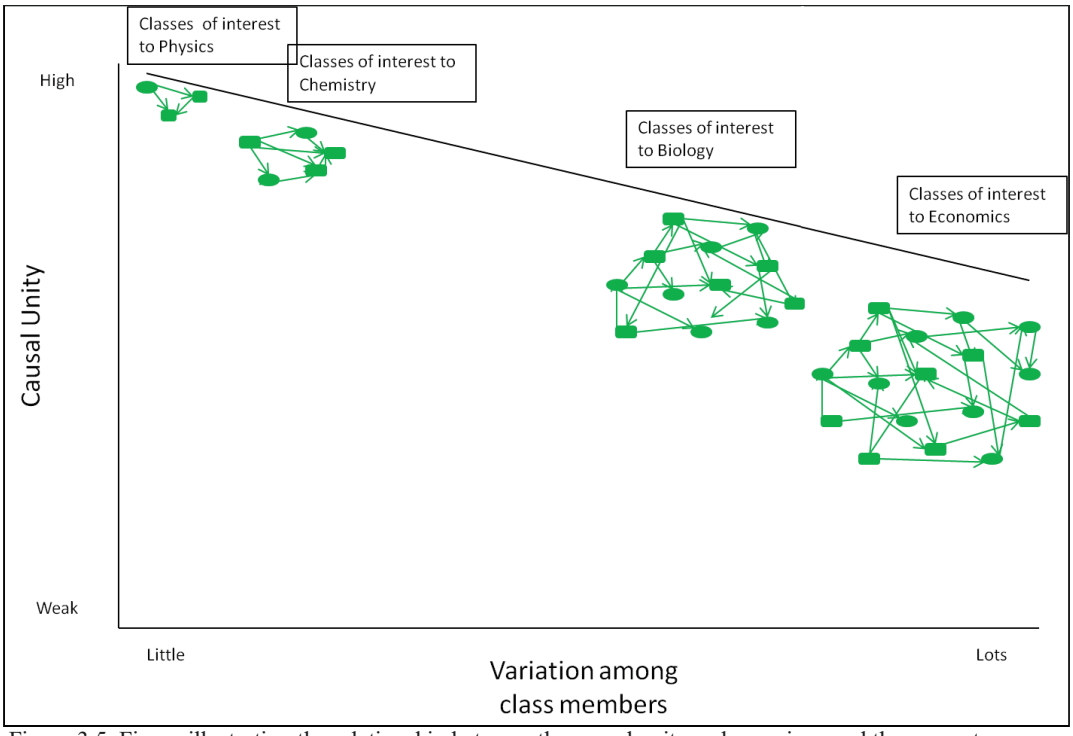

Figure 3.5. Figure illustrating the relationship between the causal unity a class enjoys and the amount of variation in members of the class. The members of weakly causally unified classes tend to vary more—i.e., exhibit different properties—than the members of highly causally unified classes.

How much variation there is in a class—its degree of causal unity—is connected to the complexity of the class (i.e., the complexity of the objects in the class). Complex classes are ones whose members are compositionally complex—they have many interacting parts, while simple classes are ones whose members have very few interacting parts (Mitchell, 2000, 2002, 2003; Craver, 2007). The members of a complex class are more likely to vary than the members of a simple class because there are an increased number of causal interactions taking place between the components of complex objects that can be influenced and lead to variation (Mitchell, 2002). I offer the following points in support of this claim.

1. Complex objects (e.g., humans) are comprised of a large number of interacting parts.

2. The properties that a particular object (e.g., a human) possesses are a function of its interacting parts (and, perhaps, the environment that interacts on these parts).

3. Each point of interaction is an opportunity for things to be different—i.e., for properties to vary.

4. The more interacting parts an object has, the more avenues for variation there are.

5. The more avenues for variation there are, the more likely things will vary.

6. Classes of complex objects are more likely to vary than classes of simple objects.

Point 6, combined with the idea that causal unity is defined by amount of variation, suggests that complexity will be related to causal unity such that more complex classes will tend to enjoy less causal unity (i.e., exhibit more variation), while simple classes will enjoy greater causal unity (see Figure 3.6).

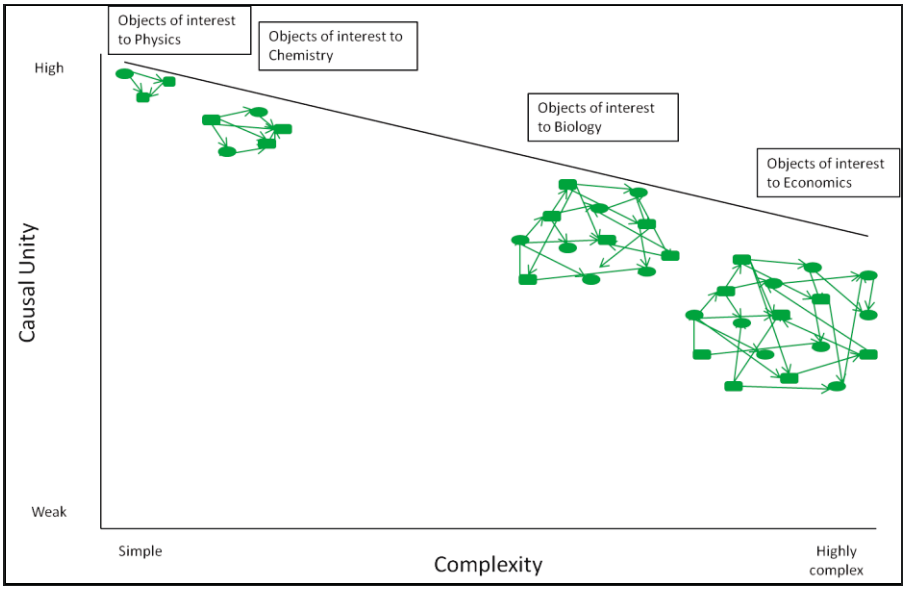

Figure 3.6. Figure illustrating the relationship between the causal unity a class enjoys and the complexity of the class (i.e., the complexity of the objects in the class). Complex classes tend to enjoy more causal unity than simple classes.

Now that we have drawn a connection between complexity and causal unity, we are in a position to explain the connection between complexity and strength.[74] Generalizations about complex objects tend to be less strong than those about simpler objects because complex objects tend to be less causally unified. A class's degree of causal unity is tied to the strength of the generalizations that class supports because generalizations have to do with the projection of

[74] The kernel of this idea is also present in Mitchell (2000, 2002).

properties from a few instances of the class to the class as a whole and the properties of classes with low degrees of causal unity are less likely to fully project (i.e., they will not be found in all class members). The properties of weakly causally unified classes do not fully project because of the variation among members that result from the variation in causal structure. Highly causally unified classes, on the other hand, support generalizations that are relatively strong because the members of these classes tend to share most of their properties.[75] Generalizations concerning *Cebus olivaceus* (weeper capuchin), for example, are weaker than those concerning gold because the members of biological classes are more complex and, therefore, tend to enjoy less causal unity than the members of chemical classes. I consider the connection between causal unity and complexity at greater length is section 3.3.3.

The previous remarks suggest that there is a connection between complexity and strength and that this has to do with the causal unity a class enjoys. I now want to offer several thoughts on why some sciences tolerate less causally unified classes (i.e., more variation in the class and weaker generalizations) than other sciences. The degree of causal unity a science requires has to do with how much variation the science allows among class members before it affects their identity *as* class members—i.e., before it judges a potential class member to not be an actual member of the class (see figure 3.7). This amount is likely to differ among sciences. Sciences interested in complex objects probably allow for less causal unity since they would have no objects to investigate and project over if they required absolute identity among class members (i.e., high causal unity).[76] If biologists were only allowed to investigate classes that are highly causally unified, for example, the domain of biology would be greatly reduced—no investigation of species, organs, etc. Thankfully, biology allows its classes of interest—cell, organs, species,

[75] Why this is the case is not obvious. It could be that simpler objects have fewer properties and because they have fewer properties, each one is more important for identity. Accordingly, classes of simple objects will not tolerate much variation among members because each property is very ontologically significant—i.e., an important part of the object.

[76] This claim follows if you accept the claim in the previous section that classes of complex objects tend to exhibit more variation than classes of simple objects.

ecological niches, etc.—to enjoy low levels of causal unity. This is why the members of the species *Homo sapiens*, very complex objects, can vary along so many dimensions (e.g., eye color, number of limbs, ability to reproduce, etc.) while still maintaining their identity as a member of the species. Sciences such as physics which are interested in less complex objects are likely to require greater causal unity—change a feature of an electron and it ceases to be an electron. That the sciences interested in complex objects require lower degrees of causal unity (i.e., greater variation among class members) helps to explain why they are interested in weaker generalizations—namely, because classes that are less causally unified support weaker generalizations.

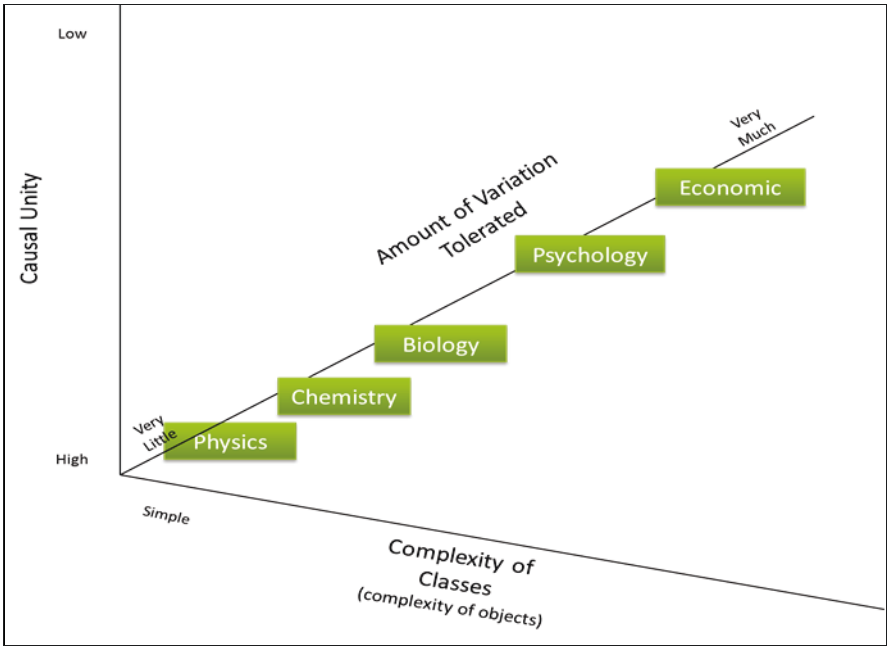

Figure 3.7. Figure representing the relationship between the complexity of objects of interest, variation among class members (i.e., unity), and amount of variation tolerated by sciences.

Recognizing that scientists are interested in generalizations with varying degrees of stability and strength and that the degrees of stability and strength of interest may be related to the complexity of the objects of interest is an important step in understanding actual scientific

practice. By employing the pragmatic approach to laws, we are more likely to recognize this feature of scientific generalizations, and as a result, we are more likely to garner a better understanding of scientific practice—namely, that scientists are interested in domains of inquiry that enjoy varying degrees of causal complexity and that they represent this complexity with generalizations that enjoy varying degrees of stability and strength. As Mitchell (2000, 2002) mentions, if we insist on continuing to employ normative approaches that privilege only the strongest and most stable generalizations, then we are liable to obscure this fact about scientific practice.

Let us recap the pragmatic account of laws. Laws are generalizations of causal patterns that enjoy varying degrees of stability and strength that scientists employ for the purposes of explanation, prediction, and intervention. These generalizations are deserving of the title 'laws' because scientists often consider such generalizations laws and because these generalizations play the role in science that philosophers have often assumed was reserved for laws. These laws of specific sciences tend to enjoy certain degrees of stability and strength and the strength and stability enjoyed seems to be related to the causal patterns of interest (i.e., the complexity of the objects of interest) to the various sciences. Mitchell (2000) has more to say about her pragmatic account of laws. In particular, she is interested in discussing the various ways that the generalizations of causal patterns of interest may be represented. These considerations involve a discussion of the cognitive manageability and abstractness of these representations. While I find these aspects of her approach intriguing, I will not address them here since my interest in her work mainly has to do with the emphasis it places on understanding scientific phenomena in terms of the roles that they play in scientific practice.

Before moving on to a consideration of the pragmatic approach as a general methodology, it is probably worthwhile to respond to a potential worry that might arise concerning my description of the normative approach. In particular, I want to consider the

objection that the normative approach and the pragmatic approach really do not differ very much insofar as alleged normative approaches may actually have derived the norms that they propose for laws of nature from how they believed such laws were actually employed by scientists.[77] If normative approaches take these norms to be derived from scientific practice, goes the objection, then it is hard to see what distinguishes normative and pragmatic approaches. While it may be the case that normative approaches do sometimes take their claims to be informed by scientific practice, it is more difficult to see that they actually are motivated by, and interested in, a proper understanding of scientific practice. That this is the case is evidenced by the fact that some accounts of laws—ones that I take to be normative such as most Regularity Empiricist accounts (Hume, 1777; Ayer, 1998), Best-system Empiricist accounts (Lewis, 1994) and Necessitarian accounts (Dretske, 1977; Armstrong, 1983) —are willing to argue that some generalizations that scientists recognize as laws are not laws. At the limit are those accounts that hold that there are no laws because none of the generalizations of interest to scientists meet the proposed norms, norms that the objection alleges are derived from actual scientific practice (Van Fraassen, 1989; Cartwright, 1999; Giere, 1999).[78] Such an outcome is deeply problematic for the objection since it would require that normative approaches derive their norms from an appreciation of actual scientific practice but that these very same norms show that scientific practice mistakenly employs law terminology.[79] That an approach—i.e., the normative approach—could possibly propose that there are no laws of nature even though scientists regularly talk about and propose such laws suggests that the approach does not derive its understanding of laws from an

[77] I thank Steven Gross for bringing this potential objection to mind.

[78] It is worth noting that Cartwright (and perhaps others) does not appear to think that the norms of laws are derived from actual scientific practice but she does seem to accept that the norms determine what counts as a scientific law. For more on this, see Mitchell, 2000.

[79] I do not mean to suggest that no norms for laws can be discovered which would show that some alleged laws are actually not laws. After all, even scientists can make mistakes. What I am suggesting is that it is deeply problematic if acceptance of a theory of laws compels us to see a large number of well-established and generally accepted laws of nature as not constituting laws.

understanding of scientific practice. The pragmatic approach, on the other hand, takes the actual practices of science to be central to understanding laws of nature and other scientific phenomena. As a result, the pragmatic approach will continue to recognize laws, at least, wherever such terminology is employed by scientists though they may question the existence of laws based on the norms relevant to that domain. This final point which introduces the possibility of deriving norms from within the pragmatic approach will be considered in greater detail in the next section.

3.2.2 The pragmatic methodology

In the previous sections, I spent considerable time discussing various approaches to understanding laws of nature. The motivation for this was two-fold: 1) to illustrate that employing the pragmatic approach can help us garner a better understanding of scientific reasoning and the actual practices of science, and 2) to provide an example of how the approach can be employed to develop accounts of scientific phenomena. I now want to consider how the approach can be thought of as a general pragmatic methodology. This methodology has three steps:

1. Start with a generally recognized platitude about the relevant phenomena (e.g., laws are generalizations of interest and use to scientists);
2. Look to actual scientific practice to see what we can learn about the relevant phenomena (e.g., science is interested in generalizations that represent causal patterns that can be employed for the purpose of explanation, prediction, and intervention; different sciences are interested in different sorts of causal patterns, some causal patterns are deeply complex, others are relatively simple; the stability and strength enjoyed by a generalization is often intimately connected with the complexity of the causal pattern the generalization represents);
3. Take the phenomena to be defined by the role it plays in actual scientific practice (e.g., laws are generalizations that represent causal patterns that are employed for explanation, prediction and intervention).

The pragmatic methodology provides a means to gain a better understanding of how scientific phenomena are actually employed and understood by scientists. As a result, it is likely to outperform normative approaches when it comes to providing an understanding of scientific phenomena that is consistent with scientific practice.

Though the pragmatic approach is not *essentially* normative, it is capable of providing normative criteria on scientific phenomena. This capacity issues from our ability to tease out normative constraints on phenomena from the way that those phenomena are actually employed in scientific practice. When it comes to laws, for instance, it is quite likely that the various sciences will require certain degrees of strength and stability from the generalizations that they employ for explanation, prediction, and intervention. That different sciences may recognize generalizations with differing degrees of strength and stability as interesting and useful suggests a commitment to norms of laws *for that science*.[80] The upshot of this is that what counts as a law for one science may not count as a law for another science since the two may have differing views on the degree of stability and strength required for theoretical usefulness (see Mitchell, 2000, 2002: Lange, 2000). The take home message: the pragmatic approach is capable of delivering normative constraints on scientific phenomena. Unlike normative approaches, however, these constraints are not imposed from the outside and they are not maintained if they do not properly characterize actual scientific practice; rather, they are derived from the actual practices of science and are, therefore, thoroughly consistent with the pragmatic approach.

The methodology of the pragmatic approach is unique in that it allows the actual practices of science to guide the philosophical understanding of science. Its employment leads to a rather deflationary view of many key scientific concepts: laws just are generalizations that represent the causal patterns that scientists employ to explain, predict, and intervene in the world; explanations just are attempts to better understand the world; scientifically interesting and useful classes just are those classes that scientists find interesting and useful. Importantly, the deflationary fallout of the pragmatic approach does not put an end to deeper philosophical investigation into science. For example, when it comes to laws, the pragmatic approach reveals

[80] And, perhaps also, *for time X* since the pragmatic approach may recognize that norms may vary not just by science but by time. Building time into the formula could help us track, and make sense of, changes to the norms that occur at different points in time. The use of indexing the norms to time may also allow us to better appreciate the epistemic context in which a science's norms were proposed and taken to apply.

that the strength and stability of laws vary among the sciences. This finding led us to inquire why this is the case. The answer we uncovered is that the stability and strength a science's laws enjoy appear to be related to the causal complexity of the domain in which that science is interested. As a result, we gained a better understanding of laws—i.e., the generalizations that scientist employ for the purposes of explanation, prediction, and intervention.

Understood as a general methodology, the pragmatic approach is important for theorists interested in understanding scientific phenomena as they are employed by scientists. As such, it is likely to prove well-suited for theorists engaged in projects that are guided by a commitment to methodological naturalism. For this reason, I will be employing this methodology as I attempt to get a better understanding of scientific kinds. It is to an examination of scientific kinds that we now turn.

3.3 Scientific Kinds: the pragmatic approach[81]

As already noted, the goal of this chapter is to give a more developed account of MCs, the sort of entities that Partial-Objectivism holds medical researchers focus on identifying and understanding. Since medicine aims to be scientifically grounded, it stands to reason that it should be primarily interested in, and focused on, classes of objects that are scientifically respectable—i.e., that constitute scientific kinds. Accordingly, MCs, if they are the central focus of medicine, should constitute scientific kinds. Understanding what this claim amounts to and what it tells us about MCs requires saying more about scientific kinds. That is the aim of this section: to apply the pragmatic methodology in order to get a better understanding of scientific kinds. Perhaps unsurprisingly, the upshot of this work will be an account that sees the nature of scientific kinds varying among the sciences. Once I have the pragmatic account of scientific kinds properly formulated, I will then attempt to translate this into the domain of medicine by

[81] I use the terms 'class' and 'kind' interchangeably.
Kitcher (2007) introduces a pragmatist account of natural kinds that is similar in spirit to my account of scientific kinds.

saying more concretely what the scientific kinds of medicine—i.e., MCs—look like (section 3.5). So, let us proceed by applying the three-step method I identified in the last section.

3.3.1 Platitudes concerning scientific kinds

The first step of the pragmatic methodology is to identify a relevant platitude concerning scientific kinds. My proposal is that scientists are especially interested in certain sorts of classes. This is a fact that is widely accepted. After all, most theorists recognize that *ad hoc* categories like 'things weighing less than 2.5 kg' are unlikely to be interesting classes for scientific investigation, while other classes such as 'mammal' 'metal' and 'electron' are taken to be scientifically interesting. Aside from the vague and relatively uninformative idea that scientifically interesting kinds—i.e., scientific kinds—are interesting and useful, there appears to be very little consensus on what differentiates the classes that constitute scientific kinds from those that do not. In this section, I identify some of the distinguishing features of scientific kinds. My strategy for doing this entails identifying the features that such classes possess that make them interesting and useful to scientists. Identifying such features will help us better understand the nature of scientific kinds.

3.3.2 Features of scientific kinds I: P-laws

So what sorts of classes are useful and interesting to scientists? To begin to answer this question, it may be worthwhile to take a few moments to consider the sort of reasoning in which scientists often engage. At a very basic level, scientific reasoning is non-deductive.[82] Its non-deductive nature is evidenced by the fact that scientific claims rarely, if ever, enjoy the certainty that deductive reasoning ensures. This feature of scientific reasoning follows from the ampliative character of the arguments that scientists rely on when drawing inferences: the inference,

[82] We may employ deductive reasoning in science (*modus ponens*) but what will license the truth of the premises (i.e., that P -> Q) will be a non-deductive argument. Hence, the deeply non-deductive nature of scientific reasoning.

understood as a conclusion, contains more than what is given in the premises and, therefore, it does not follow with necessity (Salmon, 1967). This fact of non-deductive reasoning is seen by many to be a shortcoming since it never delivers conclusions with the level of certainty that deductive reasoning allegedly does.[83] Others reckon the value of non-deductive reasoning is to be found in its ampliative nature as this is what allows non-deductive arguments to increase our knowledge in a way that deductive arguments which are merely explicative cannot.

Certain sorts of non-deductive reasoning appear to be particularly important to scientists. Causal inductions which involve an inference from known effects to an unknown cause(s) are a useful form of inductive reasoning employed by scientists (Lipton, 1991). This sort of inference is often counted as inductive since the conclusions concerning the alleged cause do not follow with certainty from the observed effects. Inductive generalizing, the process of making general claims based upon a finite number of particulars, is another form of non-deductive reasoning that is widely employed by scientists (Newton, 1687; Mill, 1843; Salmon, 1967; Norton, 2003; Brigandt, 2010). This type of induction is important for it is what licenses the projection of findings and explanations from a few instances of a class to the class as a whole. For this project, I will focus almost exclusively on the process of inductive generalizing. Given the role of inductive generalizing in scientific reasoning, it seems sensible to suppose that classes that

[83] The problem of induction is centered on Hume's charge that any attempt to show that inductive inferences lead to truth—i.e., that inductive inferences are "truth-tropic"—is unable to succeed (Lipton, 1991). The problem, it seems, is that any attempt to justify the truth-tropic nature of induction will have to rely on an induction and, therefore, be circular since there is no deductive argument that can be given for inductive reasoning (Hume, 1777; Lipton, 1991; Curd & Cover, 1998). While I recognize the importance and the force of the problem of induction, I do not aim to offer a solution. Because this leg of my project is concerned with understanding how scientists reason, and the sort of classes of objects that scientists are interested in for their reasoning practices, it does not seem necessary to engage this problem head on. That scientists engage in inductive reasoning that leads to generalizations is evidenced by 1) the fact that scientific claim(s) are taken to be confirmed or falsified by evidence, 2) the generally recognized fact that scientific inferences are ampliative in nature, and 3) the fact that scientists attempt to develop general claims from the investigation of a few instances.

My decision to not attempt to provide a solution to the problem of induction is not meant to suggest that I think the problem does not deserve to be taken seriously. What I do mean to suggest is that it is not necessary to solve this problem since I am more interested in how scientists reason than whether they are justified in this sort of reasoning. Nonetheless, I should note that I do take the success of science as evidence that the reasoning practices of scientists are more or less justified even though this sort of evidence would surely not satisfy the inductive skeptic.

support inductive generalizations are likely to be useful and, therefore, of interest to scientists. This fact suggests a first blush attempt at identifying a feature of scientific kinds:

Feature I (first-blush): *scientific kinds are classes that support inductive generalizations.*

It is unlikely, however, that scientists are equally interested in all inductive generalizations. Indeed, it seems that certain sorts of inductive generalizations are going to be of particular interest to scientists and that the classes that support *these* generalizations will be the ones that constitute scientific kinds. So what can we say about the generalizations of interest to scientists?

An easy way to answer the question just posed is to rely on Mitchell's pragmatic account of laws. Recall that Mitchell's (1997, 2000, 2002) proposal was that scientists are interested in generalizations that can be employed for the purposes of explanation, prediction, and intervention and that these generalizations are deserving of the title of laws. This move pushed her to hold that laws should be understood as generalizations about causal patterns that enjoy varying degrees of strength and stability and that scientists employ for the purposes of explanation, intervention, and prediction. To avoid confusion, I will refer to laws understood according to Mitchell's pragmatic approach as Pragmatic-laws or P-laws. I propose that we identify the generalizations of interest to scientists with P-laws. The upshot of this move is that we can identify scientific kinds as those kinds that support P-laws—i.e., scientific kinds are those classes that support generalizations that represent causal patterns that can be employed for the purposes of explanation, prediction, and intervention. Our refined description of feature I:

Feature I: *scientific kinds are classes that support P-laws*[84].

[84] Nothing of theoretical weight is meant to hang on the use of the term 'P-law'. If one is uncomfortable with calling such generalizations 'laws', I am happy to rename them 'law-like generalizations'. I have only chosen to use 'P-law' since it helps to illustrate that the account that I am proposing is thoroughly committed to the pragmatic approach to understanding scientific phenomena.

More should be said about the P-laws that scientific kinds support. The first feature of P-laws that should be made explicit is their logically contingent nature. As noted earlier (section 3.2.1), not all inductive generalizations are of interest to scientists. Indeed, there seems to be a particular interest in generalizations which have empirical content, an interest that likely follows from an understanding of science's domain as the empirical world. This aspect of scientifically relevant generalizations is expressed by the widely accepted idea that laws of nature are logically contingent. Because this proposed feature issues from an appreciation of the actual domain of science, it stands to reason that a pragmatic account of laws would recognize logical contingency as a feature of P-laws. Accordingly, we can say that P-laws *are* logically contingent and that the kinds that support such logically contingent generalizations (i.e., scientific kinds) are empirical in nature—i.e., abstract objects like numbers would not count as scientific kinds. Of course, one could question the idea that the domain of science is the empirical world since some sciences may be understood to have a domain that is non-empirical.[85] I confess that I'm not sure whether any such science(s) exists. Nonetheless, if such a science were to exist, I would be happy to amend my claim to the empirical sciences alone. After all, it may be doubted that the domain of science is the empirical world if we take pure mathematics to be a branch of science. But, the importance of empirical content in scientific generalizations and the empirical nature of the classes projected over can hardly be questioned if we confine our discussion to the empirical sciences.

The second feature of P-laws that deserves further mention is that they enjoy varying degrees of stability and strength. For this reason, the pragmatic approach proposed a move beyond the simple law/accident dichotomy to a continuum of degrees of stability and strength (see section 3.2.1.b). In order to understand how we can assess the stability and strength of P-laws, we will need to explore the relationship between P-laws and counterfactual reasoning.

[85] I thank Steven Gross for introducing this possible objection.

Counterfactual reasoning is a process that explores how things would be if the facts were other than how they actually are—i.e., how things would be in counterfactual scenarios. Counterfactual reasoning has traditionally been employed in philosophy of science as a means to distinguish laws of nature from accidental generalizations (Lewis, 1973; Lange, 2000; Broadbent, 2008). The idea is that we can consider whether a generalization would hold true under various counterfactual scenarios with the understanding that the status of the generalization as a law of nature or an accidental generalization depends on the range of counterfactuals that it supports. As others have pointed out, the problem with this sort of reasoning is two-fold (see e.g., Lange, 2000). First, all generalizations, even those that are obviously accidental, support some counterfactuals—e.g., every coin in my pocket would still have been silver even if I wore a different shirt, or different shoes, or forgot to brush my teeth this morning. Second, even generalizations that are taken to be "paradigmatic" laws are unlikely to support the entire range of counterfactuals. If it is the case that no law enjoys physical necessity, then there will be some counterfactual scenario in which every law fails to hold true—e.g., objects might have been able to travel faster than the speed of light, if things had been different during the earliest moments of the universe. Such considerations suggest that counterfactual reasoning may not be helpful if we are looking for a way to distinguish laws of nature from accidental generalizations. This is no problem for the pragmatic approach to laws for it squarely rejects such a distinction and posits in its place a continuum of generalizations of varying degrees of stability and strength. Counterfactual reasoning is useful in helping us to better understand the stability and strength various P-laws enjoy by exposing the range of counterfactuals they support, a range that is intimately connected to the contingency of these generalizations.[86] The P-law *No uranium sphere is a mile in diameter*, for example, supports a wide range of counterfactuals since there are very

[86] Lange (2000) spends considerable time discussing the role that counterfactuals play in understanding laws of nature.

few counterfactual scenarios in which it would not hold true.[87] This is a P-law that appears to enjoy a high degree of stability and strength, a claim evidenced by counterfactual reasoning and an understanding of the relatively small number of prior conditions that this generalization depends on to hold.

The third noteworthy feature of P-laws has to do with the role of employing these laws in scientific practice. After all, many classes support generalizations that are logically contingent and support some counterfactuals but are not employed by scientists for the purposes of explanation, prediction, and intervention. The generalization *All gold spheres are less than a mile in diameter* is both logically contingent and supports some counterfactuals (e.g., it would still have held even if I moved to Texas as a child), but it is not employed by scientists for the purposes of explanation, prediction, and intervention. Because it is not employed by scientists for these purposes, it does not constitute a P-law. If it were so employed, then the pragmatic approach would deem it a P-law.

In conclusion, the three ingredients necessary for a P-law are:

1. The generalization needs to be logically contingent;
2. The generalization needs to support some counterfactuals; and
3. Scientists need to actually employ the generalizations for the purposes of explanation, prediction, and intervention.

Exactly how wide a range of counterfactuals a generalization needs to support to qualify for P-law status is unclear. Furthermore, any attempt to impose a normative constraint on the range required for P-laws in *general* seems contrary to the pragmatic methodology. To stay true to this methodology, I propose that a science taking interest in a generalization should count as *prima*

[87] This example is discussed by both Lange (2000) and Mitchell (2002).

facie evidence that it supports a wide enough range of counterfactuals to qualify as a P-law *for that science*. The upshot of this move, in line with my suggestion from section 3.2.3, is that the various sciences may have differing standards for what counts as a P-law since they may require their P-laws to support a wider range of counterfactuals or enjoy a higher degree of stability and/or strength (Lange, 2000; Mitchell, 2000, 2002, 2003). This feature of P-laws can be used to devise norms for P-laws for a given science since we can take the varying standards on counterfactuals and degree of stability and strength as the norms on P-law-hood *for that science*. That such norms will be science-relative entails that a generalization may count as a P-law for one science given its understanding of the strength and stability required for P-law-hood but not count as a P-law for another science.

This section has identified the first feature of scientific kinds—they support P-laws. Accordingly, species are a scientific kind for biology because they support P-laws about sex-ratios in populations; humans are a scientific kind for biology because they support P-laws about species-typical biological traits; objects with mass are a scientific kind for physics because they support P-laws about gravitational force on Earth; money is a scientific kind for economics because it supports P-laws about good and bad money; etc.. This section has focused on describing P-laws in order to clarify the nature of scientific kinds—namely, that they are classes that support generalizations of a certain type, P-laws. If scientists are interested in uncovering P-laws, as I have suggested they are, then it is important that they focus on identifying and understanding classes that can actually satisfy this aim. It is my claim that scientific kinds are the sort of classes that do, indeed, satisfy this aim. These remarks about the relation between scientific kinds and P-laws, however, raise the further question, what is it about scientific kinds that allows them to support P-laws? It is to this question that we now turn.

3.3.3 features of scientific kinds II: causal unity

Why do scientific kinds support P-laws? The ability of some classes to support P-laws seems to issue from the causal unity these classes enjoy. As noted previously, a class enjoys causal unity, or is causally unified, when its members possess a common causal structure (see section 3.2.1.b). Because the members of these classes possess a common causal structure they are likely to exhibit similar properties—i.e., a shared property cluster (Boyd, 1991, 1999). Classes are causally unified—i.e., they enjoy a common causal structure and an associated property cluster—because of a common causal mechanism. The common causal mechanism could be singular or it could be a nexus of properties that induce each other to co-occur (see figure 3.8).[88] In the first case, the common causal mechanism is the single causal factor that gives rise to the other properties. In the second case, the mechanism is the causal nexus created by the interacting properties (see Boyd, 1991, 1999; Kornblith, 1993; Craver, 2009; Nickel, 2010). In both cases there is causal unity resulting from a common causal mechanism that sustains a property cluster. Causal unity insures that the members of a class share, at the least, some properties in common.

Figure 3.8. Figure representing two types of causal mechanisms and how they sustain property clusters.

How many properties any given causally unified class possesses probably depends on the class's shared causal structure. The members of the classes of interest to physics (e.g., fundamental particles) may share very few properties in common in virtue of the relatively simple

[88] There could be other types of causal mechanisms that can play the role of causally unifying classes. I leave it to others to speculate on what these might be.

nature of their shared causal structure (i.e., they are presumed to be structure-less), while members of the classes of interest to biology (e.g., species) may share a comparably larger number of properties in virtue of the complex nature of their common causal structure (biological organisms are relatively complex causal structures involving billions, if not, more causal interactions).[89] The members of the classes of chemistry (e.g., the elements) fall between fundamental particles and species—they have fewer shared properties than species and more than electrons and their causal structures are more complex than those of fundamental particles but less complex than those of species (see Figure 3.9).

The degree of causal unity a class enjoys, which is determined by the amount of variation found in the class, presents as differing amounts of variation in the properties exhibited by class members.[90] The members of classes that enjoy high causal unity exhibit very little variation in properties (e.g., fundamental particles, elements), while the members of complex classes tend to exhibit greater variation (e.g., species). Furthermore, the number of properties associated with a class appears to be related to the degree of causal unity the class enjoys since complexity is related to causal unity—i.e., more complex classes (i.e., classes whose objects have many properties) enjoy less causal unity, while simpler classes (classes whose objects have fewer properties) enjoy greater causal unity (see section 3.2.1.b).[91] Gold, a relatively simple class (i.e., it has a small property cluster) enjoys high causal unity (the properties shared by instances of gold are likely to occur in all members of the class), while *Canis lupus familiaris,* a relatively complex class (i.e., it has a large property cluster) is only moderately

[89] This sort of consideration, focused on the relatively few properties the classes of physics are likely to share, led Machery (2009) to suggest that the classes of fundamental physics are probably best thought of as not constituting natural kinds (i.e., scientific kinds). Without independent reason to think that natural kinds (i.e., scientific kinds) should share a relatively large number of properties, something Machery never seems to offer, his claim seems unjustified. I discuss the connection between scientific kinds and natural kinds in greater depth in section 3.4.

[90] This idea is hinted at in Mitchell (2002).

[91] Recall that degree of causal unity is determined by the amount of variation among class members which results from the amount of variation in causal structure.

causally unified (the properties shared by *Canis lupus familiaris* are unlikely to occur in all class members).

Electrons **Structureless** **Highly Causally Unified**	**Gold (Au)** **Weak causal structure** **Highly Causally Unified**	**Canis lupus familiaris** **Causally complex structure** **Moderately Causally Unified**
• Negative charge • -1.602×10^{-19} coulombs • Invariant mass = 5.489×10^{-4} atomic mass units • Spin -½ particle • Properties of both waves & particles	• Atomic weight of 79 • Standard Atomic weight: 196.966 • Tensile strength 120 MPa • Dense: 19.3g per cm^{-3} • Malleable (Moh's hardness: 2.5) • Melting point: 1064.18 C • Yellow • 36 radioisotopes (195 is stable for @180 days)	• Dichromatic vision • Temporal resolution between 60-70 Hz • Auditory frequency range between 40-60,000 Hz • Brain dominated by olfactory cortex • Four legs • Tails (though some may be born with no tail) • Socially intelligent • Many properties associated with mammals such as heart, 2 kidneys, mammary glands

Figure 3.9. Table of objects from the various sciences that enjoy different degrees of causal unity and possess property clusters of varying size.

The causal unity of a class is what allows us to generalize findings from a few instances of the class to the class as a whole (see section 3.2.1.). This is not a novel claim. The role that causal unity plays in inductive generalizations has been recognized and discussed by numerous philosophers, most notably Richard Boyd (1991, 1999, 2010) Hilary Kornblith (1993), Ruth Millikan (1999), Bernhard Nickel (2010), and Howard Sankey (1997). The take home message of all of these theorists is that the non-deductive reasoning that scientists engage in, especially inductive generalizing, is most likely to succeed when they reason over causally unified kinds. Since uncovering P-laws is essentially a practice of inductive generalizing, it seems to follow that one can best satisfy the aim of uncovering P-laws by focusing on causally unified kinds. This suggests a second feature of scientific kinds, a feature that helps to explain why scientific kinds support P-laws:

Feature II: *scientific kinds are classes whose members enjoy causal unity.*

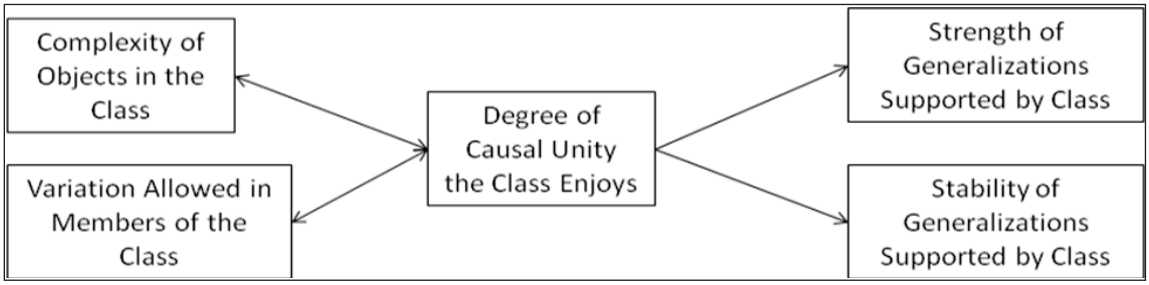

Figure 3.10. Figure representing connection between Causal unity and strength and stability and causal unity and complexity and variation.

The causal unity of a class is connected with the strength and stability of the P-laws the class supports (see Figure 3.10). This claim follows from the work in section 3.2.1.b that connected complexity with causal unity and complexity with both strength and stability. As further support for this claim, consider the following examples. The class of electrons is highly causally unified because electrons are simple objects. This class supports P-laws that are strong and stable. Currency, a class from economics, is weakly causally unified because of its complex nature. The P-laws that this class supports are unstable and weak. The various mammal species, a set of biological classes, enjoy moderate causal unity owing to the moderately complex nature of the objects in these classes. They support moderately stable and moderately strong P-laws. These examples suggest that the degree of causal unity that a class enjoys is probably related to the degree of stability and strength of the P-laws the kind supports: highly causally unified kinds support strong and stable P-laws, less causally unified kinds support less stable and weaker P-laws. These considerations suggest an amendment to feature II of scientific kinds:

Feature II b: *scientific kinds enjoy varying degrees of causal unity and the degree of causal unity that a kind enjoys is related to the degree of stability and/or strength of the P-laws the kind supports.*

One may question the idea that kinds can enjoy differing degrees of causal unity as a result of their compositionally complex nature.[92] The worry might be that all classes that enjoy causal unity are actually similarly causally unified and that the alleged difference in causal unity is simply a reflection of our lack of understanding and appreciation of the causally complex nature of these kinds. If all kinds are, in fact, equally causally unified, then this would suggest that understanding how causally unified a kind is may be less informative than I propose and that it is probably best to leave it out of consideration. This worry is not without merit. If we think of causal unity as being tied to the ability to predict the features of kind members (i.e., to predict which properties a class-member will possess), then it could be that degrees of causal unity actually just reflect a lack of fully appreciating the causal complexity of kinds. Accordingly, the reason that kinds such as species appear less causally unified than kinds like the elements is because we don't fully appreciate, or have not identified, all of the causes that play a role in giving rise to members of species-kinds, while we do understand this about the elements. If we understand causal unification in this manner, then there is reason to think that all kinds may be equally causally unified. But, I am here interested in a specific notion of causal unity, one that understands unity as being tied to the degree of variation found in the shared causal structure. For my notion of causal unity, possessing a less unified structure in virtue of allowing for variation and differentiation in causes is what determines causal unity, not our ability to predict the features of kind members (though predictive ability is relevant insofar as the causal unity of a class will determine the sort of P-laws the class supports).

A final point to clarify, one I hinted at earlier, is the idea that various sciences may recognize differing degrees of causal unity as important. Physics, for instance, may only be interested in classes that enjoy a high level of causal unity. Chemistry may be another science that places relatively high demands on the degree of causal unity required of scientifically

[92] The following objection is due to Michael McCloskey.

interesting classes. The emphasis on such high degrees of causal unity seems to be mirrored in the emphasis these sciences place upon P-laws that are highly stable and strong. From these observations, one could, perhaps, devise a normative constraint on the scientific kinds of these sciences: they *ought* to enjoy a high degree of causal unity. Biological scientific kinds seem to require less causal unity than the kinds of physics and chemistry, perhaps because the objects of interest tend to be much more causally complex and the generalizations of interest tend to be weaker and less stable. But, biological scientific kinds seem to be more causally unified than some other scientific kinds like those from economics or anthropology. The classes of interest to economists, for example, seem to enjoy an even lesser degree of causal unity than those of interest to biologists, perhaps because the causal structures of such classes are so causally complex. After all, the causal structure of currency includes much more than the material the currency is made of (if it is made of anything at all)—the structure also includes the psychological and social causes that give rise to money in the first place. And, perhaps unsurprisingly at this point, the P-laws of economics are even weaker and less stable than those of biology suggesting once again that the degree of causal unity and the degrees of strength and stability of interest may vary from science to science.

So let us recap this second feature of scientific kinds before moving on to the third feature. Scientific kinds are classes that enjoy varying degrees of causal unity. The degree of causal unity that a scientific kind enjoys is related to both the causal complexity of the kind and the degrees of stability and strength of the P-laws it supports. There may be normative constraints that apply to scientific kinds and these constraints may vary from science to science. One likely constraint on scientific kinds has to do with the degree of causal unity that a given

science requires for a class to count as scientifically interesting and useful. Hence, these constraints will be indexed to particular sciences.[93]

3.3.4 features of scientific kinds III: scientific explanation

A third feature of scientifically useful and interesting classes is that they tend to figure in scientific explanations. This may seem like a point hardly worth mentioning since the appearance of a class in an explanation suggests that scientists have already taken an interest in the class—i.e., that it has already been recognized as a theoretically interesting and useful class. The point that I wish to emphasize, however, is that scientists find these classes interesting not simply because they actually do figure in an explanation(s) but because the fact that they do suggests that they are likely to support P-laws that make them interesting in their own right. Let me begin with some brief remarks on explanation before I develop this point in detail.

Science's interest in explanations is rather straightforward; explanations help us better understand why things are the way that they are. This is the case for all sorts of explanations: mechanistic explanations help us get a better understanding of the mechanisms (organized parts and activities) that gives rise to a phenomenon of interest (Bechtel & Richardson, 1993; Machamer, et al., 2000; Craver, 2007), causal explanations help us better understand the causes that give rise to an event (Salmon, 1989), and deductive-nomological explanations help us better understand which general laws a particular phenomenon is subsumed under (Nagel, 1961; Hempel, 1965). All of these forms of explanation help us to better understand the world and there

[93] Determining what the normative constraints of a given domain are is perhaps best accomplished by observing scientists working within the domain.

is no obvious reason to think that any of them are better than the others at aiding us in this endeavor, though some may be more useful than others for specific domains of inquiry.[94]

That a class figures in a scientific explanation is evidence that it is of interest to scientists. But the appearance of a class in an explanation does more than just signal that scientists already have an interest in the class; it also suggests that the class may be deserving of further interest, particularly, an interest focused on uncovering the P-laws the class supports. Uncovering these P-laws is scientifically important because it provides a better understanding of the class which can then be used to develop more, perhaps deeper, explanations. Consider the following example. We begin by taking the appearance of the voltage-gated-ion-channel in both causal and mechanistic explanations of neural action potentials as reason to investigate what, if any, P-laws the class 'voltage-gated-ion-channel' supports. As a result, we discover that voltage-gated-ion-channels have a particular molecular structure; namely, they consist of four membrane spanning subunits, each of which is composed of six membrane spanning alpha helices, arranged in a ring-like shape with a voltage-sensitive channel gate. This finding suggests the following P-law: *all voltage-gated-ion-channels are composed of four membrane spanning subunits and a voltage-sensitive channel gate*. Knowing that this is a P-law of voltage-gated ion channels translates into knowledge that said channels generally have this structure. This fact will allow us to project what we learn about how the gating mechanism works in several ion channels to the class as a whole. The upshot is that we will get a deeper explanation of how neurons generate an action potential because we will have a better understanding of how voltage-gated-ion-channels work—e.g., the change in membrane potential causes a change in the conformation of the membrane spanning units which causes the gate to shift in a way that allows certain ions to pass through the opening created by the four sub-units arranged in a ring. This deeper explanation of the action potential is

[94] Psychology, for instance, is likely to focus on mechanistic explanations and very few, if any, D-N explanations. Physics, on the other hand, seems to be interested in developing D-N explanations but less interested in mechanistic explanations.

made possible by a deeper understanding of voltage-gated-ion-channels, in particular, an appreciation of the P-laws that the class supports. This example is meant to illustrate that we are warranted in taking the appearance of a class in a scientific explanation as *prima facie* reason to think that the class is deserving of further attention. The fact that science often seeks to develop deeper explanations of phenomena is evidence, though admittedly only anecdotal, that science actually does take an interest in the classes appearing in scientific explanations for the reasons that I have proposed.

The above points suggest the third feature of scientific kinds:

Feature III: *scientific kinds are classes that figure in scientific explanations.*

The third feature also provides a sort of normative constraint on scientific kinds—scientific kinds *ought* to figure in scientific explanations. That a class figures in an explanation does not, however, guarantee that the class constitutes a scientific kind for simply figuring in such an explanation is no guarantee that the class supports P-laws or enjoys causal unity. After all, some of our explanations may turn out to be mistaken and, as a result, the classes that figured in these failed explanations (e.g., caloric and phlogiston) may be found to not exist, and, therefore, unable to support P-laws. Nonetheless, it still seems that scientists are interested in classes that figure in scientific explanations since doing so indicates that the class has already been recognized as useful and is, therefore, probably worthy of deeper investigation. In many cases, scientists will learn more about the P-laws that the class figuring in the explanation supports. In other cases, further investigation of a class may reveal that the class does not actually support P-laws of appropriate strength and stability indicating that the class does not constitute a scientific kind for a certain science. In both cases, further investigation of the class proves useful since it helps us better understand the nature of the class—i.e., that it constitutes a scientific kind or that it does not constitute such a kind.

3.3.5 P-scientific kinds

So let us recount what we have learned about scientific kinds by applying the pragmatic approach. First, we have learned that scientific kinds—i.e., the classes that scientists take to be interesting and useful—are those that support P-laws, enjoy causal unity, and figure in scientific explanations. We also learned that what counts as a scientific kind may vary from science to science since different sciences may require differing degrees of strength and stability for a generalization to count as a P-law, and/or differing degrees of causal unity for the class to count as causally unified. In order to emphasis the pragmatic methodology that I have employed to devise this account, I will label it the P-scientific kind account.

The P-scientific kind account is a general account of scientific kinds insofar as it offers a description of what makes a class scientifically interesting and useful, namely, that the class supports P-laws, enjoys causal unity, and figures in explanations. These three features are interconnected: that the class enjoys causal unity helps to explain how it supports P-laws, and that it supports P-laws helps to explain why it figures in scientific explanations. The account can also be used to say something more specific about the scientific kinds of the various scientific disciplines since it is possible to locate normative constraints on discipline specific scientific kinds (i.e., the scientific kinds of a particular scientific discipline). The idea is that we may be able to locate, or derive, these discipline specific normative constraints by focusing on the fact that classes may need to support P-laws that enjoy a certain degree of stability and strength and/or enjoy causal unity of a certain degree in order to be considered interesting and useful to a specific scientific discipline. Physics, for example, may only recognize classes that support highly stable and extremely strong P-laws as constituting scientific kinds suggesting a normative constraint on the scientific kinds of physics—that they ought to support highly stable and strong P-laws. Such discipline specific normative constraints, if they are to be found, are derived from actual scientific practice. That is just to say, the constraints are meant to encapsulate the scientist's views about 1)

the degrees of stability and strength that a generalization must enjoy in order to count a as P-law for that science, and 2) the degree of causal unity that a class must enjoy to support the right sort of generalizations (i.e., P-laws) for that science. These two features of scientific kinds normally work in tandem: the degree of stability and strength a discipline's P-laws must enjoy is connected with the degree of causal unity the classes of interest must enjoy and *vice versa*. To recap, the important features of P-scientific kinds are:

I. **Role in P-laws:** *scientific kinds are classes that support P-laws where P-laws are understood to be those generalizations about causal patterns that enjoy varying degrees of strength and stability and that scientists employ for the purposes of explanation, intervention, and prediction.*

II. **Character of causal unity:** *scientific kinds are classes whose members enjoy causal unity.*

- ***a. Character of inter-relation:*** *scientific kinds enjoy varying degrees of causal unity and the degree of causal unity that a kind enjoys is related to the degree of stability and/or strength of the P-laws the kind supports.*

III. **Role in scientific explanation:** *scientific kinds are classes that figure in scientific explanations.*

IV. **Discipline specific normative constraints:** *There are often discipline specific normative constraints on scientific kinds. Such constraints reflect the views of scientists from within a given discipline about 1) the degrees of stability and strength that a generalization must enjoy in order to count a as P-law for that discipline, and 2) the degree of causal unity that a class must enjoy to support the right sort of generalizations (i.e., P-laws) for that discipline.*[95]

It is worth pointing out that the role of normative constraints on P-scientific kinds takes on added significance when the task is to better understand the P-scientific kinds of a certain discipline. Because my project is interested in such a task—i.e., developing a deeper understanding of the scientific kinds of medicine (MCs in general and the MCs of psychiatry in particular)—the normative aspect of P-scientific kinds will figure prominently in later portions of this chapter and the next.

[95] All of the classes from a given science may not allow for equal amounts of variation even though they may all be more or less equally causally complex. For example, some MCs may not tolerate any variation even though the "broken-down" system is highly causally complex. In these cases, we would have a class that is highly causally complex and highly causally unified.

Before moving on, two further points about P-scientific kinds are worthy of mention. First, the P-scientific kind account is a realist account of scientific kinds insofar as it takes the shared causal structures (and causal mechanisms) that unify P-scientific kinds to exist in the world.[96] This point needs to be stated explicitly since some may think that the pragmatic account of scientific kinds is implicitly committed to an instrumentalism about scientific phenomena. The pragmatic approach that I employ has no deep commitment to instrumentalism aside from the very superficial instrumentalist idea that we need to consider how scientific phenomena are used by scientists in order to understand them. But, the pragmatic approach is also not necessarily committed to realism about scientific kinds or laws for that matter. After all, if it turned out that actual scientific practice concerning classes and generalizations was insensitive to causal facts about the world, then this would have been reflected in the pragmatic account of scientific kinds and laws. That is to say, if observation revealed that scientists consider any generalization a law that plays a certain role in scientific reasoning regardless of whether it tracks any causal patterns in the world, then the P-law account, reflecting this observation, would be an instrumentalist account of laws. What I have suggested, following Mitchell (2000, 2004), is that scientists do not appear to be insensitive to such causal structures and are, therefore committed to (at least) a weak form of realism. The fact that the P-scientific kind account is a realist account of scientific kinds means that P-scientific kind status is an objective matter of fact. Accordingly, once we have determined what the discipline specific normative constraints on P-scientific kinds are, then it is a matter of fact whether some class constitutes a P-scientific kind for that science.

Second, something should be said about those classes that do not yet play a role in science but enjoy the causal unity necessary to support P-laws and figure in scientific explanations. These are what I will call potential P-scientific kinds. Potential P-scientific kinds are classes that do not yet figure in any scientific explanations but enjoy causal unity and support

[96] For similar "weak" realist positions see Dupre (1993), Kitcher (2007), and Boyd (2010).

P-law-like generalizations (i.e., generalizations about causal patterns that have not yet been employed by scientists for the purposes of explanation, prediction, and intervention).[97] These classes do not count as actual P-scientific kinds because they are not yet employed by scientists for explanation, prediction, and intervention purposes. Potential P-scientific kinds become actual P-scientific kinds and P-law-like generalizations become P-laws when scientists take an interest in them—i.e., when the P-law-like generalizations they support are employed for explanation, prediction, and/or intervention purposes.

In this section I have presented the P-scientific kind account. More work remains to be done in order to fully develop the account and draw out all of its theoretical consequences. This is a task that goes beyond the scope of this paper. For now, what I have presented ought to be enough to allow us to proceed with the job at hand—developing a better understanding of medical P-scientific kinds. But before we do that, we should briefly explore how the P-scientific kind account relates to natural kind accounts.

3.4 P-scientific kinds and natural kinds

The goal of the previous section was to provide a general account of the classes that scientists take to be theoretically interesting and useful. To do this, I employed the pragmatic approach which places emphasis on how scientific phenomena are used by scientists. The theoretical upshot of this was the P-scientific kind account. This work is not the first attempt to say something about scientifically interesting classes (i.e., scientific kinds). Indeed, there is a long history of attempts to clarify why some classes are scientifically interesting and others are not. Much of the work in this area has supposed that the scientifically important classes, especially the classes that ought to figure in scientific laws and scientific explanations, are those

[97] It could be that these kinds are not currently employed because they have not yet been identified as classes that enjoy causal unity and are able to support P-laws.

that constitute natural kinds (Putnam, 1973; Fodor, 1974; Boyd, 1991, 1999, 2010; Khalidi, 1993; Collier, 1996; Bromberger, 1997; Griffiths, 1997; Ellis, 2001). In this section, I assess several accounts of natural kinds since they are competitors to my P-scientific kind account. I will suggest that the essentialist account of natural kinds fails as a general account, while the Homeostatic Property Cluster account (hereafter, the HPC account) seems to succeed. I then go on to explain how the P-scientific kind account and the HPC account are related to each other. I suggest that the two theories are consistent with each other such that HPC kinds constitute P-scientific kinds and *vice versa*. Nonetheless, I propose that the P-scientific kind account has elements that are improvements on the HPC account; in particular, the fact that it makes explicit that the degree of causal unity scientific kinds enjoy, and that the stability and strength of the P-laws these kinds support may vary from science to science, helps to remind us that different normative constraints on scientific kinds may apply to different sciences. I suggest that another virtue of the P-scientific kind account is that it enjoys a break from the metaphysically laden terminology of natural kind accounts making it less prone to lead to conceptual confusion. Let us begin by considering the essentialist account of natural kinds.

3.4.1 The essentialist account of natural kinds

The essentialist account of natural kinds is often traced back to Locke's discussion of real, but unknowable, essences in nature (1690). Contemporary essentialist thinkers have taken on board the idea that natural kinds possess real essences but, contra Locke, have argued that we can gain knowledge about these "essences" through scientific investigation. The essentialist account was brought to prominence in the work of Kripke (1972) and Putnam (1975), but it has enjoyed substantial support by contemporary theorists such as Brian Ellis (2001). There are two key features to this account:

1. For each natural kind, there are necessary and sufficient conditions for kind-membership- presumably, a kind-specific essence.

2. Natural kinds are matters of fact, not natters of convention.

There is a third assumed, if not always explicitly posited, feature of essentialism.

3. The kind-specific essence is intrinsic to the entity.

Essentialism is a normative account of natural kinds.[98] As such, it holds that there are normative constraints on classes constituting natural kinds that are not derived from scientific practice; rather, these constraints are determined via philosophical reasoning about how certain classes should be if they are to be scientifically interesting.[99] This does not mean, of course, that the account is insensitive to scientific practice. Indeed, a core feature of the account is that we can learn about natural kinds through scientific investigation. What the normative aspect of the account entails, however, is that the findings from science are relevant as a means to understand the nature of individual natural kinds (i.e., the nature of kind-specific essence), not to understanding the nature of natural kinds in general.

The normative nature of the essentialist account makes it unlikely to function as an account of scientific kinds. Recall that an account of scientific kinds should be general in the sense that it should respect the platitude that scientific kinds are those classes that are theoretically interesting and useful to scientists. To be a successful account of scientific kinds then, an account must do more than just offer an account of the interesting and useful classes of some sciences (e.g., physics and chemistry)—it needs to offer an account that ranges over all the sciences! For this reason, an account of scientific kinds needs to be able to say what distinguishes

[98] I see the essentialist account as a normative approach to natural kinds since it is not sensitive to contemporary scientific practice in understanding what counts as a natural kind. It could be that essentialism started as a descriptivist account of natural kinds—i.e., Locke may have proposed essentialism based on the science of the 1600's and Putnam and Kripke may have made their proposals based on the science of the 1970's as well. But, the account lost its descriptivist flavor and became a normative approach when it stopped being sensitive to how scientists understand and reason about natural kinds (even if they do not use the terminology of 'natural kinds'), a feature of the essentialist account revealed by the fact that contemporary essentialists hold that natural kinds may be relatively few in number (see Ellis, 2001).

[99] Pragmatic accounts would, in contrast, look at the classes that scientists actually use to figure out the constraints on scientifically interesting and useful classes.

scientific kinds from non-scientific kinds understood as general classes. The essentialist account fails to do this. After all, it may be the case that the classes of interest to sciences like physics and chemistry are properly characterized by essentialism since the microstructures that these sciences focus on may actually count as kind-specific essences. It is unlikely, however, that other higher-level sciences like biology, psychology, or economics investigate classes that can be said to possess kind-specific essences (Boyd, 1991, 1999; Hacking, 1991, 2007).[100]

It is worth pointing out that the essentialist account still enjoys major support by some philosophers, especially those that focus on issues in physics and chemistry, two areas of science that might posit and investigate classes of entities that actually do possess the three features noted above (see Ellis, 2001; Bird & Tobin, 2012). Indeed, I don't doubt that essentialism properly characterizes the scientific kinds of some scientific disciplines (e.g., physics scientific kinds and chemical scientific kinds). My point has simply been to show that the account is unsuited as an account of scientific kinds in general.

[100] Of course, the essentialist may try to dodge the charge that essentialism does not actually account for scientific kinds understood as a general class by arguing that their notion of an 'essence' has been misunderstood and that a proper understanding will reveal the account's ability to handle this charge. So we may wonder, what might the essentialist mean when she talks about 'essences'? Whatever it is, it seems that she is committed to the claim that two entities belong to the same kind because they possess the same kind-specific essence (Craver, 2009, discusses the problems that issue from this idea at length). This claim raises the further question, what does it mean for two kind-specific essences to be "the same"? Perhaps the idea is that essences are simply identical microstructures, two structures sharing all but their temporal properties. If this is the case, however, then it is unlikely that there are going to be very many sciences that traffic in natural kinds since the classes of interest to most scientific disciplines are unlikely to share identical microstructures at a fine grain of analysis (e.g., one may have an extra-molecule). That essentialism entails that only some sciences traffic in natural kinds suggests that essentialism fails as an account of scientific kinds since scientific kinds are, by definition, classes that scientists *in general* are interested in. A response to this may be that the microstructures only need to be similar enough at a course enough level of analysis. If this is the move, then essentialism needs to say something about what it means for something to be "similar enough" (For more on this point, see Craver, 2009). And, as Quine reminded us many years ago, spelling out what it means for something to be similar to something else is a task that is not nearly as easy as it may appear (1969).

Another point to appreciate about redefining 'essence' so it covers all cases is that it is likely to obfuscate and make it more difficult to learn about scientifically interesting classes. This charge is similar to that aimed at the employment of *ceteris paribus* clauses to extend the range of laws insofar as it holds that the redefinition of 'essence', employed as a means to extend the range of essentialism, will likely cover over important, perhaps informative, differences in the classes of interest to the various sciences (Mitchell, 2002).

3.4.2 HPC accounts[101]

The Homeostatic Property Cluster account (hereafter, HPC account) was introduced by Richard Boyd (Boyd, 1991, 1999, 1999b, 2010) in order to account for those types of entities that are investigated in the special sciences by broadening the scope of the theory of natural kinds. Boyd's work was originally driven by his recognition that most sciences privilege certain sorts of entities because they support inductive generalizations that are theoretically useful (i.e., that can be employed in explanations, predictions, and interventions), a feature traditionally attributed to natural kinds. The widespread privileging of these classes of entities is what Boyd referred to as the "enthusiasm for natural kinds" (1991). The problem with this enthusiasm, as Boyd saw it, was that the only available account of natural kinds—the essentialist account—was incapable of explaining this widespread recognition of natural kinds. This fact, however, did not lead Boyd to conclude that these classes were not natural kinds; rather, it pushed him to propose a new theory of natural kinds that would include many of the classes for which there was great enthusiasm to treat as natural kinds. Boyd's new proposal sought to broaden the range of natural kinds by moving away from the two features central to the essentialist account and focusing instead on the property-inducing (i.e., homeostatic-inducing) casual mechanisms associated with the entities. His position is summarized nicely in the following passage:

> "There are a number of scientifically important kinds (properties, relations, etc.) whose natural definitions include property clusters, the unity of which is causal, rather than conceptual. The natural definition of one of these homeostatic property cluster kinds is determined by the members of a cluster of often co-occurring properties and by the ("homeostatic") mechanisms that bring about their occurrence." (Boyd, 1991, 141)

As this passage notes, according to the HPC account, the defining feature of a natural kind is not a set of properties that are necessary and sufficient for kind-membership, but a property-cluster

[101] The HPC account is sometimes dubbed "neo-essentialism" since many take HPC kinds to be bound by a causal essence. The HPC's break from essentialism is often taken to result from the idea that the causal essence does not function as a sortal essence, a role essentialism seems to attribute to essences. It is a matter of debate just how "essential" HPC kinds are.

that is "brought about" by some underlying causal mechanism(s). The mechanisms are said to be causal because they are taken to be causally responsible for the co-occurring properties and homeostatic since they cause the properties to occur in *most* instances of the kind.[102] While the homeostatic/causal mechanism(s) associated with a kind plays the role of the defining feature of the kind, proponents argue that it would be a mistake to equate the causal mechanisms with the necessary and sufficient property, or essence, of the kind.[103] After all, there may be more than one common causal mechanism that is responsible for sustaining the property cluster, and it is not necessary that every instance of the kind possess all of the causal mechanism(s) (Kornblith, 1993; Boyd, 1999; Wilson, 1999; Brigandt, 2009). The above passage suggests the following features of natural kinds according to the HPC account:

1. Membership in a natural kind is not based on the possession of a set of necessary and sufficient properties,
2. Properties associated with a natural kind tend to cluster together because of the underlying mechanism that gives rise to these properties,
3. The causal mechanism(s) which sustains the relevant property cluster of a natural kind can be considered the defining feature of the kind.[104]

In the years since Boyd first introduced the HPC account, a number of theorists have attempted to clarify and develop it in numerous ways (Brigandt, 2003, 2009; Griffiths, 1997, 1999, 2004; Kornblith, 1993; Wilson, 1999; Wilson et. al, 2007). The three basic commitments discussed above, however, still remain central to almost all of these variant HPC accounts.

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[102] For more on the idea of homeostasis see Bernard (1856).

[103] This is a claim that is often made by proponents of the HPC account—that the causal mechanism is not a defining feature of the kind. Some HPC proponents, however, reject this claim and take this mechanism to constitute something sufficiently close to a necessary and sufficient property of kind-membership that it is deserving of the title "necessary and sufficient property of kind-membership". Such a move has led some to view the HPC account as a "neo-essentialist" position. I do not intend to argue in favor of either of these interpretations as I am merely canvassing the conceptual space of the HPC account in order to see how it compares with my account of P-scientific kinds.

[104] One way that the HPC account diverges from traditional essentialist accounts is that there is no requirement that the causal mechanism is intrinsic to the entities in question—e.g., some HPC theorists hold the common homeostatic mechanism of a species to be its line of descent.

The HPC account, like the P-scientific kind account, appears to be an attempt to give a general account of the classes that scientists find interesting and useful. This feature of the account is revealed by Boyd's drive to maintain and explain, rather than deny and explain away, "natural kind enthusiasm" throughout the sciences. That Boyd sought to better understand these scientifically interesting classes by considering the inductive practices, and the classification schemes that support such practices, of actual scientists is evidence that the HPC account, at least partially, is properly construed as rooted in the pragmatic methodology. Indeed, Boyd seems to come to similar conclusions about HPC kinds that I came to about P-scientific kinds: they enjoy causal unity, they support inductive generalizations of a sort, and they figure in explanations. If these two accounts are so similar, one may wonder, what value is there in introducing and employing the P-scientific kind account. Is doing so just another instance of reinventing the wheel?

To answer this question, let us consider the ways in which these two accounts differ. First, the P-scientific kind account emphasizes that classes of interest may vary in degree of causal unity and that the generalizations that such kinds support may vary with respect to stability and strength. By making these features of scientific kinds explicit, the P-scientific kind account can easily explain variation with respect to scientific kinds among the different sciences. That such variation exists is not inconsistent with the HPC account but it is also a feature that is not explicitly built into it. Building this feature explicitly into the P-scientific kind account is a strategy to ensure that such differences are noticed. After all, these differences are central to understanding the various sciences and any account that fails to capture this is likely to gloss over important aspects of scientific practice.

Second, the P-scientific kind account has a principled means to determine when two causal mechanisms are similar enough to be considered common causal mechanisms. The HPC account runs into problems since it does not specify the level at which we should look for

similarity when assessing mechanisms and it is possible that the mechanisms that appear similar at a higher level are found to differ at a lower level bringing into question claims of similarity (Craver, 2009). For example, two human visual systems that appear similar at the systems level may, nonetheless, appear quite unique and distinct at the cellular level? Does this finding mean that these two visual systems share a common causal mechanism or not? As Craver suggests, the HPC account does not seem to have a ready answer to this question (2009). The problem of determining when there is similarity is likely to generalize since most objects and processes will have minor differences at the lowest level. The P-scientific kind account isn't bothered by this problem since it can rely on the levels relevant to a specific science to determine the appropriate level to look for similarity in causal mechanisms. In cellular biology, for instance, the relevant level will be the cellular level, not the molecular or systems level. For molecular biology the relevant level to look at for similarity will be the molecular level, not the quantum level. Hence, even if there are differences at the quantum level, it will not matter, for what is relevant for molecular biology will be similarity of molecular level parts. Some sciences, such as the neurosciences, tend to be intra-level. For these domains, it may be the case that we are either forced to consider similarity at the lowest relevant level, or recognize that the science itself should be understood as multi-level—i.e., that there are molecular level neural P-scientific kinds, cellular level neural P-scientific kinds, systems level neural P-scientific kinds, etc. (see Craver, 2007, 2009). When it comes to the above question concerning the visual systems, the P-scientific kind account appears to have a ready answer: the two visual systems share a common causal mechanism for systems level neuroscience, but not for cellular level neuroscience. That the account has such an answer issues from the fact that it does not require us to consider non-relevant lower levels when assessing similarity. The HPC account, on the other hand, seems to have little to say about when and where one draws the line when assessing mechanisms for similarity (Craver, 2009)

Third, the P-scientific kind account eschews natural kind terminology since it is likely to lead to conceptual confusion. The sort of conceptual confusion I have in mind is like that discussed by Griffiths et al. concerning innateness (2009). These authors argue that even though there may be a successor concept of innateness that outperforms previous concepts, it would be unwise to continue calling this a concept of innateness since the term 'innateness' has been associated with so many ideas and any concept termed 'innateness' is likely to function as a "conceptual sinkhole" that brings us back to those earlier associated ideas. The term 'natural kind' is likely to function as a "conceptual sinkhole" in a similar manner. After all, there is a long history of associating natural kinds with essences that are necessarily intrinsic, of thinking of natural kinds as objects or processes that must be naturally occurring, etc. that may not hold true for all scientifically interesting classes. By continuing to insist on terming scientific kinds 'natural kinds', we run the risk of being sucked into the "conceptual sinkhole". By steering clear of this terminology, the P-scientific kind account guards against associating past metaphysical baggage associated with natural kinds.[105]

I take the above three points as grounds to think that the P-scientific kind account outperforms the HPC account. Importantly, I see the P-scientific kind account as sympathetic with the HPC account. As already noted, the two accounts share several core features and it seems that all P-scientific kinds are HPC kinds, and all HPC kinds are P-scientific kinds. Accordingly, arguments concerning the HPC kind status of a class easily translate into arguments

[105] A final problem with the HPC account is that some proponents present the common causal mechanism as being a causal essence that is not a sortal essence—i.e., that the causal essence cannot be used to sort entities into those within the class and those not in the class (Samuels, 2009; Gelman & Hirschfeld, 1994, 1999). This claim is a variant of the claim that the common causal mechanism is not a necessary and sufficient property of kind-members. The problem with the distinction between causal and sortal essences is that it is unclear why we should not think that the causal essence also functions as a sortal essence. Perhaps it is because causal essences may admit of borderline cases and sortal essences cannot allow this? In any case, if causal essences function as sortal essences, then it seems like the causal essence of the HPC account plays the very same role that the essence plays in essentialism and, therefore, inherits some of the unwelcome theoretical baggage of essentialism. If the above analysis holds, then the only difference between the two may be that the HPC account has wiggle room since it can attempt to tie similarity of essences to a certain level deemed especially relevant by a given science (what I earlier propose the P-scientific kind account explicitly argues for), while essentialism seems required to hold that the essence is similar at the deepest of levels.

about the P-scientific kind status of the class. This fact allows one to engage in debates about the HPC kind (i.e., natural kind) status of certain classes even if one is primarily interested in the P-scientific kind status of classes. The difference between the two accounts is to be found mainly in the emphasis that the P-scientific kind account places on making explicit, and understanding, the connections between the causal unity a kind enjoys, the P-laws the kind supports, and the explanations it figures in. Exposing these connections makes it easier to appreciate the nature of scientific kinds for the various sciences, a task which can provide us with normative constraints on that science's scientific kinds. And, by helping us better understand scientific kinds, the P-scientific kind account helps us better understand scientific practice. Now that the P-scientific kind account has been introduced and shown to outperform the HPC account, I will move on to a discussion of the P-scientific kinds of medicine, medical kinds.

3.5 Medical Kinds: the P-scientific kinds of Medicine

Recall that just because the P-scientific kind account takes the rather deflationary view that scientific kinds just are those classes that support P-laws, figure in explanations, and enjoy causal unity, it does not follow that there is nothing more to be said about P-scientific kinds. It seems that much of the theoretical work to be done will center on understanding the intricacies of science specific P-scientific kinds, specifics involving constraints on P-law strength and stability and required degree of causal unity. Additionally, there may be science specific features of P-scientific kinds that are worth exploring. An interesting feature of the P-scientific kinds of evolutionary biology, for instance, is that they are related via lines of descent and that they often have parts that are homologous. The P-scientific kinds of functional biology are likely to enjoy a nested nature insofar as many classes of interest to these theorists (e.g., cells) are composed of sub-classes that also constitute P-scientific kinds (e.g., neural cells, glial cells, skin cells, etc.). The P-scientific kinds of other sciences are also likely to possess unique features that distinguish

them from other sub-classes of P-scientific kinds. In this section, I clarify the P-scientific kinds of medicine.[106]

Given that medicine aims to be scientific insofar it strives to be grounded in biological theory, it follows that there will be P-scientific kinds that are specific to the domain of medicine. The classes that constitute medical P-scientific kinds would be those that enjoy causal unity, support medical P-laws, and figure in medical explanations (hereafter, I use 'medical kind' or 'MKs" as short hand for medical P-scientific kinds). These kinds will also be the ones that figure in a medical taxonomy (i.e., a nosology). Given my remarks from earlier, I want to suggest that the classes of interest to the purely objective leg of medicine (i.e., the research aspect of medicine), medical conditions (i.e., MCs) are the obvious candidates for constituting MKs.[107] To see whether these classes actually do constitute such kinds, we will need to explore whether MCs actually do enjoy causal unity, support P-laws of interest to medicine, and figure in medical explanations. After arguing that MCs constitute MKs, I turn my attention to a discussion of some special features of medical kinds, features that hinge on the role of etiological and pathophysiological factors in MCs and how these factors are relevant to both the way that MCs are typed and the development of a nosology given a partial-objectivist understanding of medicine.

3.5.1 Medical Kinds: The P-scientific kinds of medicine

Recall from the previous chapter that MCs is a term used to refer to those classes whose instances share a cluster of signs and symptoms[108] in virtue of a common malfunctioning

[106] It should be remembered that when I speak of medicine, I have in mind medicine understood in accordance with partial-objectivism.

[107] By addressing the P-scientific kind status of MCs, we are also investigating the status of diseases as P-scientific kinds since only MCs are potential diseases.

[108] In common medical terminology, symptoms are often taken to be the subjective indicators of a condition, while signs are taken to be the objective indicators of a condition.

biological part—i.e., an abnormal biological state. Accordingly, broken tibia, hepatitis, respiratory infection due to influenza virus, atherosclerosis, and Huntington's disease constitute MCs if the instances of each class involve a common malfunctioning part that gives rise to the associated cluster of signs and symptoms. But do MCs constitute MKs? If MCs are of theoretical interest to medical researchers, theorists, and practicing physicians because they figure in medical explanations, support medical P-laws, and enjoy the appropriate sort of causal unity, then the answer seems to be yes. I believe that MCs do constitute MKs.

Like all P-scientific kinds, MCs enjoy causal unity in the sense that they share a property cluster (i.e., the cluster of signs and symptoms) in virtue of a common causal mechanism. The common causal mechanism of a MC is the malfunctioning biological part.[109] The malfunctioning biological part that sustains the associated cluster of signs and symptoms may occur at the genetic, molecular, cellular, or system level. The accompanying cluster of signs and symptoms is a proper part of the MC, though what confers identity is the malfunctioning part. This is just to say, the cluster of signs and symptoms is not an identity conferring aspect of a MC: the malfunctioning part is. Accordingly, in theory, one can enjoy a MC even if they do not exhibit any of the symptoms or signs associated with the MC. That the malfunctioning part confers identity serves to drive home the point that MCs enjoy causal unity.[110]

Because MCs enjoy causal unity they are likely to support the P-laws of interest to medical theorists (i.e., medical P-laws). The P-laws of interest to such theorists typically center on: the etiology of MCs, the normal courses of MCs, the symptoms associated with MCs, and the possible intervention strategies for MCs. That these conditions support such P-laws allows us to project what we learn from a few instances of the condition to the condition as a whole. This, in

[109] Recall that what makes the state biologically abnormal is that the biological part is E-systemically malfunctioning.

[110] At this point, one may worry that I have not yet attempted to explain the role of etiological factors in MCs. I discuss the role of etiological factors and their relevance to typing MCs and MKs in section 3.5.2.

turn, allows us to predict with relatively little experience how an instance of the MC is likely to unfold and how it will respond under certain interventions. Such knowledge is sure to be of special importance to medical theorists since it allows for a richer understanding of these conditions and, therefore, should make diagnosis and treatment interventions, when called for, more accurate.

Given that the human body in which MCs unfold is an extremely complex system, it is likely that medical P-laws will only enjoy an intermediary degree of strength and stability. Of course, the P-laws supported by some MCs may enjoy greater or lesser degrees of strength and/or stability. Huntington's disease is a MC that is likely to support P-laws that enjoy a high degree of strength and stability given the nature of the malfunctioning part—a genetic mutation that is practically fully penetrant—i.e., we can be fairly certain of progression, symptoms, etc. (Murphy, 2006). Tay Sachs is another MC that is likely to support highly stable and very strong P-laws as it is also, more or less, fully penetrant. Atherosclerosis, on the other hand, is a MC that is likely to support P-laws of less strength and stability since some humans have a constitution that allows them to compensate for the disruption to arterial function and, therefore, not follow the "typical course of progression" or express some, or even many, of the associated symptoms, while the constitutions of other humans are such that they are unable to compensate and they will follow the "typical course of progression" and express the full range of symptoms (Murphy, 2006, 2009b). Given the varying nature of malfunctioning biological parts and the complexity of humans understood as biological systems, we ought to expect that the P-laws that MCs support will enjoy varying degrees of both strength and stability.

Finally, MCs are likely to figure in medical explanations. If we know that someone has a particular set of signs/symptoms, then one way to explain this would be to posit that they have a certain MC. Conversely, if we know that a person has a certain condition, then this helps to explain why they exhibit a certain cluster of signs and symptoms as well as why they are likely to

respond to, or not respond to, certain interventions. There are other explanatory roles that MCs will play in medical theorizing but space constraints do not allow for a full discussion. In any case, that these conditions do figure in explanations of interest to medicine should be beyond serious doubt.

In the above paragraphs, I have argued that MCs fulfill the role of MKs (i.e., the P-scientific kinds of interest to medicine understood as a partial-objectivist endeavor). Accordingly, it seems quite reasonable to claim that MCs of all types—i.e., infectious MCs, congenital MCs, autoimmune MCs, and externally induced MCs—constitute MKs given that MKs are simply those classes that enjoy causal unity, support P-laws of interest to medicine, and figure in medical explanations. MCs that have a viral or bacterial infection as an etiology have a malfunctioning part that results from the introduction of the infectious agent. The malfunctioning parts of infectious MCs can take many forms depending on the nature of the infectious agent. Furthermore, the infectious agent itself is not a part of the MC though it is often necessary to sustain the condition. Congenital MCs involve a dysfunction in the process of genetic signaling and reproduction that results in a malfunctioning biological part. Congenital hearing loss is one of the best known congenital MCs. The malfunctioning part of this MC is a malfunctioning auditory system, though it is almost certain that dysfunctions occur at lower levels (e.g., malfunctioning protein synthesizing mechanisms) which are ultimately responsible for the system-level malfunctioning auditory system. The malfunctioning part responsible for auto-immune MCs is the person's own immune system. This biological part can dysfunction in several ways giving rise to the various auto-immune MCs such as rheumatoid arthritis, eczema, Celiac disease, and autoimmune hepatitis. Finally, 'externally induced MC' refers to what are often considered injuries—e.g., broken bones, bullet wounds, etc. The malfunctioning biological part of these MCs is almost always systems or even person level. Broken tibia, for instance, is an

externally induced MC since all instances of broken tibia share a common malfunctioning part, namely, the broken tibia, which gives rise to a shared cluster of signs and symptoms.

Because MCs constitute MKs, we are now in a position to assert that MCs are the classes that do—and, indeed, should—figure in a nosology.[111] Furthermore, because biological parts from any level can malfunction—i.e., there are genetic level MCs (e.g., malfunctioning genetic mechanisms), molecular level MCs (e.g., malfunctioning molecular mechanisms), cellular level MCs (e.g., malfunctioning cellular components), and systems level MCs (e.g., malfunctioning systemic mechanisms)— and there is no obvious reason to think that medicine privileges any one level over the others[112], we should expect our nosology to include MCs from these various levels.[113] We should not think, however, that a nosology will simply list the known and assumed MCs. Rather, like other scientific taxonomies, a good nosology will organize its entities of interest—the MCs—in a way that illustrates how they are related to each other (Cowan, 1955; Rogers, 1958; Dupre, 2006; Zachar, 2008). By organizing the MCs in this manner, the nosology becomes a powerful tool by representing connections among the MCs in a way that can be easily understood and exploited by theorists. A potential upshot of this type of "relational organization" is that it may allow theorists to identify other "groupings" that constitute MKs—i.e., super-or sub-groupings of MCs. In order to clarify this idea, let us consider how the taxonomies of chemistry and evolutionary biology are organized.

[111] It should be noted that I am only interested in a nosology for medicine understood as a partial-objectivist practice.

[112] Though there is definitely a perception that medicine will, or should, eventually be able to understand all MCs in terms of malfunctions at the genetic level, it is unlikely that this is realistic any time in the near future. Furthermore, it is not clear that understanding all MCs in terms of malfunctioning genetic parts is the best strategy even if it turns out that all MCs involve a malfunction at the genetic level. See Murphy (2006).

[113] The level of a MK is determined by the level at which the malfunctioning part exists: if the dysfunction occurs at the molecular level (i.e., if it is a dysfunction in a molecular part), then it will be a molecular level medical kind; if the dysfunction is found at the cellular level (i.e., if it is a dysfunction in a cellular part), then it will be a cellular level medical kind, etc.

A chemical taxonomy organizes the various elements—chemical P-scientific kinds—into super- and sub-classes with the understanding that the various super- and sub-classes also constitute theoretically interesting and useful classes—i.e., chemical P-scientific kinds (see Figure 3.11). This sort of organizational structure helps us understand that any element that happens to be a member of a super-class chemical P-scientific kind (e.g., any element that is a member of the chemical P-scientific kind "noble gas") will support certain P-laws and figure in certain explanations of interest to chemists in virtue of a shared common causal mechanism associated with the super-class (e.g., for the noble gases, a full outer electron ring). The classification structure also helps us understand that each element constitutes a singular chemical P-scientific kind, including those that belong to the super-class "noble gas", since each element supports a variety of P-laws and figures in a number of explanations that the other noble gases do not and this is in virtue of an element-specific common causal mechanisms (i.e., the number of protons in the nucleus of an element). Finally, the structure allows us to appreciate that even the elements which constitute chemical P-scientific kinds are comprised of a number of sub-classes (e.g., the isotopes) that also constitute chemical P-scientific kinds since they support P-laws and figure in explanations that all instances of the element kind do not, and they do this in virtue of unique common causal mechanisms (i.e., the number of neutrons in the nucleus).[114]

[114] It should be noted that the common causal mechanism that determines membership in an element level chemical P-scientific kind (i.e., the atomic number) is not the same causal mechanism that determines whether or not a class belongs to the super-class chemical P-scientific kind (i.e., the full outer electron ring) or the sub-class chemical P-scientific kind (i.e., the number of electrons), though it may be a component part of the kind (e.g., neutrons are a part of an element even though they are not the causal mechanism that imparts identity and unity to the element).

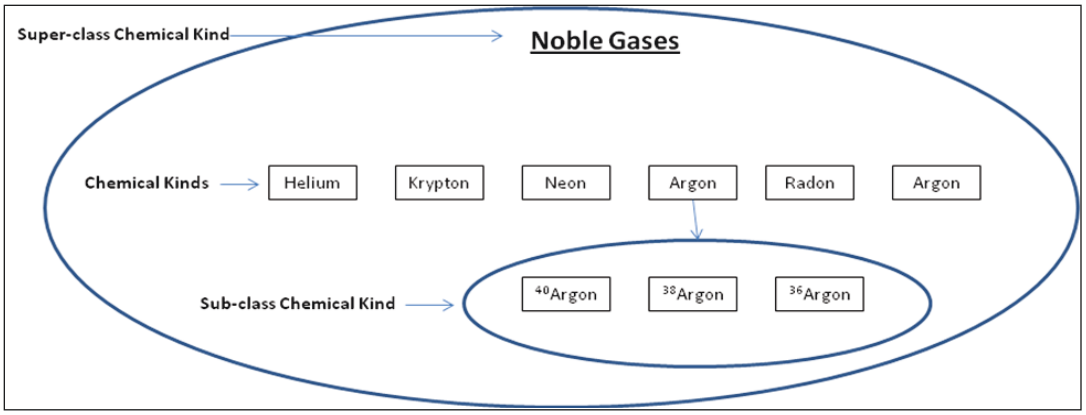

Figure 3.11. Figure representing nested kinds from chemistry.

The P-scientific kinds of evolutionary biology are arranged in a similar nested manner. There are certain evo-bio P-scientific kinds, *genera*, that are subsets of larger, more encompassing evo-bio P-scientific kinds, *classes*, that are themselves subsets of larger evo-bio P-scientific kinds, *orders*. These various groupings constitute evo-bio P-scientific kinds since they enjoy causal unity in virtue of specific causal mechanisms (i.e., common lines of descent) which allow these groupings to figure in explanations and support P-laws of interest to evolutionary biology.

The above points are meant to illustrate that good scientific taxonomies do not simply list a science's P-scientific kinds; rather, they represent the various ways that the kinds are related to each other (Cowan, 1955). By helping us appreciate how these kinds are related, the taxonomy may help us identify groupings of P-scientific kinds—i.e., super-classes and/or sub-classes—that themselves constitute P-scientific kinds and should be included in the taxonomy. The upshot of these remarks to this project is that we will need to consider the various ways that MCs may be related to, and different from, each other when developing a nosology. In order to do this, let us now consider the role of both etiological factors and pathophysiologies (i.e., pathologies) in MCs.

3.5.2 Etiological factors & pathologies[115]

There are two sorts of causal factors relevant to how MCs are typed: etiological factors and pathologies. Following Murphy (2006, 2009b), I understand etiological factors to be the causes of pathologies and pathologies to be the malfunctioning biological parts (i.e., the abnormal biological states) that cause the cluster of signs and symptoms associated with a MC. Because pathologies are identified as the malfunctioning biological parts that constitute MCs (see section 2.4.2.4), pathologies will obviously be central to typing MCs—different pathologies entail different MCs. Given our previous identification of MCs with MKs, it follows that pathologies will also prove central to typing MKs—different pathologies entail different MKs.[116] Accordingly, different MCs will appear as distinct entries in the nosology since they constitute MKs.

So what about etiological factors? Are they relevant to typing MCs? And if they are, is this reason to think that etiology-specific MCs (i.e., the MC understood as resulting from a particular etiology) constitute MKs that are sub-classes of the MK constituted by the etiology-non-specific MC (i.e., the MC with etiological information abstracted away)? If so, should these sub-class MKs be represented in the nosology? Or does the occurrence of multiple etiological pathways for a single MC negate the MK status of that alleged MC? It is to these questions that we now turn.[117]

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[115] The remarks in this section are heavily influenced by Murphy's (2006, 2009, 2009b) account which focuses on the various causal factors that may be relevant to medical conditions.

[116] Having identified pathologies with malfunctioning biological parts, in the remainder of this work I will use the two terms 'pathology' and 'malfunctioning biological part' interchangeably. The term 'pathology', as it is here employed, does not necessarily entail a judgment of harmfulness or undesirability. Rather, it is used as a technical term in line with Murphy (2006, 2009b) to distinguish between the various sorts of causal factors involved in MCs.

[117] For more on these points see Broadbent (2009). Broadbent discusses how some MCs are monocausal while others are multifactorial. I believe that Broadbent's work is correct insofar as it suggests that some MCs are likely to result from fairly specific, singular causes (e.g., deficiency disorders, viral infections), while others require a number of causal factors to be simultaneously present in order to realize the MC (e.g., chronic non-communicable conditions such as high-blood pressure, heart disease, hypertension). A point that Broadbent doesn't discuss, but that seems worth mentioning, is that some MCs can result from a variety of distinct etiological factors. These MCs differ from those that

First, the issue of whether multiple etiological pathways for a single MC problematize the MC's status as a MK. In most cases, a malfunctioning biological part (i.e., a pathology) can result from a number of different etiological factors (i.e., causal influences). Atherosclerosis (i.e., hardened arteries), to borrow Murphy's example, can result from high-fat diet, smoking, or genetic factors (2006; A.D.A.M.). But, just because a malfunctioning part can result from a multitude of etiological factors does not mean that the MC no longer possesses causal unity since all instances of the MC will possess a common malfunctioning part (i.e., the pathology). To return to our previous example, that the malfunctioning part associated with the MC atherosclerosis (the hardened arteries) can result from a number of different etiological factors does not mean that the class atherosclerosis lacks causal unity since the malfunctioning arteries count as the common causal mechanism that unifies the class and sustains the cluster of signs and symptoms in most instances. The example of atherosclerosis is meant to illustrate that a single malfunctioning biological part can be identified as the identity conferring component of a MC even when the malfunctioning part can result from a variety of etiological factors. The upshot: MCs that can result from a multitude of etiological factors still constitute singular MCs—i.e., they constitute a MK—since they enjoy a common malfunctioning biological part (i.e., pathology) (see Figure 3.12).[118]

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result from multiple etiological factors insofar as they are MCs that can result from a variety of factors that need not co-occur. Some of the cases that Broadbent labels multifactorial—e.g., hypertension, heart diseases, etc.)—are examples of what I have in mind. After all, the MC heart disease does not only occur when all of the possible etiological factors are present. It can occur when only a single etiological factor is present (e.g., the genetic predisposition to heart disease or a high fat diet). My work is primarily interested in cases of this sort—i.e., those where multiple etiological factors can give rise to the same MC.

A further issue that I have with Broadbent's work is that he fails to note the distinction between etiological and pathological factors. His claims seem to center on etiological, not pathological, causal factors. Accordingly, it does not really show that MCs do not enjoy a single, pathological factor as I argue below. Rather, he merely shows, as I recognize, that some—indeed, most—MCs result from a variety of etiological factors. I discuss the distinction between etiological and pathological factors in detail in the next section.

[118] This idea is in Murphy (2006, 2009b).

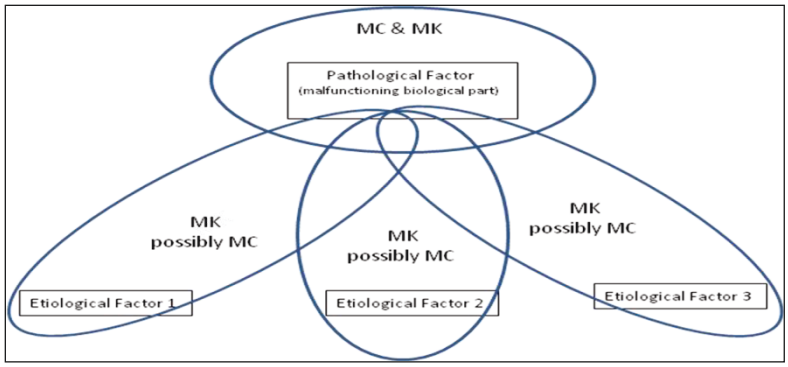

Figure 3.12. Figure illustrating the relationship between MCs, MKs, etiological factors, and pathologies.

The above considerations should not be taken to mean that etiological factors are irrelevant when typing MCs and developing a nosology.[119] After all, information about how malfunctioning biological parts arise in the first place (i.e., etiological information) is theoretically important since it is often the case that the malfunctioning part relies on some etiological factor in order to be maintained.[120] This is especially the case in MCs that result from viral or bacterial infections. Viral hepatitis, for instance, relies on the presence of the hepatitis virus as a means of maintaining the malfunctioning biological part—i.e., the malfunctioning liver. If the virus is eradicated from the body, the liver often returns to normal functioning.[121] By knowing that a given case of hepatitis is caused by the hepatitis C virus (hereafter, HCV), doctors are in a better position to understand how to best intervene in order to restore the patient's

[119] A steady state existence is enjoyed by a pathology when the abnormal biological state is sustained in the absence of the relevant etiological factors. Cortical blindness resulting from a blow to the skull is a steady state pathology since the abnormal biological state of the visual cortex is maintained even when the blow which caused the abnormality—i.e., that caused the visual cortex to malfunction—is no longer present. Many medical conditions may never reach a steady state, a fact attested to by the ability to reverse many medical conditions.

[120] Determining which etiological factors are relevant to maintaining a condition, as well as determining which factors are relevant in the genesis of a condition, is a difficult matter. Much work in philosophy and science are focused on better understanding how we should distinguish causes from background conditions. Some of the most interesting work focuses on causes as difference makers where difference making is spelled out in terms of changes under interventions. See Mackie (1965), Woodward (1997), Murphy (2006), Craver (2007), and Broadbent (2009).

This is a topic that goes beyond the scope of this paper. Furthermore, much of what I will be discussing has to do with causes that have been identified, even if the entire causal nexus necessary for the condition is unknown (e.g., it is often the case that one's genetic makeup predisposes them, or makes them susceptible to some condition but it is not yet understood how we are to determine the influence of such factors on conditions).

[121] But not always since the liver could be sufficiently damaged that it now enjoys a steady-state malfunction.

health—i.e., by attacking the virus. In other non-viral cases of hepatitis, such an intervention may prove useless since the malfunctioning liver is initially caused, and then maintained, by some other causal factor—e.g., excessive lipid storage in hepatic cells. Information about etiological causal factors that give rise to malfunctioning biological parts is likely to be theoretically interesting and useful to medical theorists and, therefore, should be taken into account when typing MCs and developing a nosology (Murphy, 2006). Employing this typing practice results in nested MKs: a single MK (the etiology-non-specific MC) that is composed of several sub-classes that constitute MKs (the etiology-specific MCs).[122] To explain the sort of typing practice I have in mind and what it means for nosology, an example may prove useful.

3.5.2.a Etiological factors and nested MKs: hepatitis as a case study[123]

'Hepatitis' is the term used to refer to inflammation of the liver. Hepatitis has a number of causes: some are bacterial, some are viral (e.g., Hepatitis Virus A, B, and C), some metabolic (non-alcohol induced fatty liver), and some are toxicological (e.g., alcohol induced fatty liver). Hepatitis can be acute or chronic. Acute hepatitis refers to a bout of inflammation lasting less than six months, while chronic hepatitis lasts for more than six months. All instances of hepatitis share a common malfunctioning biological part—an inflamed liver that has trouble performing its functions of synthesizing certain proteins, detoxifying blood and lymph fluids, and producing specific hormones.[124] This part, the inflamed liver, induces a non-specific[125] cluster of signs and

[122] Nested P-scientific kinds are likely to be a widespread phenomenon in most sciences.

[123] This section is based on the following sources: Kountouras et al., 2003; Marchesini et al., 2003; Das & Kar, 2005; Gramenzi et al., 2006; Matsuzaki et al., 2007; Ambade & Mandrekar, 2012; Below are web sources used to develop this section:

- http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0002139/
- http://www.umm.edu/altmed/articles/viral-hepatitis-000078.htm
- http://hepcchallenge.org/

[124] Note that the immune system is functioning properly by attacking and causing the inflammation but the inflammation causes the liver to function improperly. This is often the case with conditions involving infectious agents: the proper function of one part may cause another part to malfunction.

[125] None of these symptoms are unique to hepatitis.

symptoms that includes: jaundice, fatigue, swelling of the liver and other body parts, fingernail and toenail problems, weakness, etc. All forms of hepatitis can lead to scarring of the liver tissue as a result of inflammation and the death of hepatic cells but this is most common in chronic forms. Scarring, or fibrosis, becomes cirrhosis when the scarring is widespread and affects the flow of blood through the liver. If left untreated, cirrhosis can lead to complete liver failure.

Given the above, it seems reasonable to claim that hepatitis constitutes a MC and, therefore, a MK. After all, each instance of hepatitis involves a common malfunctioning part (i.e., inflamed liver) indicating that the class enjoys causal unity. All instances share a cluster of signs and symptoms in virtue of the malfunctioning part and the properties mentioned in this cluster figure in medical P-laws about hepatitis—e.g., *Hepatitis can lean lead to fibrosis and cirrhosis*, *people with hepatitis are likely to experience fatigue and weakness*, *people with hepatitis are likely to experience increased inflammation if they consume alcohol*, etc. And, hepatitis seems to figure in medical explanations: we may be able to explain a patient's symptoms as a result of the person having hepatitis or we may be able to explain why alcohol consumption is making a patient's condition worse by citing the patient as having hepatitis. Given the description of MKs as those classes that enjoy causal unity as the result of a common malfunctioning part, support P-laws of interest to medical theorists, and figure in medical explanations, hepatitis does seem to constitute a MK.

But what about the etiological factors responsible for hepatitis? Are they relevant to typing and determining MK status? The answer seems to be yes. Let me explain why. An interesting fact about most cases of hepatitis is that they can be treated and, often, any damage done can be reversed given the regenerative nature of the liver. But, in order to treat any given case of hepatitis, it is important to appreciate the etiology of the malfunctioning part since the malfunctioning part in hepatitis—the inflamed liver—is usually maintained in virtue of the

relevant etiological factors.[126] Hepatitis resulting from HCV infection is sustained by the virus, while fatty liver hepatitis (i.e., steatohepatitis) is sustained by either alcohol consumption or metabolic disorder. Eradicate the virus in the first case, or halt alcohol consumption or treat the metabolic disorder in the second, and the inflammation usually subsides. Alcohol consumption, the metabolic disorder, and the HCV are all etiological factors that can cause hepatitis. But, these various etiological causes travel along different "paths" and, in many cases, will require different treatment interventions. Hepatitis caused by HCV occurs when HCV invades liver cells and reproduces triggering an immune response that seeks to destroy the virus by destroying infected cells. Through some unknown mechanism(s) the HCV is able to induce premature apoptosis in hepatic cells and cause an over-active immune response which increases inflammation and leads to further hepatic cell death. Steatohepatitis, or hepatitis caused by fatty liver, occurs when there is a disruption to the mechanism that regulates lipid storage in hepatic cells. This results in an overabundance of fat stored in these cells, a condition that somehow leads to inflammation of the liver (Gramenzi, et al. 2006). Disturbance to the mechanism that leads to heightened lipid storage can occur in two ways. In the first, alcohol disrupts the lipid regulation mechanism. In the second, a disorder in the metabolic system causes the disruption in the lipid regulation mechanism. These comments illustrate that the various etiological factors that give rise to hepatitis involve unique causal mechanisms. What I now want to suggest is that this is grounds to treat hepatitis as a MK that is composed of several sub-classes, typed according to these various inflammation inducing mechanisms, which also constitute MKs (see Figure 3.13). Let me offer some reasons to accept my proposal.

[126] This is evidenced by the fact that removal of the relevant etiological factor(s) often leads to a remission of the hepatitis.

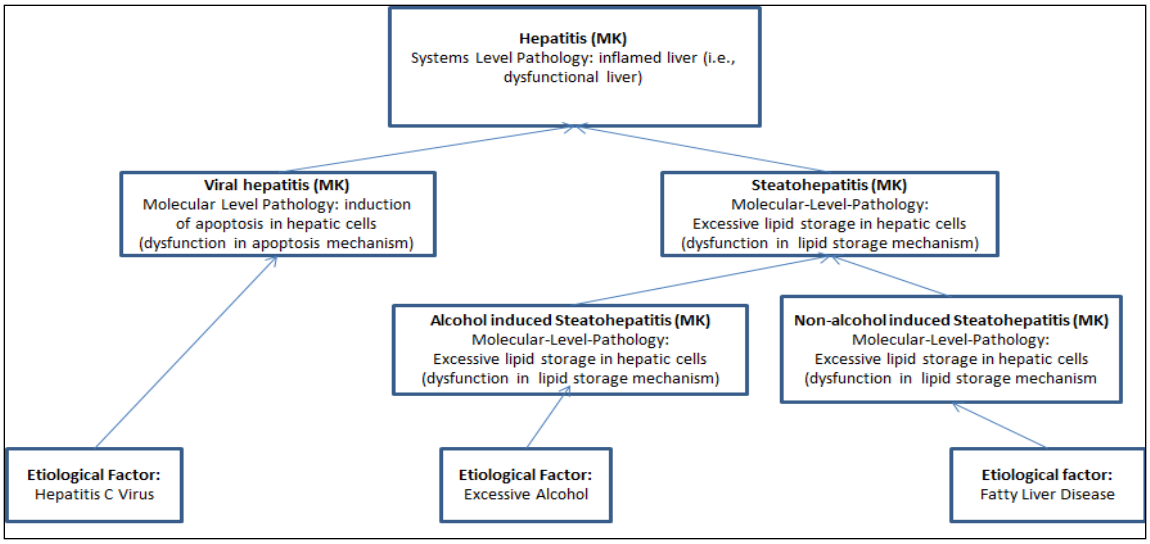

Figure 3.13. Figure illustrating the nosological entry for hepatitis.

First, each sub-class appears to enjoy causal unity insofar as all instances of the sub-class possess a common causal mechanism: HCV is the causal mechanism of one sub-class, alcohol is the common causal mechanism of another sub-class, and metabolic disorder is the common causal mechanism of the third sub-class. That the various sub-classes actually do involve different causal mechanisms is attested to by the different treatment interventions employed for each sub-class—e.g., inter-feuron for HCV hepatitis, prohibition of alcohol for alcohol-induced hepatitis, etc. Second, while all of the sub-classes share a cluster of signs and symptoms in virtue of being instances of hepatitis, each sub-class shares an additional cluster of signs and symptoms that is not shared by the other sub-classes. The additional cluster of signs and symptoms issues from the different mechanisms (i.e., etiological factors) that give rise to the hepatitis. Accordingly, each sub-class also appears to support sub-class specific P-laws: *Viral hepatitis is likely to respond to interfeuron*, *Alcohol-induced steatohepatitis results from oxidative stress which is caused by alcohol metabolism*, etc. Finally, the sub-classes figure in medical explanations. A common explanation for why one hepatitis patient responds to one sort of treatment but not another is because the patient has a certain sort of hepatitis (i.e., viral hepatitis as opposed to non-alcohol induced steatohepatitis). The upshot of these considerations is that we

are epistemically justified in thinking of hepatitis as being a MK that is composed of several sub-classes that constitute MKs that are typed according to etiology (i.e., the etiology-specific MCs).

The above example suggests a key feature of MCs that is relevant to MK status, and, therefore, to nosology—namely, that a MC that constitutes a MK can be composed of several etiology-specific sub-classes that constitute MKs (i.e., MKs may be nested) (see Figure 3.11).[127] In some cases, some or all of the etiology-specific sub-classes of a MC constitute unique MCs since the etiological factor just is a malfunctioning biological part, while in other cases some or all of the sub-classes may not constitute unique MCs even though they do constitute MKs. Infantile Tay-Sachs disease[128], a rare genetic condition that affects children soon after birth and usually leads to death by 4 years of age, illustrates the first possibility well.[129] Infantile Tay-Sachs involves a system-level MC (i.e., dysfunction in the nervous system owing to premature nerve cell death), a cellular-level MC (i.e., a deficiency of the enzyme Hexosaminidase that leads to overaccumulation of gangliosides in nerve cells), a molecular level MC (i.e., a malfunction in the folding mechanism involved in protein transport that leads to the deficiency of the Hexosaminidase A enzyme) and a genetic-level MC (mutation in the Hexa gene on human chromosome 15).[130] The MC at each level is a chief etiological factor of the related higher-level MCs.[131] HCV hepatitis illustrates the case of the etiology-specific sub-class that does not constitute a unique MC even though it does constitute a MK since the chief etiological factor is

[127] The idea that etiological factors may play a role in nosology is present in Murphy (2006).

[128] See http://www.curetay-sachs.org/about.shtml and http://ghr.nlm.nih.gov/condition/tay-sachs-disease

[129] It may also be the case that some etiology-specific sub-classes of hepatitis—viral hepatitis, alcohol induced steatohepatitis, non-alcohol induced steatohepatitis—constitute MCs since they just are malfunctioning parts from a lower-level (i.e., molecular level).

[130] It isn't clear to me whether the mutation on the chromosome is merely an etiological factor or a MC in its own right. After all, the genetic mechanism is not necessarily malfunctioning; rather, the "mutated" information is being processed and the output of this process, the proteins that do not perform their job properly and lead to a decrease of the hexodisamindase A enzyme, is what is malfunctioning.

[131] I say "chief" because it seems to be a necessary component of infantile Tay-Sachs. As Murphy (2006) notes, it would not be a sufficient factor as other background conditions will also be necessary, even if it is almost fully penetrant (e.g., the child will need to live to the age where the MC arises).

the HCV, which is not a malfunctioning biological part, that gives rise to the higher-level MC (e.g., hepatitis). Regardless of whether or not the etiology-specific sub-classes constitute MCs, noting these sub-classes in the nosology is justified because these classes constitute MKs (and, therefore, are likely to be interesting and useful to medical theorists) even if they do not constitute unique MCs.

In this section I have attempted to illustrate the various ways that MCs can be typed and the sort of nosology to which this typing practice gives rise. I have argued that MCs constitute MKs and that etiology-specific MCs also constitute MKs. Accordingly, many MKs will be nested since there are often a variety of different etiologies that give rise to a single MC. I then went on to note that some etiology-specific sub-class MKs may constitute unique MCs if the common causal mechanism of the sub-class just is a malfunctioning biological part. Such instances are likely to be quite common given that many MCs expressed at higher-levels (i.e., at the systems level) are actually caused by malfunctioning parts from a lower-level (i.e., malfunctions at the molecular or cellular level). In other cases, some or all of the etiology-specific sub-classes may not constitute unique MCs since the relevant causal mechanism will not always be a malfunctioning biological part—it might be, for instance, a virus, bacteria, or parasite.

A commonality among the previous cases is that the etiological factor plays a sustaining role in the MC—i.e., it plays a role in maintaining the MC by sustaining the malfunctioning of the relevant part. Accordingly, such factors are likely targets for treatment intervention.[132] It is for this reason, perhaps, that medical theorists recognize information about the etiology of a MC as important. But, it is not always the case that etiological factors play a sustaining role. In some cases, MCs enjoy a steady state existence—the malfunctioning part does not require the chief etiological factor to continue to malfunction. The MC cortical blindness caused by a blow to the

[132] Murphy (2006, 2009b) discusses etiological factors as potential targets of intervention.

head enjoys a steady state existence insofar as the etiological factor—i.e., the blow—is not required to sustain the malfunctioning part (i.e., the malfunctioning visual system). In cases such as this, it will probably be unnecessary to type according to etiology since intervening on the relevant etiological factor (e.g., the instrument that struck the head) is incapable of treating the steady state MC (e.g., the cortical-blindness). There may be other cases, however, where a MC enjoys a steady state existence, but it is still important to have information about the etiology since doing so can help to halt further disruption to the relevant part's function. Atherosclerosis, for instance, cannot be reversed but one can stop the condition from worsening. In order to do this, however, it is important to know something about the etiology-specific sub-classes of atherosclerosis since halting further disruption of the relevant biological part (i.e., stopping further hardening of the arteries) will require knowledge about how best to intervene on the relevant causal factor(s) (i.e., the actual etiological factor(s)). Accordingly, it may be useful to type atherosclerosis according to etiology even if it does enjoy a steady-state existence since the mechanisms that give rise to atherosclerosis are likely to vary by etiology (i.e., smoking, genetics, and diet are likely to give rise to atherosclerosis via distinct causal pathways). With these remarks on the role of etiological and pathological factors in typing MKs and developing a nosology completed, I now want to briefly consider the possibility that non-medical kinds may be of interest to medical theorists.

3.6. On the possibility of non-medical kinds of interest

Up to this point I have tried to drive home the idea that MCs and many etiology-specific sub-class MCs are MKs and that MKs are important for medicine understood as a partial-objectivist endeavor since they support P-laws of interest to medical theorists and figure in medical explanations in virtue of enjoying causal unity. It is important to explore, however, whether there are other classes which do not constitute MKs that are interesting to, and useful for,

medicine. If there is good reason to think that there are non-MKs of interest to medical theorists, then this would present a deep problem for my account since I take MKs to simply be those classes that are theoretically interesting and useful to medical researchers and physicians. Fortunately for me, there is not good reason to think this but there are reasons why one *may* think this. Let me explain what those reasons are and why they are not good reasons for thinking that there are non-MKs that are theoretically interesting and useful for medicine.

The first sort of class that one may be tempted to think illustrates that non-MKs are of interest to medical theorists are those classes alleged to figure in medical P-laws and medical explanations but whose causal unity remains in question.[133] I term classes that may constitute MKs but whose MK status is in question given a poverty of information about the class 'potential MKs'.[134] Many classes in their theoretical primacy may constitute potential MKs: medical P-laws and medical explanations in which the class figures have been proposed but little else is know about the nature of the class. As investigation continues, however, more will likely be learned about the classes—in particular, we will come to learn 1) whether the class actually does figure in any medical P-laws and medical explanations, and 2) whether the class is causally unified (i.e., whether it has a common malfunctioning biological part). The upshot of these considerations to the issue at hand is that there may be classes that are theoretically interesting for medicine that do not constitute MKs, but this may simply result from the primitive theoretical knowledge that we have about the class. That is to say, whether or not these classes are really of interest to medical theorists will hinge on whether they actually constitute MKs—i.e., whether they enjoy causal unity, support medical P-laws, and figure in medical explanations—suggesting

[133] These classes may track what are sometimes referred to as syndromes.

[134] It is likely that many 'potential MKs' will be treated as if they are MKs until proven otherwise since they support medical P-laws and figure in medical explanations. Where the burden of proof should rest—upon the one claiming MK status or the one questioning it—is a difficult matter. Regardless of where the burden lies, it seems quite clear that theorists have a duty to recognize defeasibility, respect findings, and accept defeating evidence (for more on this, see section 2.5.2).

that the sustained interest that medical theorists have in a class is intimately connected with the MK status of the class. It seems to follow, then, that all classes of interest to medical theorists will be MKs and potential MKs and interest in potential MKs will be dropped if the class is shown to not constitute an actual MK.

The above point argues that medical theorists are only interested in classes that constitute MKs. This is, in fact, a pretty weak claim given the fairly weak nature of MKs. Nonetheless, an opponent may argue that medicine must be interested in other sorts of knowledge besides that having to do with MKs if we recognize that things like cosmetic surgery and various sorts of enhancements are tasks that fall within the domain of medicine. This seems correct…medicine probably does have some interests that extend beyond MKs. I do not doubt this. What I am suggesting here is that medicine is not interested in *classes* that do not constitute MKs. The fact that medicine is interested in practices that are only questionably medical—i.e., things like cosmetic surgery and enhancements—is not, therefore, a strike against my claim. What would be a defeater is evidence that medical theorists maintain a theoretical interest in a condition even when they recognize that the condition does not enjoy causal unity, support medical P-laws, or figure in any medical explanations. Barring cases such as this, my claim seems justified.

3.7. Conclusion

I began this chapter by arguing for the P-scientific kind account. After presenting this account, I suggested that it outperforms, but is still consistent with, the HPC account of natural kinds. I then argued that MCs constitute MKs—i.e., the P-scientific kinds of interest to medicine. Next, I proposed that what matters when it comes to medicine is whether a class constitutes a MK. This led me to discuss the distinction between etiologies and pathologies in MKs and how such factors may be relevant to how we type MCs and how we develop a nosology. My final move was to consider the possibility of non-MKs of interest to medicine. With these remarks on MCs and

MKs out of the way, the goal of presenting my view of medicine—Partial-Objectivism—is completed. I now turn my attention to a discussion of Scientific Psychiatry.

Chapter 4: Scientific Psychiatry

4.1. Introduction

In this chapter I make use of the work on medicine and MKs from previous chapters to develop and discuss my account of psychiatry, Scientific Psychiatry (hereafter, SP). As already noted, SP is a term I employ to refer to an interpretation of psychiatry in accordance with the Partial-Objectivist view of medicine, one that sees mental disorders "as caused by distinctive pathophysiological processes in the brain" (Murphy, 2009, 103). Accordingly, psychiatry is to be seen as a branch of medicine that is theoretically grounded in the mind-brain sciences and whose subject matter is the malfunctioning mind-brain. While the terminology I sometimes employ is new, the general idea behind SP is well established in the work of theorists such as Samuel Guze (1989, 1992), Nancy Andreasan (1997), Eric Kandel (1999), Jacob Hohwy & Raben Rosenberg (2005), and Dominic Murphy (2006). Much of what I will have to say about SP derives from, and is consistent with, the work of these medical-cum-scientific accounts of psychiatry. Features that strike me as unique to SP, as well as major divergences from these friendly accounts, will be noted along the way.

The chapter proceeds as follows. In section two I discuss the confused nature of contemporary psychiatry. Section three describes the general theory of the mind with which I will be working. Section four lays out the commitments of the medical model. I begin with the general commitments, commitments presumably shared by all medically-based accounts of psychiatry, and then move on to the more specific commitments of the version of the medical model I employ. These more specific commitments are derived from the preceding work on partial-objectivism and MKs. Section five explores psychiatry according to my version of the medical model—SP. SP is described as a multi-level, inter-level discipline that is theoretically grounded in the mind-brain sciences and whose domain is the malfunctioning mind-brain. In this

section, I also spend time discussing the MKs of interest to SP—namely, mental medical conditions (MMCs)—and the sort of etiological factors that are uniquely relevant to these classes. The sixth section discusses SP's system of dual nosologies and then illustrates its revisionary nature by showing how it understands several "alleged mental disorders": major depression, obsessive-compulsive disorder, narcissistic personality disorder, and the paraphilias (e.g., pedophilia, bestiality, sadism, etc.). Section seven considers how SP might respond to Szaszian "anti-psychiatry" challenges, and section eight explores the therapies of relevance to SP. I conclude by proposing, in general agreement with Andreasan (1997) and Murphy (2006), that SP is best thought of as clinical mind-brain science.[135]

4.2. The difficult and confusing state of contemporary psychiatry

Before developing the SP account, it may be worthwhile to remind readers that contemporary psychiatry still finds itself in a difficult and confusing state even though there appears to be consensus among theorists that psychiatry is best understood as a branch of medicine. A primary way that this confusion presents itself is in the fact that psychiatrists are trained as medical doctors and seem committed to basing their theories about mental disorder in science, yet folk conceptions and evidentially unsupported theories about the mind and mental health are still allowed to play a theoretical role in psychiatry that they are not allowed to play in other branches of medicine (Murphy, 2006, esp. chapter 3; Paris, 2005). The major factor responsible for these conceptions and theories playing a role in psychiatry seems to be acceptance, whether explicit or implicit, of the belief that the mind is utterly distinct from the body (i.e., brain). Many take this belief to imply that the tools and techniques employed to understand the body—i.e., the theories and methods of medicine—are incapable of shedding light

[135] This does not mean that psychiatry will be nothing but clinical mind-brain science since there are likely to be sub-specialties (i.e., sub-practices within the medical practice of psychiatry)—i.e., perceptual psychiatry whose domain is one or more malfunctioning perceptual systems, executive function psychiatry whose domain is the malfunctioning executive system(s), memory psychiatry whose domain is the malfunctioning memory system(s), etc.

on how the mind works and how it can break down.[136] Acceptance of this belief seems to entail that psychiatry, the discipline focused on understanding how the mind breaks down, should require a different methodology and grounding than the medical disciplines focused on the human body.[137] Commitment to such ideas about the limited role of medical thinking and methods appear to have opened the door for unscientific and outdated theories about the human mind and mental life to play a role in psychiatric theory. At the very least, this view of the mind as utterly and deeply distinct from the body has led to the development of theories such as Freudian Psychoanalysis and Jungian Analytical Psychology, theories which were allegedly better suited to inform us about human psychopathology than anything we could learn from examining the human brain (see Freud 1920). Such views of the human mind continue to influence psychiatry, especially with respect to how psychiatric disorders are understood and treated, even though they are often lacking in evidential support. The difficult and confusing state arises for psychiatry when these evidentially suspect theories and outdated conceptions conflict with the scientific theory in which psychiatry, understood as a branch of medicine, should be grounded (Murphy, 2006).[138]

One can appreciate the state that results from this conflict by considering how contemporary psychiatry understands its own domain—i.e., understands what counts as a mental disorder or psychiatric condition. If psychiatry is to be scientifically grounded, then its domain of inquiry ought to be determined by the relevant grounding science(s)—e.g., the psychological

[136] Such ideas about the radical distinction between the nature of mind and body go back to ancient times and still persists in the population at large, perhaps as a result of our evolved cognitive systems (Bloom, 2004).

[137] This sort of reasoning is probably also responsible for the position that mental disorders cannot be brain disorders and *vice versa*. Indeed, it has led some theorists (e.g., Tomas Szasz) to claim there are no mental disorders since mental disorders are either brain disorders or they are just problems in living—i.e., they are different ways of living that should not be medicalized—neither of which are mental disorders. I consider Szasz's position in detail in section 7 of this chapter.

[138] Interestingly, no other branch of medicine appears to be hostage to folk-theory in the way that psychiatry is. It is also important to point out that one need not take on any particular metaphysical stance in order to respect the idea that medicine ought not to be influenced by non-scientific theories. The idea is simply that if medicine is to be scientifically grounded, then it ought to be grounded only in scientific theories.

and/or cognitive sciences, broadly construed. Accordingly, the sort of phenomena that psychiatry recognizes as falling within its domain *should* include the sort of phenomena in which these sciences are interested. It would stand to reason, then, that cortical blindness, a malfunction in the visual system resulting from an insult to the occipital cortex, should count as falling within the domain of psychiatry since contemporary psychological science recognizes the visual system as a mental system *par excellence* (Murphy, 2006). Contemporary psychiatry as represented by the DSM-IV TR, however, does not treat cortical blindness, or most other disruptions to perceptual systems, as psychiatric conditions.[139] Psychiatry's confused state is further revealed by the fact that contemporary psychiatry not only leaves out many conditions that seem to fall squarely within its domain as determined by the relevant science(s), it also includes many conditions that it seems ought not to be included. After all, many alleged mental disorders such as histrionic personality disorder, explosive personality disorder, conduct disorder, and narcissistic personality disorder appear to be "problems in living" and not actual mental disorders, insofar as individuals may enjoy these "conditions" even when there is no malfunction in the mental systems recognized by contemporary psychological science (Szasz, 1960; Murphy, 2006).[140]

Some may doubt that the situation described above is an indication of difficulty or confusion in contemporary psychiatry. Indeed, many are likely to be shocked by proposals to widen the domain of psychiatry to include conditions like cortical blindness while removing the many personality disorders which have a long history of being considered psychiatric conditions. In response, however, we might ask those who defend contemporary psychiatry's chosen domain to provide a principled reason for its determination. That is just to say, we might ask for an

[139] Apahsias provide several more examples of phenomena that are recognized as involving psychological systems but are not seen as mental conditions or mental disorders insofar as they do not appear in the DSM-IV TR.

[140] "Problems in living", a concept borrowed from Tomas Szasz (1960), are "conditions" that result from the difficulties of daily life but do not issue from a malfunctioning mental part. They are, allegedly, the result of the basic fact that life is sometimes hard. I discuss "problems in living" in detail in section 4.7.

explanation of how the domain of psychiatry, as it is represented in the DSM-IV TR[141], for instance, is determined. It is my suspicion that the domain is as influenced by outdated theories about the mind and mental disorder as it is by scientifically grounded theories.

What I have just offered is a psychological explanation of psychiatry's confused state.[142] There is also an historical explanation on offer. The Nobel Prize winning neuroscientist Eric Kandel shares his insights on how understanding the history of psychiatry can help us understand its present situation. He writes

> [i]n the years following World War II, medicine was transformed from a practicing art into a scientific discipline based on molecular biology. During that same period psychiatry was transformed from a medical discipline into a practicing therapeutic art. In the 1950s and in some academic centers extending into the 1960s, academic psychiatry transiently abandoned its roots in biology and experimental medicine and evolved into a psychoanalytically based and socially oriented discipline that was surprisingly unconcerned with the brain as an organ of mental activity…Moreover, as the limitations of psychoanalysis as a system of rigorous, self-critical thought became apparent, rather than confronting these limitations in a systematic, questioning, experimental manner, and perhaps rejoining biology in searching for newer ways of exploring the brain, psychoanalytic psychiatry spent most of the decades of its dominance—the period from 1950 to 1980—on the defensive. Although there were important individual exceptions, as a group, psychoanalysts devalued experimental inquiry. Consequently, psychoanalysis slid into an intellectual decline that has had a deleterious effect on psychiatry, and because it discouraged new ways of thought, it has had a particularly deleterious effect on the training of psychiatrists. (1998, 458)

Kandel's remarks remind us that the struggles which psychiatry faces are at least partially the result of historical circumstances.[143] Importantly, however, the reason that non-medical psychiatric approaches were chosen over their medical competitors was probably tied to the

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[141] At the time of writing, the DSM-V was being released. For the most part, the comments here apply to the DSM-V. See Addendum on the DSM-V for more on this.

[142] This recounting is deeply influenced by Murphy's work on the state of contemporary psychiatry (2006).

[143] Murphy (2006) also offers a detailed explanation of the historical causes responsible for contemporary psychiatry's confused nature.

rather resilient and perennial belief concerning the radical distinction between mind and body that I discussed earlier. To be sure, there were some early attempts to understand psychopathology and psychology in biological/medical terms but these projects seem to have been abandoned in favor of approaches that embraced the uniqueness of mind (e.g., Freud's rejection of his earlier neurological approach to psychology—i.e., that of his 1895 unpublished manuscript—for psychoanalysis). Kandel's historical explanation of psychiatry's confusing state is valuable nonetheless as it can help us better appreciate the sort of outdated concepts and theories that are probably still influencing contemporary psychiatry.

Another cause of the confusing state is a general lack of understanding among theorists and the public of the relationship between evidence, prediction, and theory confirmation. The fact that some of the treatments and therapies proposed by non-medical psychiatric approaches actually did prove efficacious probably made psychiatry's situation even more difficult since proponents of these non-medical approaches (esp. proponents of psychoanalysis) often took the efficacy of the therapies as evidence of the truth of the theories the treatments were built upon, theories that are now taken to be mistaken (e.g., Freud's psychosexual theory of mental disorder). Of course, the fact that a theory (e.g., the psychosexual theory) entails some outcome (e.g., psychoanalysis will be efficacious) and the outcome is found to be the case (e.g., psychoanalysis is efficacious) does not necessarily mean that the theory (e.g., the psychosexual theory) is true for the outcome may be consistent with any number of other, non-identical theories.[144] Accordingly, that a therapy or treatment works is no guarantee that the theory the therapy is built upon is true.[145] Unfortunately, not everyone recognizes this important fact about the relationship between theories, prediction, and evidence. As a result, the efficacy of these therapies/treatments was, and

[144] See Quine (1951) and Duhem (1914) on under-determination.

[145] In section 4.8.c I will have more to say about how and why the efficacy of a treatment or therapy does not necessarily count as evidence in favor of the psychological theory upon which the therapy/treatment is based.

often continues to be, taken as confirming evidence of these suspect theories of psychopathology and these theories, in turn, continue to influence psychiatry.

While the above reasoning may explain why non-scientific theories have played a role in psychiatry in the past, it fails as an apology for contemporary psychiatry's employment of non-scientific theories for two reasons. First, even if science could not inform us about our mental lives, this would not be a reason to think that our commonsense conceptions, or non-evidentially supported theories, about the human mind and mental disorder are correct. Before we allow non-scientific theory such a powerful role, we will need an independent argument that it can, in fact, provide explanations where science allegedly fails. At present, no such argument appears to be available. Second, we actually do have good scientific theory about the human mind and mental life in the form of mind-brain science and we can use this to gain an understanding of mental disorder and mental health that is grounded in scientific theory. The upshot of this second point is that psychiatry need not continue to live its conflicted life: it can happily reject grounding in folk theory and unsupported psychological theories and opt to ground itself in the mind-brain sciences. Making such a move is tantamount to re-interpreting psychiatry according to the medical model.

As already noted, most theorists (though certainly not all) already seem to accept that psychiatry *ought* to adhere to some sort of medical model. So just arguing that such a position *ought* to be accepted does not really seem to have the sort of theoretical upshot necessary to deal with the confusion that I have just been discussing. Rather, what is needed is a medical model account of psychiatry that succeeds where others have failed—that is, what is needed is a model that offers theoretical simplicity and consistency instead of the difficulty and confusion that plaques contemporary psychiatry. In the remaining chapters I offer such an account. But before I do this, more should be said about the view of the mind we will be assuming.

4.3. On the mind and brain

In the previous section I suggested that commitment to a certain view of the mind-brain, namely, one that sees the two as utterly distinct, is an outdated theory that is partially responsible for psychiatry's confused state. I also argued that such a view ought not to play a role in a scientific psychiatry. These claims raise the question, what is the view of the nature of the mind and the relationship between the mind and brain this project assumes? This section offers an answer to that question.

Theories about the nature of mind and how the mind is connected or related to the brain arise from the idea that humans possess both physical and mental properties. Physical properties are properties that are recognized by contemporary physics. They include things like mass and extension. The alleged existence of a mind and mental states are taken as evidence that there are mental properties. Numerous theories about the nature of mind have been proposed. I review four here: dualism, idealism, reductive physicalism, and non-reductive physicalism.[146]

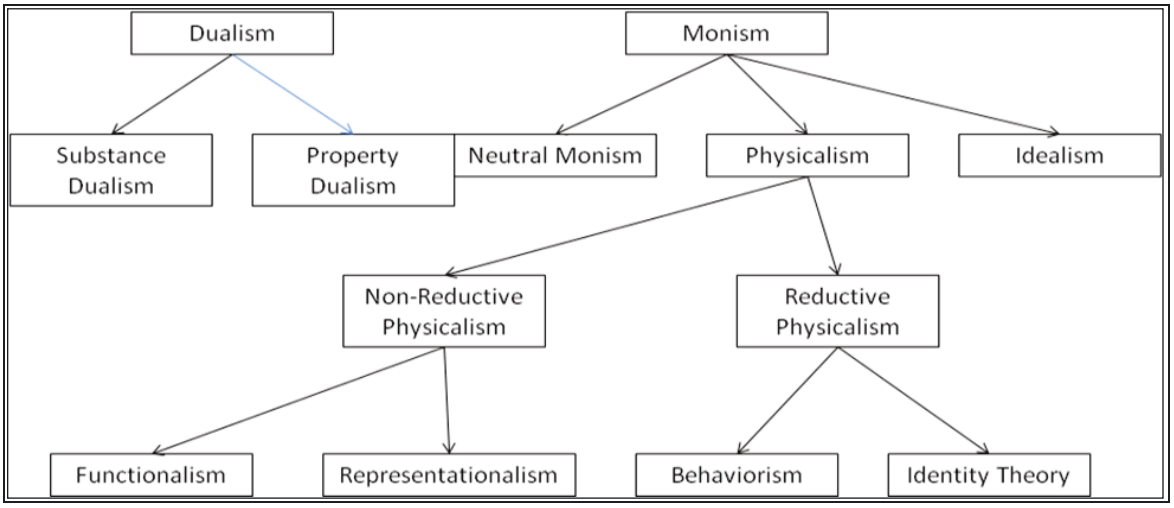

Figure 4.1. Figure illustrating philosophical accounts of the mind.

[146] There are often multiple accounts within each theory (e.g., psycho-neural identity theory and ontological behaviorism are two other reductive physicalist positions). I will only discuss some of these positions in order to avoid being sidetracked.

The dualist theory that I rejected in the last section is what might be called common-sense substance dualism, a position that holds the mind and body (i.e., brain) to be distinct substances with distinct properties. As previously discussed, the greatest evidence for this claim is the intuition that the mind must be different from the body (i.e., that mental properties must be different from physical properties).[147] Though intuitions may suggest that mind and brain are distinct, they also suggest that they are connected. After all, insults to the brain often cause changes in mental states and neuro-imaging technology can show mental states correlating with brain states. Given substance dualism's stance, it will need to explain how two radically distinct substances are able to causally interact. As of now, a satisfactory explanation is wanting and that is probably why the mind-brain sciences do not accept a dualist theory of the mind and brain. Another problem with substance dualism is that it is unclear what sort of substance the mind might be if it is not physical. This uncertainty does not mean, of course, that dualism is false. But, it makes dualism an unattractive option if one is committed to a methodological naturalism that only allows objects/entities recognized by our best current science.

In contrast to dualist theories, monistic theories posit that there is only one type of "stuff" in the universe. There are several theories of mind that are committed to monism. Idealism is a monistic theory that holds everything is ultimately reducible to "ideas" or the mind. Explaining the nature of the mind is not a problem for idealism since it holds the mind (and mental properties) to be fundamental. Idealism, however, is an unsatisfactory account of the mind for our project since our commitment to methodological naturalism requires that we only accept claims that are consistent with our best current science. Because contemporary science does not accept idealism, this project will also not accept it.[148]

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[147] There are philosophical arguments for dualism that do not hinge on empirical evidence that I will not cover.

[148] This project does not reject idealism because it thinks it is wrong as a metaphysical position; rather, it rejects idealism because it is not recognized by contemporary science. If contemporary science were to recognize idealism as the proper description of reality at some future point, then this would be the theory of mind that would constrain SP.

A more likely monistic theory for our purposes is physicalism. At its core, physicalism holds that all processes, events, and entities in the universe are, or arise from, physical processes, events, and entities. Physicalist theories of mind, therefore, understand the mind (i.e., mental states) to arise from, or be identical to, physical states (i.e., states characterized by physical properties). There are two broad categories of physicalist theories: reductive and non-reductive. Reductive theories hold that mental states can be reduced to physical states, while non-reductive theories argue that even though mental states are physical states, they cannot be reduced to them (Kim, 1998; Heil, 2004).

The identity theory is a reductionist theory of mind that posits mental states, processes, and events just are brain states, processes, and events. Early proponents often argued for the identity theory on grounds of parsimony (Smart, 1959, 2012). For example, they would point out that pain behavior results from c-fiber stimulation and c-fibers are stimulated by pain causing events like tissue irritation. They would then reason that the most parsimonious explanation of these facts is that pain is identical with c-fiber stimulation. These proponents assumed that all mental states—beliefs, desires, etc.—are identical with brain states in a similar manner. A problem with the identity theory is that many, if not all, mental states appear to be multiply realizable (Putnam, 1967; Fodor, 1974; Bickel, 2013). Multiple realizability holds that a single mental state can be realized or implemented in a number of different physical (and, perhaps, non-physical) mediums. If multiple realizability is correct, then mental states cannot be identical to brain states since a single mental state can be implemented in different brain states.

Recognizing the limits of the identity theory, many philosophers now accept a non-reductive physicalist theory of mind.[149] Non-reductive physicalism has two core commitments:

[149] Functionalist theories of mind, a common strain of non-reductive physicalist theories, take mental states and processes to be functional states that are defined by the causal roles they play. Though most functionalists recognize that these states are implemented in physical processes (i.e., brain states), they deny they are identical with brain states. Rather, they are abstract states that play a role that can be realized in a number of different systems/mediums (Heil, 2004).

1) mental states are physical states, and 2) mental states cannot be reduced to physical states. Non-reductive physicalists posit a supervenience dependency relationship between the physical and the mental in order to make these two seemingly contradictory claims consistent. If true, the supervenience of the mental on the physical entails that there can be no change in the mental without a change in the physical. It does not, however, entail that the mental can be reduced to the physical for this requires that each mental state type (e.g., belief) be identical with a physical state type (e.g., a certain pattern of neural activity), and supervenience only insures that each mental state token (e.g., a particular belief in a particular person) is identical to a physical state token (e.g., a particular pattern of neural activity). As Fodor (1974, 1997) notes, to get the type identity necessary for reduction, we would need bridge laws that allow the laws of psychology to be derived from the laws of physics (see Nagel, 1961). The problem, Fodor contends, is that there are no bridge laws (i.e., statements expressing a contingent identity between mental and physical predicates) because of the multiply realizable nature of the mental. The non-reductive nature of the mental has not gone unchallenged (see Kim, 1992; Bickle, 2013).

The problem with non-reductive physicalism is that some have taken it to suggest that investigation of the physical (namely, the brain) is unlikely to tell us anything about the mental since the mental is not reducible to the physical. This line of reasoning is what prompted Fodor to argue for a strong form of special science autonomy—i.e., the idea that psychology would not, and could not, be informed by the findings of the neurosciences (1974, 1997). A cursory glance at work in the mind-brain sciences ought to be enough to convince anyone that Fodor was wrong. Indeed, a central assumption of current scientific attempts to understand the mind-brain is that we can use findings from the various mind-brain sciences to inform and constrain each other (McCauley & Bechtel, 2001). Because our project will take on the mind-brain sciences' view of the mind, it will not accept any version of the non-reductive physicalist position that entails strong autonomy of the special sciences.

Given our desire to adopt a view of the mind consistent with that of the mind-brain sciences, we might gain an understanding of the view we should assume by looking at how these sciences understand the mind. Because these are empirical sciences, they seem to accept, at least as a methodological principle, that the fundamental nature of the universe is physical.[150] Accordingly, they will recognize some sort of physicalist account of the mind. These sciences also seem to recognize that the mind has special properties—i.e., computational, informational, and intentional properties—that may not be able to be reduced to standard physical properties. But, unlike most non-reductive physicalist accounts, the mind-brain sciences do not appear to allow this to stop them from using the findings from the neurosciences and biology to inform and constrain their theories about the mind or the findings from psychology and computer science to inform and constrain their theories of the brain (McCauley & Bechtel, 2001). Another point to consider is that the mind-brain sciences tend to employ the practice of decomposition and localization to explain mental functions (Bechtel & Richardson, 1993; Bechtel, 2002). This process involves breaking mental functions down into component functions and then localizing these component functions in specific parts of the brain. Multiple iterations of decomposition and localization may be carried out in order to provide as complete an understanding of the function as possible. Mental functions that have been successfully decomposed and localized are often said to be explained and/or reduced. Employing this explanatory strategy presupposes a commitment to a heuristic identity theory, a theory that proposes identity between the mind and brain (i.e., mental states and brain states) as a heuristic to further scientific understanding of the mind and the brain (Bechtel & McCauley, 1999; McCauley & Bechtel, 2001).

Given these features of the mind-brain sciences, a picture of the mind that this project accepts, and SP will be constrained by, emerges. First, it is a view that is committed to a version

[150] An interesting possibility is that physics could, perhaps, come to recognize the existence of mental properties, along with physical properties, as fundamental. This position, something akin to property dualism, would be a physicalist position insofar as it defers to physics' claims about the ultimate nature of reality.

of physicalism.[151] Second, while it may not be committed to a strict identity holding between the mind and brain, the view does seem, at the least, committed to a heuristic identity holding between the mind and brain, a claim supported by the use of decomposition and localization as an explanatory strategy. For the remainder of this project, I will be assuming that the view of the mind that SP is constrained by is consistent with these ideas: physicalism, heuristic identity, and the possibility of non-reductive elements.

4.4. The Medical Model

Over the past few decades, many theorists have recognized that the time has come to put psychiatry on a firmer scientific footing in order to free it from the theoretical conflict that has led many to construe it as a dubious medical practice (Guze, 1989; Andreasan, 1997; Kandel, 1999; Murphy, 2006). Proponents of this strategy are often driven by the desire to re-interpret psychiatry according to the medical model. In its most basic form, this reconceptualization involves an application of "medical thinking and methods" to psychiatry with an emphasis upon medicine's commitment to diseases being "pathological states located in body tissue" (Black, quoted in Murphy, 2009b, 104). Accordingly, proponents of the medical model see biological theory as central to psychiatry, an idea that Samuel Guze explains nicely when he writes

> psychiatry must turn increasingly to biological science. Cultural anthropology, sociology, philosophy, and religion may all have important contributions to make to the understanding of psychopathology and its treatment, but these disciplines too will have to take into account human biology if they are to be of maximum use. The nature and development of mental functions is the centre of psychiatric interest, just as the nature and development of bodily functions generally are the centre of medical interest. Psychiatry is a branch of medicine, which in turn is a form of applied biology. It follows, therefore, that biological science, broadly defined, is the foundation of medical science and hence of medical practice. The other disciplines can and must make their contributions but they cannot displace biology from its critical role. (1989, 319)

[151] The commitment to physicalism follows from a commitment to methodological naturalism rather than a commitment to ontological naturalism. After all, it could be the case that humans do enjoy some sort of non-physical component. But, if they do, then I take it that this will not fall within the domain of the mind-brain sciences. Furthermore, I take it that this non-physical component would not count as a mental part.

My work in this chapter is another attempt to embody Guze's claim and ground psychiatry in biology as I reinterpret it according to the medical model. The reinterpretation I offer outperforms other medical model accounts, I propose, because it employs the Partial-Objectivist view of medicine that I developed in chapter two and three as the framework in which to re-conceptualize psychiatry. I label the view that emerges from this reconceptualization Scientific Psychiatry (SP) to emphasize its objective grounding in scientific theory. In the next few sections I spell out the main theoretical commitments of SP. But first, let me offer a brief review of the core features of the Partial-Objectivist view of medicine.

4.4.a. The partial-objective aspect

In chapter two I argued that medicine (for the most part) is best described as being interested in the restoration, maintenance, and improvement of health, where health is understood as freedom from disease and diseases are understood to be partially objective bodily states. I went on to argue that this understanding of medicine, Partial-Objectivism, holds that core medical practices—i.e., medicine—understand diseases to be partially objective states since they have both an objective and a subjective component. The objective component is that the state must be an abnormal biological state, while the subjective component is that the state must be deemed harmful or undesirable by a culture or society. Abnormal biological states occur when a biological part is E-systemically-malfunctioning. The E-systemic-function of a part is that function which the part actually contributes to the organism's capacity to survive and reproduce. Accordingly, an E-systemically malfunctioning part constitutes an abnormal biological state. Medical condition (MC) is a term that I introduced to refer to the purely objective component of diseases—i.e., the dysfunctional biological part/abnormal biological state. My claim in chapter two was that medicine is implicitly, if not explicitly, committed to partial-objectivism about disease. The general idea that diseases are partially objective in the sense that they are, necessarily, biologically abnormal states is widely accepted by medical theorists even though

there is disagreement about how medicine understands biological normalcy (Boorse, 1977; Wakefield, 1992; Murphy, 2006; Ereshefsky, 2009). Partial-Objectivism differs from other objectivist accounts of medicine primarily as a result of its understanding that biological normalcy should be cashed out in terms of normal biological function which should be understood in terms of E-systemic function.

Another feature of Partial-Objectivism is its commitment to the two-stage view of disease. As previously discussed, the first stage involves identifying the E-systemically malfunctioning biological part—i.e., the MC. The second stage involves the MC being deemed harmful or undesirable by the relevant community or social group. MCs are the output of first stage work and the input for second stage work. Stage one work limits stage two work since only MCs are candidates for disease status—i.e., stage two work only ranges over the output of stage one work. Allowing stage one output to determine the input for stage two work is what insures the Partial-Objectivist nature of medicine—i.e., it is what insures that diseases are partially-objective states.

An additional consequence of Partial-Objectivism's commitment to the two-stage view is that, as *scientific* practices, medical practices are focused solely on stage one work, understanding the nature of MCs. The output of this purely scientific aspect of these practices can be employed as input for a variety of stage two projects (Murphy, 2006). One of the most important stage two projects is the one that asks which MCs are harmful or undesirable in the sense that they threaten people's ability to survive. The output of this project, which falls within the domain of medicine, is an inventory of diseases—i.e., a nosology. Importantly, even though this particular stage two

project falls within the domain of medicine, the task of determining what counts as harmful or undesirable could require input from outside of this domain.[152]

Partial-Objectivism understands MCs to constitute MKs. Such kinds enjoy causal unity, support medical P-laws, and figure in medical explanations. These kinds are typed along two dimensions—pathology and etiology—and these dimensions are represented in the nosology. Pathology is the primary typing feature, while etiology is often employed for sub-typing. This typing strategy insures that MKs will enjoy enough causal unity to support medical P-laws, a feature that insures these kinds can be used by medical theorist for the purposes of prediction, explanation and intervention. Because only MCs are candidates for disease status, diseases will also constitute MKs.

4.4.b. the multi-level, inter-level aspect

Partial-Objectivism recognizes that understanding MCs is a multi-level and an inter-level endeavor. Medicine is multi-level in the sense that it does not necessarily privilege one level of explanation over another. It follows functional biology's practice of investigating the human body at a variety of levels: the genetic, molecular, cellular, and systems levels. Medicine is inter-level in two ways. First, because the hypotheses and explanations concerning medical conditions will often span levels the approach is inter-level (Craver 2007; Murphy, 2008). An example of this aspect of the inter-level nature of medical theorizing is to be found in any hypothesis that aims to explain personal level phenomena such as a cough or a fever in terms of a cellular or molecular malfunction (Schaffner, 2008). Other examples include the explanation of systemic breakdowns in the body (i.e., organ malfunctions) as the result of genetic or molecular malfunctions. The second way in which medicine is inter-level has to do with the fact that theories at one level of

[152] When our understanding of what counts as harmful and dangerous shifts, there is likely to be a shift in the inventory of diseases—i.e., different notions of "undesirable" will mean that different MCs count as diseases. The inventory of MCs, however, does not vary as a function of how we understand "harmful" and "undesirable".

explanation—e.g., the systems level—could be informed and, perhaps even constrained, by theories at a lower level (McCauley & Bechtel, 2001). If we learn, for example, that the liver can malfunction as a result of two different types of cellular break down, then this would likely inform the way we think about liver malfunction.

4.4.c. the basis in functional biology aspect

Partial-Objectivism recognizes that medicine aims to be grounded in science, in particular, functional biology. The idea behind this claim is that the normal E-systemic functioning of biological parts needs to be understood in order to appreciate when a part is E-systemically malfunctioning. Since functional biology is the branch of biology tasked with understanding such functions, it follows that medical theorizing is grounded in this sort of biological theory. That medicine is grounded in such biological theory is another point that is widely accepted by most objectivists about medicine (Boorse, 1977; Murphy, 2006, 2009; Ereshefsky, 2009). It is accepted for good reason—a cursory glance at the actual practices of medicine reveals that theorizing about MCs is informed by an understanding of the functions of biological parts. Cardiology, for instance, is essentially clinical biology of the circulatory system—it seeks to understand how to restore, maintain, and improve the health of the circulatory system and to do this it needs to understand how a non-malfunctioning—i.e., an E-systemically functioning—circulatory system works. Knowledge about the properly functioning circulatory system comes from functional biology. Dermatology, another medical practice, is also grounded in functional biology insofar as its understanding of how the skin system can malfunction is informed by an understanding of how the skin system actually works at a number of levels.

4.4.d. the proper domain of individual medical practices

A final feature of partial objectivism that has yet to be discussed centers on how the domains—i.e., the spheres of concern—of specific medical practices are determined. Given

partial-objectivism's grounding in functional biology and its emphasis on medicine being primarily concerned with understanding and treating MCs, it follows that the domain of a specific medical practice is the malfunctioning biological part in which the practice is interested.[153] This does not mean that these practices are not interested in understanding the normal function of biological parts. After all, a good appreciation of how a part works is often necessary to understand how it can breakdown and what can be done to restore function.[154] Accordingly, the domains of medical practices will actually include both functioning *and* malfunctioning biological parts. The domain of orthopedics, for example, is the musculoskeletal system with an emphasis on understanding and treating MCs in this system, while the domain of primary care practice is the whole person understood as a biological system with an emphasis on understanding and treating MCs that occur in this system (i.e., the person). Because primary care practitioners seek to "understand and treat" MCs that occur within the whole person they can be said to enjoy a wide domain—i.e., their sphere of concern is large, relatively speaking, since it is the human biological system as a whole. The wide domain of primary care practitioners means that they are likely to lack the specialized knowledge that other specialist practitioners enjoy since they must have some scientific understanding of all parts of the human and how these parts can break down. This is why primary care practitioners will often refer patients to specialists when more in depth knowledge is needed in order to diagnose and/or devise a treatment plan. Specialists, such as orthopedists, cardiologists, hematologists, etc. have relatively small domains compared to primary care practitioners since they tend to focus on malfunctions in specific parts of the human. The narrower focus allows specialists to gain a more robust and in-depth scientific understanding

[153] Medical practices have both research and therapeutic components: the research component of a medical practice centers on understanding the domain scientifically (e.g., the research component of oncology has to do with understanding the malfunction in cells—i.e., cancer—and how to treat this malfunction), while the therapeutic component is the part of the practice that deals with the actual treatment of patients.

[154] The claim that medical practices are concerned with malfunctioning biological parts—not just biological parts—issues from the idea that medicine is especially interested in understanding and treating MCs and not simply understanding biological parts.

of their domain. This is why the cardiologists' knowledge of the human circulatory system, how this system can malfunction, and what can be done to restore function is unlikely to be matched by any other medical practitioner, though their knowledge of other biological parts/systems (e.g., the musculoskeletal system, the nervous system, etc.) and how they malfunction and can be treated probably pales in comparison to that of the specialists of these systems and perhaps even to that of the primary care practitioner.

That the particular medical practices exist that actually do is most likely a matter of historical fact that may reflect nothing more than social convention. After all, there doesn't seem to be any obvious reason why there is a medical practice devoted to the foot (i.e., podiatry) but not one for the hand.[155] Another interesting fact about these domains is that they are not necessarily fixed or clear. As biological theory advances, and as science gains an appreciation of the various interconnections among parts of the body, one is likely to see the domains of the various medical practices change. If, for example, we discovered that the foot had some intimate connection to liver function, then understanding this connection and certain related aspects of liver functioning would fall within the domain of podiatry since malfunctions in the foot could be connected with malfunctions in the liver.

It is also important to understand that the above claims are not meant to suggest that only the biological sciences are ever theoretically relevant to medicine since it may turn out that non-biological sciences and methods have a role to play in helping us understand the function of biological parts and MCs. Accordingly, many medical practices may draw from a variety of non-biological sciences in order to better understand their domain. Orthopedists, for instance, are probably expected to understand quite a bit about the mechanics of locomotion (i.e., physics) since this knowledge is likely to prove important in understanding the human musculoskeletal

[155] Though there are orthopedists specializing in the hand.

system and how it malfunctions. Whether or not a science or methodology is relevant to a given practice (i.e., informs the practice's understanding of its domain) seems to hinge on whether there is empirical evidence that the science actually does help us to better understand the practice's domain—i.e., the malfunctioning biological part of interest. That there needs to be empirical evidence of the "science's" usefulness explains why we do not allow scientology to play a role in medicine—there is no empirical evidence that scientology helps us to better understand the function or malfunction of any biological parts. Physics' and chemistry's ability to do this, on the other hand, is well supported by evidence and this explains why these sciences, often in the form of biophysics, biochemistry, and neurophysiology, inform medical practices.

As already noted, almost all of the previously mentioned features of partial-objectivism are consistent with other objectivist views of medicine. The one area where there is likely to be divergence is in the way that biological normalcy gets cashed out. But as I argued in chapter two, understanding biological function as E-systemic function seems to most fully capture the sense of function medical theorists employ when reasoning about the biological abnormalities involved in MCs and diseases. With this review out of the way, I now want to examine what psychiatry looks likes if we understand it to be a branch of medicine, where medicine is understood according to Partial-Objectivism.

4.5. Scientific Psychiatry

Scientific Psychiatry, in virtue of being committed to my medical model, holds that psychiatry is a branch of medicine. Accordingly, SP sees psychiatry as the branch of medicine that focuses on the maintenance, improvement, and restoration of mental health where mental health is understood as freedom from mental diseases and mental diseases are understood to be partially objective bodily states. Like medicine in general, SP has a purely objective project, namely, developing a better understanding of the MCs that occur in the human mind-brain (i.e.,

mental medical conditions), where the mind-brain is understood to be a part of the human body. In the remainder of this section, I spell out the theoretical commitments of SP.

4.5.a. SP's domain: the malfunctioning mind-brain

Traditionally, psychiatry has been understood to be interested in breakdowns in mental health—i.e., "mental disorders". A corollary of this view is that the proper domain of psychiatry is determined by appreciating the nature of the mental and the ways that the mind can breakdown (Murphy, 2006). Unfortunately, as discussed earlier, the domain of psychiatry—i.e., its sphere of concern—has not changed in step with changes in our understanding of the nature of disease and the nature of the mental. SP is explicitly committed to the idea that the domain of psychiatry should be tied to our best scientific understanding of the mind and the mental given that psychiatry is a practice that is primarily focused upon the various ways that mental functioning can break down. This move is in line with the Partial-Objectivist approach to understanding a practice's proper domain. So what does contemporary science concerning the mind have to tell us about the domain of psychiatry?

Contemporary science's understanding of the mind and mental life has evolved to the point that it is now commonplace to speak of the "mind-brain" as the organ of mentation (broadly defined to include cognition, perception, etc.). Accordingly, the domain of psychiatry is the broken-down—i.e., malfunctioning—mind-brain.[156] The sciences tasked with helping us better understand the mind-brain are what are commonly referred to as the mind-brain and/or cognitive sciences.[157] These include, but are not limited to: genetics; molecular, cellular, and systems neurobiology; behavioral and cognitive psychology; computer science; anthropology; linguistics.

[156] This move, regarding psychiatry's domain, is employed by most committed to the medical model. See Andreasan (1997) and Murphy (2006).

[157] I use 'cognitive sciences' and 'mind-brain sciences' interchangeably. I tend to employ 'mind-brain science' since some may resist taking motor and somatosensory processes to fall within the domain of the cognitive sciences.

The move to investigate the mind-brain via these many sciences does not entail a demotion of the role of biology in understanding the mind insofar as the mind-brain is understood to be a biological system (though one that may have special properties not enjoyed by other biological parts) and the normal functions of mental parts—i.e., parts of the mind-brain—are understood to be the E-systemic functions of these parts. The relevance of the multitude of sciences, some of which are not biological, issues from the increasingly recognized fact that, when it comes to science at least, the mind is not a faculty or organ that can be easily divorced from the brain and that the mind-brain can be best understood if we employ a multitude of sciences to investigate it in a number of different ways (i.e., anatomically, behaviorally, etc.) and at a number of different levels (i.e., molecular, cellular, systems, computational, personal, etc.) (McCauley & Bechtel, 2001; Craver, 2007). Because the mind-brain sciences appear to be interested in investigating the above mentioned systems at a number of different levels (i.e., genetic, molecular, cellular, system, personal) and using a variety of methods (computational modeling, lesion studies, behavioral studies, etc.), we should also expect SP to employ a wide range of methodologies and be interested in break-downs in the mind-brain at a variety of levels. That is to say, we ought to expect the methods and theories of the various mind-brain sciences to be potentially relevant to SP.

To more fully appreciate the domain of psychiatry, it may prove useful to consider the activities—i.e., E-systemic functions—of the mind-brain since disruptions in these activities are indicators of malfunction. The easiest way to garner an understanding of the mind-brain's activities is to consider the sorts of "stuff" the contemporary mind-brain sciences investigate.[158]

[158] Nancy Andreasan has the following remarks on the domain of the cognitive sciences: "The study of mind has been the province of cognitive psychology, which has divided mind into component domains of investigation (such as memory, language, and attention), created theoretical systems to explain the workings of those domains (constructs such as memory encoding versus retrieval), and designed experimental paradigms to test the hypotheses in human beings and animals. The study of brain has been the province of several disciplines. Neuropsychology has used the lesion method to determine localization by observing absence of function after injury, whereas neuroanatomy and neurobiology have mapped neural development and connectivity and studied functionality in animal models. The boundaries between all these disciplines have become increasingly less distinct, however, creating the broad discipline of cognitive neuroscience. The term 'cognitive' has definitions that range from broad to narrow; its usage here is broad

It seems clear that cognitive states and the machinery underlying these states are of interest to the mind-brain sciences so breakdowns in cognition fall within SP's domain. Such breakdowns include disturbances to personality, decision-making, reasoning, etc. In addition to cognition, SP is interested in disruption to affect since the mind-brain sciences recognize the mind-brain to be the seat of affective states and processing. Given the role of the mind-brain in perception, disruptions to perceptual states and systems fall within SP's domain. Dysfunctions in memory and attention systems, systems that straddle the border of cognition and perception, also fall within SP's domain since the mind-brain sciences recognize these to be systems located in parts of the mind-brain. Motor and somatosensory dysfunctions that result from breakdowns in the motor neuron system (both upper and lower) or the somato-sensory system are within the domain of SP.[159] In short, SP's domain is the malfunctioning mind-brain which is realized whenever there is a dysfunction (i.e., E-systemic malfunction) in one or more perceptual, cognitive, affective, somatosensory, motor, attentional and/or memory systems.[160] In section 4.5.b I will have more to say about mental medical conditions (hereafter, MMCs), mental disorders, and E-systemic malfunctions in the human mind-brain.

For now, however, it is worth noting that contemporary psychiatry already seems to recognize that many of the previously mentioned types of phenomena are within its domain.[161]

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and refers to all activities of mind, including emotion, perception, and regulation of behavior." (Andreassan, 1997, 1568)

[159] It is probably worth pointing out that all of these systems and processes—affective, perceptual, cognitive, motor, sensory, memory, attentional—are likely deeply connected—e.g., affect probably plays a role in cognition (Dimasio, 2008), motor functions may also play a role in cognition and perception (Noe, 2004; Gibson, 1979; Bortolotti, 2010), etc.

[160] The investigation of what can, and does, go wrong with the systems, mechanisms, and processes discussed in these sciences, as well as the exploration of which methods and means are most useful in restoring normal mental functioning, appears to fall within the domain of SP insofar as SP is the branch of medicine interested in break-downs in normal mental functioning. Following Murphy, SP understands mental disorders to be those "with an etiology or symptomatology involving those properties or phenomena falling into the scope of the sciences of the mind" (2009, 61).

[161] Psychiatrists are required to pass board exams for "Neurology and Psychiatry" in order to practice.

What is lacking at the moment is consistency (Murphy, 2006). The inconsistency is evidenced by the fact that only some breakdowns in perception and only some disruptions to affect are counted as psychiatric conditions even though there appears to be widespread agreement among mind-brain theorists that affect and perception constitute mental states. The explanation for this discrepancy, as Murphy has clearly argued and I noted in section 4.2, is that the actual domain of psychiatry appears to be governed by forces beyond scientific reasoning (2006). SP, like some others, attempts to resolve these inconsistencies by taking the domain of psychiatry to be the malfunctioning mind-brain. Accordingly, conditions like cortical blindness, cortical deafness, memory loss due to brain-damage, etc. would count as falling within the domain of SP (i.e., would count as MMCs and potentially mental disorders if deemed harmful or dangerous) since they result from malfunctioning mental parts, while conditions like histrionic personality disorder, explosive personality disorder, narcissistic personality disorder and minor depressive disorder would probably not fall within SP's domain, at least insofar as they do not result from malfunctioning mental parts (i.e., would not count as MMCs and, therefore, would not constitute mental disorders even if they were deemed to be harmful or dangerous states).

At this point, it is probably worth pausing to consider two potential objections to my claims about the domain of psychiatry. First, one might object to my characterization of SP by arguing that any account of psychiatry that is so revisionary as to allow cortical blindness to count as a mental disorder while denying that status to conditions such as histrionic personality disorder is obviously misguided.[162] The quick response to this objection is to note that the revisions that SP proposes follow directly from having psychiatry adhere to the medical model. Indeed, if we want psychiatry to adhere to the medical model, then we ought to be willing to accept any revisions this requires. Of course, it could be objected that the domain of contemporary psychiatry which recognizes what I take to be the "problematic" personality disorders is also the

[162] This objection is mentioned in Murphy (2006).

result of adhering to a medical model, albeit one that is different from that which I propose.[163] The problem with this sort of response is that the domain of contemporary psychiatry just doesn't seem to consistently respect *any* sort of medical model. Put another way, contemporary psychiatry seems to draw an arbitrary line (and perhaps it doesn't even follow this line!) between what falls within its domain and what does not. This is part of the point I meant to illustrate when discussing the difficult and confused state of psychiatry in section 4.2. SP, on the other hand, offers a clear, principled account of what falls within and outside of psychiatry's domain.[164]

Second, some medical models of psychiatry would likely diverge from my characterization, arguing instead that psychiatry ought to be ultimately understood as the clinical genetic-molecular science of the mind-brain. As Murphy (2006) notes, Kandel (1998, 1999) sometimes seems to champion such a gene-centered approach in virtue of his belief that all explanations of cognition and psychopathology will ultimately bottom out at the level of genes and molecules.[165] While I agree that much of our understanding of the human mind-brain may actually revolve around gene-centered events (i.e., gene activations and in-activations), it seems unhelpful to limit the domain of psychiatry (or any medical practice for that matter) to the genetic or molecular level since it may often be useful to think in terms of higher level interactions. Following Murphy (2006, 2008), the point I wish to impress is that even if we could give full explanations of MMCs in genetic terms, doing so might not be the only, or the best, way to understand and deal with MMCs, especially given that we are dealing with a system—the mind-brain—that is embedded in a world of higher-level objects (i.e., objects not commonly described

[163] I thank Steven Gross for raising this possible objection.

[164] Having clear and principled borders does not mean there will be no problematic or "borderline" cases since we might not be able to tell whether something meets the "clear" criteria due to our own epistemic constraints or due to the nature of the object.

[165] Though he sometimes seems to allow that even though genes may play a fundamental role in our understanding of how the mind-brain works and breaks down, they don't play this role in isolation. In these moments, Kandel notes that we will need to pay attention to other factors at a variety of levels that can impact gene expression (1998).

in genetic or molecular terms).[166] In many cases, the optimal theoretical approach may involve employing higher-level descriptions of the mind-brain. Accordingly, we should expect psychiatry to make use of terminology, theories, and resources from a variety of levels just as other medical practices do.

SP embraces a multi-level approach to understanding how the mind-brain can breakdown. This aspect of SP is consistent with the way that contemporary psychiatry is practiced: psychiatric theorists are interested in understanding the functioning and malfunctioning mind-brain at the genetic, molecular, cellular, and systems levels.[167] But this is not the only way in which SP is multi-level. In addition to these various "biological" levels of investigation which are shared with other medical practices, SP also recognizes that other disciplines such as computer science, with an emphasis on understanding computational processes, and behavioral and social psychology, whose theoretical currency is personal and group level behavior, are likely to play a role in fully understanding mental functioning, and, therefore, key to understanding breakdowns in the mind-brain. The addition of these other levels of explanation issues from the human mind-brain's nature as a unique biological system: it has an intentional-cum-computational level that is simply not found in other parts of the body (Marr, 1982; Murphy, 2006, 2008, 2009b).[168] This fact led Murphy to argue, correctly in my opinion, that if there is a way that the mind-brain differs from the rest of the body, it is because it possesses unique powers in the form of its intentional/computational nature.[169] This claim should not be taken to suggest

[166] Finite resources also argue against only discussing MCs in genetic or molecular terms since doing so would require more resources and would be more difficult to cognitively manage.

[167] For evidence supporting this claim, one can simply review articles published within the last few years on psychiatric/mental disorders.

[168] Though some, such as Skyrms (2010), might argue that all systems can be understood as intentional systems.

[169] An interesting upshot of the multi-level nature of the mind-brain sciences is that there has been a move among some theorists to hold that the best we can hope to get when it comes to psychiatry is an understanding of mental disorders as sets of co-occurring symptoms that have a particular causal history but with no reference to underlying causes (Murphy, 2006, 2009b). Proponents of this view, what Murphy terms the *minimal medical model of psychiatry*, often cite a poverty of scientific understanding concerning how the computational and intentional levels are related to the

that the mind-brain differs in any radical way from the rest of the body. It simply means that we may need to draw on more theoretical resources if we wish to fully understand this part of the human biological system and how it can breakdown (Murphy, 2006, 2008).[170]

4.5.b. Mental Medical Conditions and Medical Kinds

In this section I say a bit more about the classes of interest to SP. Because SP is a branch of medicine, we will need to employ the terminology of medical conditions (MCs) and medical kinds (MKs). MCs, you may recall, are simply those classes whose instances share a common dysfunctional biological part that gives rise to a particular cluster of signs and symptoms. MKs are those classes that are likely to be of interest to medical theorists. In the last chapter, I argued that MCs are the sort of classes that are likely to be of interest to medical theorists since they enjoy causal unity, support medical P-laws, and figure in medical explanations. I also suggested that the dysfunctional biological part that causally unifies a MC just is the pathology that causally

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neurobiological levels of the mind-brain as reasons to be skeptical of psychiatry's ability to understand low-level causes of mental disorder (2006). This skepticism has led many minimalists to reject the calls for psychiatry to focus on underlying causes and to propose, instead, that psychiatry place emphasis on understanding the co-occurring symptoms and histories of mental disorders.

SP, following proponents of the "strong medical model" of psychiatry such as Guze (1989; 1992), Kandel (1997), Andreassan (1997), and Murphy (2006), rejects the minimalist stance and argues that MMCs are to be understood not just as sets of co-occurring symptoms, but as "destructive processes taking place in biological systems" (Murphy, 2009b, 104). As Murphy notes, it seems that those committed to the strong view can answer the minimalist's worries about the explanation of causes in psychiatry by noting that computational or intentional level explanations are often just explanations at a different level of the same system—namely, the human mind-brain (Murphy, 2008; Marr, 1982). Accordingly, we can, and indeed do, learn about the causes of these levels through a process of decomposition and localization (Bechtel & Richardson, 1993; Murphy, 2006; Craver, 2007): we first functionally decompose the phenomenon of interest, we then attempt to localize the various functions in specific pieces of brain tissue, we then attempt to mechanistically decompose these components, and we repeat this process of decomposing and localizing until we reach the desired level of interest. The upshot is an understanding of the physical realization of the computational and intentional levels. Further support for SP and the strong medical model is found in the fact that cognitive scientists are actively locating and developing understandings of the mechanisms underlying psychological phenomena.

[170] Another way in which the mind-brain may be unique as a part of the body is that it appears to be uniquely connected to phenomenal states—i.e., experiential states or conscious states. Giving a scientific account of such states and how they are related to the mind-brain is an incredibly difficult and, in almost everyone's opinion, unaccomplished task. Given the difficulty surrounding this task, many theorists have opted to set such research aside and focus instead on more tractable areas of research. These more tractable areas are those that have to do with understanding the biological, intentional, and computational properties of psychological/mental phenomena. That understanding these phenomena is a scientifically tractable endeavor is evidenced by the great strides researchers have made with respect to these phenomena over the past several decades. I will join this camp by opting out of the difficult research that centers on understanding phenomenal states and consciousness.

unifies a MK.[171] Accordingly, MCs are identified with MKs: whenever a class constitutes a MC it will constitute a MK. The MCs of interest to SP are mental medical conditions (MMC) and they constitute mental medical kinds (MMKs).[172]

Partial-Objectivism recognizes that the dysfunctional biological parts of MKs can occur at a number of different levels. Accordingly, SP should expect to see MMKs at these various levels: genetic level MMKs, molecular level MMKs, cellular level MMKs, systems level MMKs. This point is reflected in the fact that MMCs also occur at these various levels: there are genetic level MMCs (e.g., dysfunctional genetic mechanisms), molecular level MMCs (e.g., dysfunctional molecular mechanisms), cellular level MMCs (e.g., dysfunctional cellular components), and systems level MMCs (e.g., dysfunctional systemic mechanisms—i.e., neural mechanisms, cognitive mechanism, etc.). Furthermore, just like the MKs of medicine in general, the MMKs of SP—the MMCSs—are determined by the level at which the dysfunctional part is found: if the dysfunction occurs at the molecular level (i.e., if it is a dysfunction in a molecular part), then it will be a molecular level MMK; if the dysfunction is found at the cellular level (i.e., if it is a dysfunction in a cellular part), then it will be a cellular level MMK, etc. In some cases, it may be possible to reduce a higher-level MMK to a lower level MMK, but this need not be

[171] Peter Zachar (2000) argues that psychiatric disorders are best thought of as practical kinds, classes that exhibit "stable patterns that can be identified with varying levels of reliability and validity", and not natural kinds. I agree with much of Zachar's position insofar as he rejects the idea that psychiatric disorders constitute essentialist natural kinds. I also agree that psychiatric disorders (my MDs) are more than simple biological states since I am committed to the two stage view of MD. What I disagree with is Zachar's assumption that natural kind essentialism is the relevant position to argue against. I also want to emphasize that my P-scientific kind account differs from Zachar's practical kind account insofar as the pragmatic component of the P-scientific kind account has to do with understanding scientific kinds in terms of how they are employed and understood by scientists, while the pragmatic component of the practical kind account appears to center on the role that subjective evaluations play in determining whether or not something is a kind-member.

In his 2002 paper, Zachar further explains the practical kind account as a "general theory of scientific classification" that recognizes the potential importance of internal structures, while also claiming that these structures by themselves are hardly ever sufficient for identifying category membership. If the idea is that the structures are not sufficient alone because different projects may take different aspects of the structure to be relevant to class membership and, therefore, there are likely to be multiple, different classification systems, then the P-scientific kind account is in agreement. In Kendler et al., 2011, Zachar appears to have abandoned the practical kind model in favor of the idea that psychiatric disorders are "mechanistic property cluster kinds" (MPC kinds). MPC kinds are a descendant of Boyd's HPC kinds and are taken to be real features of the world that are not merely imposed by societies and/or cultures.

[172] Kendler et al.'s (2011) "Mechanistic Property Cluster" account of psychiatric disorders is very similar to my view of MMCs and MDs.

implemented, nor implementable, in every case. In some cases, we may recognize several different levels at which we can locate the MMK. OCD, for instance, may be a cognitive MMK insofar as it involves a breakdown in a specific cognitive mechanism—i.e., a hypothesized harm and precaution mechanism—and it may also constitute a neural MMK insofar as it involves a breakdown in a specific neural circuit—i.e., a certain frontostriatalthalamic circuit (Mataix-Cols, 2005; Boyer & Lienard, 2006, 2008).[173] Cortical blindness is similar: it is a cognitive MMK (dysfunction in the visual system) and a neural level MMK (dysfunction in the occipital cortex). MMKs which can be located at a variety of levels are likely to be the rule rather than the exception given the multi-level nature of the mind-brain sciences.

The theoretical and practical upshot of SP's commitment to the two-stage view and how this is related to MMCs and MMKs merits brief mention. For SP, stage one work is purely objective insofar as it is concerned with understanding the nature of MMCs, while stage two work asks which MMCs constitute mental disorders (hereafter, MDs) where MDs are understood to be MMCs that are deemed harmful or undesirable. The MDs of SP are partially-objective states because the subjective-evaluative stage two work only ranges over the output of the purely objective stage one work. The scientific aspect of SP is confined to work on MMCs. The inventory of MMCs—stage one output—can be, and will need to be, employed as input for the stage two project that is interested in identifying MDs. Before SP's inventory of MDs can be produced, a suitable notion of harmful and undesirable will need to be settled on. And, as I noted earlier in this chapter, reaching consensus on what counts as harmful or undesirable is likely to be a difficult process that requires drawing from a number of sources. Nonetheless, what can be said is that the inventory of MMCs is a limit on the inventory of MDs: any potential MD must constitute a MMC. Accordingly, all MDs will constitute MMKs since only MMCs are possible MDs and all MMCs constitute MMKs.

[173] Both neural and cognitive level MMKs are systems level MMKs.

More should be said about MMCs. In particular, the idea of what it means for a mental part to be dysfunctional—i.e., E-systemically malfunctioning—and how we can determine this needs to be explored. I will then have a few things to say about the role of etiologies and pathologies in MMCs, remarks that will help to illustrate how SP diverges from Murphy's view of psychiatry.

4.5.b.i. Identifying and diagnosing MMCs: the challenge of understanding, localizing, and decomposing mental E-systemic functions

Before discussing the challenges associated with identifying and diagnosing MMCs, I want to expand upon some remarks made in chapter 2 regarding identification and diagnosis. In that chapter, I suggested that a central feature of understanding MCs is appreciating how these parts E-systemically function, a task that is often accomplished by localization and decomposition—i.e., locating the biological part that is responsible for the function and mechanistically decomposing this part in order to understand the organized activity of components that gives rise to the E-systemic function (Bechtel & Richardson, 1993; Bechtel, 2002; Craver, 2007). I then suggested that theorists may often take behavior that appears at odds with evolutionary fitness, behavior that is believed to be indicative of an E-systemic malfunction, as *prima facie, defeasible* evidence that some biological part is E-systemically malfunctioning. Such reasoning is consistent with the norms of partial-objectivist medicine, I argued, since these norms do not require absolute certainty regarding the nature of MCs; rather, all they require is that medical theorists allow empirical findings about biological parts and developments in evolutionary theory to guide judgments about whether or not the state is E-systemically malfunctioning and, therefore, constitutes a MC (for more on this point, see Griffiths, 2009).

With regards to diagnosis, it is important to remember that medical theorists are not only interested in treating MCs but also in the maintenance, restoration, and improvement of health. Accordingly, it is important that theorists have the ability to determine 1) when a breakdown in

functioning is underway in a part, and 2) when the part is actually malfunctioning. Medical practitioners are often able to rely heavily on the signs/symptoms associated with MCs as indicators that the process leading to a MC, or a fully actualized MC, is present because of a relatively developed understanding of the function of biological parts. That is to say, these signs/symptoms often enjoy a high degree of reliability and validity as diagnostic criteria because of our rather developed understanding of the biological parts—i.e., the functional biology—of relevance to many medical practices. Granted, we do not have perfect knowledge of the functions of all biological parts relevant to medicine, but we do have a fairly developed understanding of the nature of many of these parts as well as an appreciation of the actual contributions that they make to an organism's ability to survive and reproduce.[174] In short, our developed understanding of the nature of MCs—especially with respect to the associated sign and symptom clusters—and the existence of good diagnostic criteria for such MCs seems intimately tied to the amount of knowledge we have about the human body.

SP's situation with respect to understanding MMCs and its ensuing ability to diagnose MMCs is slightly different from that of other branches of medicine because it is often difficult to understand the E-systemic function of mental parts. The difference in understanding does not issue from any sort of ontological distinction that holds between mental parts and biological parts. Indeed, for SP, mental parts just *are* biological parts—they are the parts of the mind-brain that can be understood under a number of different descriptions and at varying levels and grains of analysis.[175] What makes the difference is the incredible complexity of the mind-brain coupled with the difficulty of distinguishing when behavior is an indicator of a MMC (i.e., a malfunctioning mental part) or a mental performance error (Murphy, 2006). These difficulties do

[174] Murphy (2006) hints at the connection between understanding the domain and understanding breakdowns in parts within the domain.

[175] The only potentially relevant "ontological" distinction would be that there are computational/ informational and intentional levels of analysis for many mental parts that are not obviously applicable to non-mental biological parts. I doubt these extra level(s) of analysis merit consideration as ontologically distinct.

not plague all mental parts equally. For some mental parts, there is general consensus among theorists about their functions—e.g., the function of neurons is to transfer information, the function of sensory systems is to transduce information from the environment from one format into another that can be "read" by the human mind-brain. We have a decent understanding of many mental parts because of our ability to localize the function and then mechanistically decompose the relevant parts (see also Bechtel & Richardson, 1993; Murphy, 2006; Craver, 2007). In particular, we have a fairly well-developed understanding of the mental parts responsible for perception, affect, memory, sensation, movement, and attention. This well-developed understanding is evidenced by general consensus on the function of these processes (e.g., consensus that the function of vision is to provide information about the environment, etc.) and success in localizing and mechanistically decomposing these functions in specific mental parts.[176] Because we know what it is for these parts to properly function (i.e., E-systemically function), we are in a position to determine when the part is malfunctioning. Several examples may prove useful.

Consider the case of vision and visual MMCs. We begin with the idea that it is generally recognized that the function of the visual system has to do with transforming information about the environment in the form of photons into perceptual representations that other parts of the mind-brain can exploit for the purposes of action guidance and decision-making.[177] Because we know what constitutes the function of the visual system, we can gain a better understanding of how a properly functioning visual system works through the mechanistic decomposition of such a system.[178] The output of the decomposition is an understanding of how the components of the

[176] There are, of course, counter-examples to this claim. One example is the idea that the hippocampus plays a role in *both* imagination and memory, a claim about the E-systemic function(s) of the hippocampus that may be contested (Hassabis & Maguire, 2009; Buckner, 2010).

[177] The visual system may have more E-systemic functions but this is at least one of them.

[178] Such work is usually not an overnight affair and is likely to constitute a work in progress for many years.

system are organized and work together in order to give rise to the E-systemic function. This output can be employed as a yardstick to determine whether a given visual system is properly functioning. Now, let us suppose that a person presents with an apparent inability to use visual information to guide action and make decisions. Given our understanding of the visual system's function, we can assume that the presented behavior results from a breakdown in the visual system. We can then examine various components of this system to better appreciate where in the visual pathway the breakdown has actually occurred. We could, for instance, test whether visual information is available to sub-cortical mechanisms in the *superior colliculus*. If we find that the subject is sensitive to visual information that follows this sub-cortical pathway, then that would suggest that the breakdown must occur further along the visual pathway than the eye. Such refinements can continue until we locate the actual breakdown in, for instance, the occipital cortex. Of course, if we do not find any sort of breakdown in the visual system, then we may have to wonder whether the behavior is actually indicative of a visual system MMC. It may, after all, turn out that the patient does not enjoy a visual system MMC but is in a catatonic state as a result of poisoning and this explains the presenting behavior.[179] Regardless of how the actual story unfolds, the point to appreciate is that it is our rather developed knowledge of the normal functioning of the visual system (and the components of this system) that makes it likely that we will be able to determine when a visual MMC is present and when it is not.

In addition to perception, our appreciation of the nature of the mental parts responsible for attention, memory, affect, sensation, and movement also seems to be rather robust. As a result, it is likely that we will have an easier time identifying and understanding the MMCs that occur which seem connected to these mental processes. Most importantly, we will be able to determine when behavior that seems to be indicative of a malfunction in one of these parts is

[179] Another strategy to locate the breakdown might involve neuro-imaging. In this case, the sign of a breakdown would be the lack of a BOLD response in cortical regions that are normally activated during visual information processing tasks.

actually caused by a mental part that is malfunctioning, and when it is just the result of human performance failures and ineptitude.[180]

In agreement with Murphy (2006), it seems that understanding breakdowns in other mental parts, especially those involved in cognition—e.g., reasoning, desiring, and deciding—may prove more difficult. This difficulty arises for two reasons: 1) it is often unclear what constitutes proper cognition (i.e., cognition that is evolutionarily adaptive in the forward looking sense) making it difficult to appreciate the function of cognitive mental parts, and 2) even when we can determine what constitutes proper cognition (i.e., we know the function(s) of a type of cognition) it is still often very difficult to carry out the process of localizing and decomposing the function(s) in a mental part (Murphy, 2006). The potential upshot of these two points is that we may lack the ability to know what the parts responsible for these cognitive functions look like when properly functioning and, as a result, we will not be able to determine when these parts are malfunctioning. This would essentially result in a weakness in our understanding of cognitive MMCs. Given the centrality of cognition to our mental life, let me say a bit more about the two problems that arise when attempting to understand cognitive MMCs.

Concerning the first point, it is often unclear what constitutes proper cognition—i.e., a functioning cognitive system—since it is not always clear when cognition in the form of a particular cognitive strategy (i.e., reasoning, deciding, etc.) contributes to evolutionary fitness. Deciding to kill oneself seems to be an obvious mark of a cognitive malfunction until we consider

[180] Disagreements concerning E-systemic functions are bound to arise, and when they do, they could have relevance to our understanding of MMC status. One case worth considering is postpartum depression. Some theories of postpartum depression hold it to be an evolved trait to shield recent mothers from investing too many emotional and material resources in newly arrived infants since the likelihood of these offspring surviving in the environment in which we evolved would have been low (Hagen, 1999). If true, this theory would suggest that a mother's becoming depressed postpartum is an indication of an E-systemically functioning affect system. Accordingly, mothers experiencing postpartum depression would not enjoy a MMC, though we may still decide it is good to treat them since the state is deemed harmful or undesirable. Of course, if it turns out that postpartum depression is no longer adaptive in our environment, and we employ a forward looking evolutionary approach that takes current environment as relevant to determining the adaptive nature of biological functions, then this would suggest that postpartum depression is a MMC nowadays even though it was not in our evolutionary past.

that the person is only deciding to do this because not doing so has been guaranteed to lead to the death of one's wife and six lovely children. Desiring objects that one cannot have could seem contrary to evolutionary fitness since it could distract one from pursuing more attainable goals (and perhaps more chances for reproduction) until we consider the possibility that such a cognitive strategy may lead one to work harder and potentially achieve more goals that will position one for more success which could open the way to better reproductive choices (i.e., more fit mates). Deciding to forego a strategy of increasing one's wealth in order to punish someone who has transgressed a social norm seems to be a poor decision-making strategy until we consider that this action may have a deterrent effect on others, thereby increasing the chance that other group members will not transgress the norm. These cases are meant to illustrate that it is often difficult to determine which cognitive strategies are likely to be the result of a properly functioning cognitive system. If we cannot tell which strategies contribute to evolutionary fitness, then we will lack the ability to determine which cognitive parts are E-systemically functioning or malfunctioning.

But all is not lost. After all, it is possible that in the future we will have better evolutionary theory at our disposal to help us understand cognitive function.[181] And even at

[181] An appreciation of cognitive functions—i.e., the norms of cognition—will likely require considering what evolutionary theory has to say about the evolutionary value of these cognitive processes. Such a project might also need to consider the fact that part of the difficulty in determining the proper function of these cognitive mental parts is connected with the social-cum-cultural aspect of humans. Accordingly, understanding the proper functioning of these parts—i.e., understanding the E-systemic functions of these parts—may require an appreciation of the role that culture, society, and environment play in the evolutionary fitness of organisms.

The development of evolutionary theories dealing with the interaction between organisms and their environments in the form of Dual Inheritance Theory (Boyd & Richerson, 1985), Niche Construction Theory (Odling-Smee, Laland, & Feldman 2003), and Jablonka and Lamb's general theories of Inheritance Systems (Jablonka 2001; Jablonka & Lamb 2005) are likely to help us better understand the E-systemic function of cognitive mental parts. Furthermore, a better understanding of humans as social creatures may provide a way to adjudicate between competing accounts of what should count as the proper E-systemic function of these cognitive mental parts. Consider how such an approach may be relevant to understanding reasoning and decision-making. One possibility is that findings from social psychology and the psychology of decision making will reveal that that the "appropriate" decision-making strategy may change as a result of one's context. If this were the case, then we ought to expect a number of differing strategies to be employed in different circumstances. Social psychology would help us understand what counts as proper decision-making in a given set of circumstances. Deviations from "the correct strategy" in a given context, then, would count as bad reasoning, and when that bad reasoning results from a break down in a relevant cognitive mental part, we would have the presence of a MMC. Of course, deviations from the "correct-for-this-context" reasoning strategy would not *necessarily* indicate an E-systemic malfunction in a cognitive mental part since humans are imperfect cognitive creatures and may simply employ incorrect strategies.

present, as Murphy notes, there are some cases where there is general consensus that cognition is malfunctioning even though we may not fully understand what constitutes proper cognitive function—i.e., we are not sure what cognitive strategies are adaptive in the forward-looking sense (Murphy, 2006). These are generally cases where behavior is extremely abnormal in the sense of statistically abnormal. It may be up in the air, for instance, whether the decision-making system of the person who chooses to punish over maximize income is malfunctioning, but there is likely to be little disagreement over whether a person's continuing to take bad decisions in the face of overwhelming reasons to the contrary is an indication of a cognitive malfunction. The take home message is that we need not be utterly pessimistic about our prospects for understanding cognitive functions—i.e., the function of cognitive mental parts.

The frustrating part, however, is that even if we can come to understand cognitive functions, we are still faced with the next step of localizing the function in a mental part which can then be mechanistically decomposed. This brings us to the second point discussed above. The complex nature of the systems believed to be responsible for cognition mean that it will be very difficult to carry out the process of localization and decomposition (Murphy, 2006). If we cannot carry out this process, then we face the problem of not being able to determine when "poor cognitive performance"—i.e., behavior that is *prima facie* indicative of a malfunctioning mental part—results from a MMC or human performance error (i.e., stupidity, lack of attention, the employment of bad strategies that have been learned, etc.) (Murphy, 2006). As an example, consider the case of addiction.[182] Let us assume that there is consensus that the decision-making employed by addicts is decidedly poor—i.e., it is behavior that is likely indicative of a malfunctioning mental part. Of course, just because the behavior is indicative of a malfunctioning mental part does not mean that it is actually caused by such a part since it could simply be the result of bad choices. If we want to determine whether addiction (or an "instance

[182] The following example borrows heavily from Murphy (2006) which has a detailed discussion of addiction.

of addiction") is actually a MMC or not, we will need to be able to determine if the addictive behavior is the result of a malfunction in a mental part—a malfunction that leads people to make decidedly poor choices in the face of overwhelming evidence—or whether the addictive behavior results from poor decisions that are not caused by a malfunctioning mental part. If the first explanation is correct, then addiction (or the "instance of addiction") would seem to constitute a MMC; if the second is, then it would seem to follow that addiction (or the "instance of addiction") is not a MMC. Deciding which explanation of addiction is correct requires that we be able to localize and mechanistically decompose decision-making in order to determine whether the responsible mental part is functioning or malfunctioning in the addict(s).

The point to appreciate is that even if we could determine what constitutes cognitive function—i.e., what reasoning is evolutionarily good reasoning, what desiring is evolutionarily good desiring, etc.—we would probably still encounter difficulty when attempting the process of localization and decomposition. The reason for this difficulty is captured nicely in Fodor's "first rule of the non-existence of cognitive science": the more central a process is, the more unlikely we are to know anything about how it is realized in the mind-brain (Fodor, 1983). While I disagree with Fodor's overwhelming skepticism concerning our ability to understand the mechanisms responsible for cognition, his worry seems at least partially grounded insofar as central processes—especially the sort likely responsible for cognition—are probably widely distributed, incredibly complex, and, therefore, difficult to localize and decompose. And, as already noted, if we cannot carry out the process of localization and decomposition, then we will face greater difficulty when attempting to distinguish between competence and performance errors—i.e., between poor cognitive functioning due to a MMC or poor cognition otherwise unspecified.[183]

[183] Competence errors would indicate malfunction, while performance errors are more likely to indicate poor reasoning strategies, or choices, that do not result from a malfunctioning part.

The issues mentioned above seem more or less correct about the difficulties that are likely to arise when attempting to understand the mental parts responsible for cognition. Where SP diverges from the skeptical position that sees these difficulties as insurmountable, a position that Murphy (2006) seems to endorse, is by holding that we will probably be able to locate several cognitive "hotspots"—i.e., mental parts that are likely to be involved in cognition and about which we have a fairly developed mechanistic understanding, parts such as the cingulate cortex, the dopamine system, and the frontal cortex. The upshot of the identification of these cognitive "hotspots" is that we can do partial localizations and decompositions that may prove useful in understanding when a MMC is responsible for "poor cognition" and when it is not. We can, for example, take it as a given that individuals presenting with consensus level "poor decision making" need to have these decision-making "hotspots" investigated for malfunctions.[184] Of course, just because no breakdown is found in these "hotspots" does not mean that the decision-making system is not E-systemically malfunctioning for it could be that some as yet unidentified component of the mental part responsible for decision-making is malfunctioning and, therefore, responsible for the poor decision-making. Nonetheless, looking at "hotspots" may prove useful when we lack a mechanistic understanding of the whole mental part.

These last few sections suggest that we may have difficulty determining when some behaviors that seem indicative of a cognitive malfunction actually result from such a malfunction. Accordingly, we will be unable to say for certain whether the state is a MMC or simply a variant of normal cognitive functioning that is imperfect and sometimes goes awry. Furthermore, in some cases there may not even be consensus as to whether the exhibited behavior is odd, while in other cases there may be general consensus. Taken together, these points suggest that SP's understanding of cognitive MMCs is likely to be slow moving and highly speculative, while its

[184] "Consensus level poor decision-making" is decision-making that is so bad almost everyone would recognize it as such.

understanding of perceptual, affective, attention, memory, motor, and sensory MMCs is likely to be more developed and certain.[185] It is also likely that the way that SP understands and deals with cognitive MMCs will be highly revisionary given that contemporary psychiatry is liberal in its recognition of cognitively based mental disorders, while SP demands that these MMCs result from E-systemically malfunctioning mental parts.[186]

4.5.b.ii. Pathologies & etiologies: the causal factors of MMCs

SP's focus on understanding the cause(s) of MMCs is part of what secures its status as a branch of medicine properly grounded in science. But causal talk is never easy, especially when we are dealing with causally dense systems such as the human mind-brain (Zachar, 2008). Accordingly, employing the distinction between etiological and pathological factors as discussed in chapter three becomes a useful heuristic to sort through the myriad causes that should be considered when attempting to provide a causal description of MMCs (Murphy, 2006, 2009). A brief review of the distinction as applied to MMCs and an example is provided before we move on to a discussion of etiological factors unique to MMCs.

Under etiological factors are included all causes that play a role in giving rise to a certain mental pathology. The pathology itself is the actual dysfunctional mental part that realizes the condition in the organism. Put another way, etiological factors can be thought of as causes of the pathology and the pathology can be thought of as the cause of the signs and symptoms typical of the condition—i.e., the sign/symptom profile associated with a MMC (Murphy, 2006, 2009b).[187]

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[185] Murphy (2006) comes to a similar conclusion.

[186] I discuss this point in greater detail in section 4.6.

[187] As discussed in chapter 3, the above distinction is founded upon the idea that "many factors can interact to produce the pathology that is common to all cases of a condition [i.e., disorder]."(Murphy, 2009b, 113) This is just to say, the etiology of a particular disorder may vary from instance to instance while the pathology remains the same.

A particular MMC will share a common mental pathology[188] even though this pathology may have a number of potentially different etiologies. Obsessive-Compulsive Disorder (hereafter, OCD) appears to be a MMC that is realized by a specific mental pathology—namely, a malfunction in a certain frontostriatalthalamic circuit. The malfunction of this circuit (i.e., the pathology), however, can result from a variety of genetic and/or environmental factors.[189] Cortical blindness is a MMC that is realized via a breakdown in the functioning of the occipital cortex. This pathology can result from a number of different etiologies—e.g., physical insult to occipital cortex, neuro-developmental problems, restricted blood-flow via stroke, etc. In both cases we have a MMC—a single pathology—that can result from a number of different etiological factors.

In line with my previous remarks on MKs, it may sometimes be the case that etiological factors are relevant when typing and determining MMK status.[190] Etiologies seem to be particularly relevant to typing when the etiological factor is a "sustaining" factor—i.e., when its presence is needed to sustain the malfunctioning mental part. The importance of typing according to these factors stems from the possibility that the best treatment interventions may be directed at the sustaining factors. In cases where the mental part is in a steady state of malfunctioning—i.e., when it does not require a sustaining etiological factor in order to persist in malfunctioning—it is probably less important to type according to etiology. After all, it doesn't matter much whether a person's visual cortex is damaged by a hammer, baseball bat, or tire-iron—what matters is the damage done. In conclusion, it seems reasonable to think that etiological factors will play a similar role in typing MMKs that they play in typing MKs: sometimes a MMK will be composed of several sub-classes that are comprised of etiology-

[188] I use 'mental pathology' to refer to a pathology in the mind-brain with no commitment to the level at which the pathology should be described.

[189] For more on the potential genetic and environmental factors, see section 4.6.b.

[190] For more thoughts on typing by etiology, see Murphy (2006), esp. Chapter 10.

specific MMKs and sometimes a MMK will not be composed of etiology specific sub-classes. In section 4.6.b. I offer several examples to illustrate how MMCs can be typed by pathology and etiology.

The proposed way of talking about MMCs as being realized by malfunctioning mental parts that cause sign/symptom profiles does not require that the conditions be described at any particular level. Just like the mind-brain can be discussed at many different levels, pathologies—i.e., mental part malfunctions—can also be described and/or located at any of these levels. We may, for instance, locate the pathology at the cellular level or we may locate it at the systems level. We may even be able to locate the pathology at a number of different levels but prefer one over the other if doing so provides more explanatory power. Such an approach is often termed pluralistic because it sees a variety of explanations at a variety of levels as being potentially central to understanding a given phenomenon—here, MMCs. What all of the causal factors—i.e., pathologies—cited in this plurality of explanations have in common is that they are "realized in brain tissue" (Murphy, 2006, 13). A commitment to pluralism is appropriate for SP since it is theoretically grounded in the mind-brain sciences, sciences that are notoriously explanatorily pluralistic as a result of their multilevel nature (McCauley & Bechtel, 1999, 2001).

The pathology-etiology distinction is useful to employ in SP, an unsurprising conclusion given its role in medicine and SP's attempts to fashion itself as a branch of medicine (Murphy, 2009b). What now needs to be considered is how SP's understanding of pathologies and etiologies may differ from other medical model accounts, particularly that proposed by Murphy (2006).

4.5.b.iii. SP's emphasis on breakdowns: diverging from Murphy

Until now, much of my discussion on the nature of MMCs has focused on the necessity of a malfunction in the mind-brain. This claim invites questions, however, since the borders of

the mind are somewhat up for debate. If the mind is understood as a computational information processor, then one could take this to mean that the information that the mental part processes also constitutes part of the mind. Such reasoning seems to have led Murphy to hold that a properly functioning mental part that is operating over "deviant input" would count as a MMC (2006, 73-77). Accordingly, for Murphy, a person would count as enjoying a MMC if her brain parts were functioning properly but she was repeatedly given bad input in the form of unwarranted negative appraisals. Contrary to Murphy, I reject the idea that bad input fed into a properly functioning brain part by itself constitutes a MMC since there is no obvious breakdown in the mental part—i.e., mind-brain. Such a claim commits me to the idea that the information that mental parts operate on are not proper components of these parts (that is to say, the content of the information is not a proper mental part even though the vehicle that carries the information may be a component of the mental part). I accept this commitment and propose the following: SP takes the border of the mind to be synonymous with the border of the brain. The upshot of this divergence with Murphy is a deeper commitment to the idea that MMCs are realized in brain tissue. Let us consider an example to illustrate how SP's understanding of the boundary of the mind-brain, and what this means for MMCs, differs from Murphy's view.

Imagine that a person whose mental parts are properly functioning is constantly given unwarranted, negative appraisals from family and friends. We will call the unwarranted negative appraisals the person is subject to the "deviant input" being feed into the otherwise properly functioning system. Let us further imagine, not unrealistically, that the person will start to exhibit a sign/symptom profile that is commonly associated with depression. Let us also imagine that in some cases this particular sign/symptom profile is indicative of a breakdown in a mental part realized in the brain—e.g., a breakdown in the dopamine or serotonin system. Of course, being enlightened practitioners of SP, we will understand that this particular sign/symptom profile can result from breakdowns in different mental parts that are realized in different brain parts and that

it can also result from non-MMCs such as "problems in living". We will also understand that our best treatment options hinge on understanding whether a mental part is broken-down and if some part is, what particular part that might be. So, we will attempt to get a better sense of which part is compromised by employing a number of different tests—e.g., blood tests, neuro-imaging, detailed life history, etc. Now imagine that we discover through the life history that the patient is subject to much unwarranted, negative criticism at the hands of family and friends. All other tests suggest that the patient's mind-brain is functioning normally—i.e., there are no breakdowns in any "part" of the brain. In the end, we recognize that "deviant input" is the culprit—the cause—of the sign/symptom profile the patient exhibits.

Now, I take the difference between Murphy's view and my view to be that Murphy would recognize the above as an instance of a MMC, while SP would not recognize this as such since the sign/symptom profile does not result from a malfunctioning mental part where mental parts are understood to be realized in brain tissue. That is just to say, Murphy's way of understanding input as a component of the mind-brain would have us believe that there is something wrong with the patient—i.e., that the patient enjoys a dysfunctional mental part. But as far as I can tell there is nothing wrong with the patient's mind-brain. Indeed, if there is something wrong with anyone's mind-brain, it would seem to be the people that continue to heap unwarranted, negative appraisals on this person—can't they determine what amount of negative appraisal is warranted? The upshot is that treating "deviant input" fed into an otherwise properly functioning mental system as constituting a malfunctioning mental part seems to misattribute breakdowns. After all, we don't say that a person's vision is bad if they are shown a picture under poor lighting conditions; rather, we attribute the poor visual experience and the inability to recall fine detail to the ambient lighting. The case of "deviant input" seems analogous. For Murphy, the imagined condition seems to belong within the domain of psychiatry. For SP, the condition may need to be treated but it does not fall within the domain of psychiatry since there is

no breakdown in mental functioning. In conclusion, the SP I envision is brain-based in a way that Murphy's view is not.

The above story is presented in an incredibly clean manner. Indeed, I made sure to imagine the case in the easiest way possible to draw attention to how SP differs from Murphy's view when it comes to the boundaries of the mind and the identity of mental parts. What I now want to do is reconsider some aspects of this toy case in order to say something about the importance of considering deviant, or bad, input as a special etiological factor of MMCs.

4.5.b.iv. The special etiologies of MMCs

While I disagree with Murphy's understanding of the significance of "bad input", I think he is correct to draw attention to the role that such input may play in MMCs. The plastic nature of the human mind-brain—exemplified by its ability to have its structure altered due to information from the environment—suggests that "bad input" is probably better understood as a potential etiological factor of MMCs rather than a pathology itself. If true, this would suggest that the above case may have glossed over an important possibility: that the constant "bad input"—i.e., unwarranted, negative appraisals—that the patient suffered actually led to changes in her mind-brain. That is to say, it seems possible that the "bad input" could have altered the patient's mental parts culminating in an actual dysfunctional mental part. But notice that even if this was the case, it would still not follow that "bad input" was part of the MMC—it was, merely, an etiological factor that made the actual MMC—i.e., the dysfunctional dopamine or serotonin system—become realized. And, it is entirely possible that removing the bad input would allow the system to "return to normal". In this sort of case, it seems quite obvious that the etiological factor would be functioning as a "sustaining" etiological factor of a MMC—i.e., its presence is needed in order to sustain the malfunction in the mental part. Not all cases will be like this. It may sometimes turn out that a "switch" has been flipped and the etiological factor is no longer

needed in order to sustain the condition. Even in this latter case, however, the nature of the "bad input" has not changed—it is still merely an etiological factor.

The above considerations suggest that "bad input" may be best understood as a sort of etiological factor that is unique to MMCs given the nature of the mind-brain as an information processing system. That information can have such an impact on mind-brain functioning is widely acknowledged by mind-brain scientists (Lillard & Erisir, 2011; Fuchs & Fluegge, 2014). It is a fact also embodied in contemporary practices such as psychotherapy and cognitive-behavioral therapy that are aimed at restoring proper mental functioning by manipulating input feed into the mind-brain (for more on CBT's relevance to SP see section 4.8.). Interestingly, in the cases of therapeutic intervention, no one seems tempted to say that receiving "good input" counts as grounds for being mentally healthy even though it may cause one to become mentally healthy—i.e., be an etiological factor of restoring proper mental functioning. Why, we might ask Murphy, should the case of bad input be thought of differently? With these comments on the details of MMCs and MMKs completed, I now turn my attention to a discussion of SP's nosology and its revisionary nature with respect to what it recognizes as MDs.

4.6. SP's nosology(s)

I noted earlier that SP is committed to the two-stage view of MDs. Now, I want to explore what this commitment says about the nosology(s) (i.e., taxonomies or classification schemes) of relevance to SP. First and foremost, SP recognizes at least two nosologies: one that is research oriented and one that is clinically oriented. [191] This move differs from contemporary psychiatry which takes a single nosology in the form of either the ICD-10 or the DSM-V as

[191] This idea is borrowed from Murphy (2006).

employable for both of these purposes.[192] The problem with employing a single nosology for multiple purposes is that it may actually hinder progress since nosologies amenable to the two very different tasks of research and diagnosis may need to differ in significant ways (Murphy, 2006). A nosology for research, for instance, should focus on describing and understanding relationships among causal mechanisms, while a nosology for clinical purposes will need to insure high inter-rater reliability (Murphy, 2006). Accordingly, it is probably best to have separate nosologies that are designed for these two specific tasks.

One way to draw the distinction between research and diagnostic nosologies is to rely on the distinction between first and second stage work (Murphy, 2006). Because SP is primarily interested in better understanding MMCs, it follows that SP's primary interest is in developing a nosology of MMCs. This nosology is essentially designed for research purposes, with the idea that better understanding these conditions will make treatment and diagnosis, in the long run, better. The nosology of SP—the research oriented nosology—can be employed for a number of purposes. One purpose that it will surely be employed for is the development of another nosology of MMCs that need to be, or should be, treated (i.e., MDs). Notice, however, that employing the research nosology for such a purpose does not entail that it will be taken on wholesale since not every MMC needs to be, or should be, treated (especially, and perhaps solely, those MMCs that enjoy their status as such because the relevant part fails to make its normal contribution to an organism's ability to reproduce). The clinical nosology designed to aid the practice of diagnosis for treatment purposes will only need to mention the MMCs that are deemed harmful or undesirable. Developing the clinical nosology, therefore, requires second stage work, evaluative work that is absent from the research oriented nosology. Once the SP nosology has been subjected to the evaluative criteria, the output is a nosology of MDs. This nosology ought to

[192] At the time of writing there has been a break within sections of the diagnostic and research communities over the use of the DSM as the nosology of choice. NIMH, the nation's largest mental health research institute, has recently announced it will stop employing the categories and terminology of the DSM and instead preferentially fund research that is centered on better understanding the symptoms and signs associated with DSM categories (Insel, 2013).

make diagnosis of such disorders as easy as possible. It should also allow for inter-rater reliability (i.e., multiple clinicians ought to diagnosis similarly). But, it need not be a-theoretical. Following Murphy (2006), I conceive of this clinical/diagnostic nosology as being explicitly grounded in our best biological theory given that it is derived from the SP nosology which is purely objective and explicitly theoretical insofar as it sees MMCs as essentially involving malfunctioning mental parts. The development of separate purpose-driven nosologies should help to improve research, diagnosis and treatment (Murphy, 2006).

It is important to realize that SP sees its nosology as a work in progress that will evolve as we gain a better understanding of the mind-brain.[193] Because of the close connection between how well we understand the mind-brain and the development of SP's research nosology, there are likely to be many unanswered questions concerning alleged MMCs for years to come. After all, even though we understand much about the mind-brain, there is much more that we do not yet know. This should not, however, dissuade us from pursuing a nosology that is grounded in, and aims to classify according to, causal mechanisms; rather, it should serve as a reminder that the classification is a work in progress that traffics in defeasible claims about breakdowns in the mind-brain.[194]

The incomplete nature of a scientific research nosology will likely affect the clinical nosology (Murphy, 2006). After all, we cannot wait until we have filled out the entire research nosology to devise the clinical nosology for we need to be able to employ the one for clinical purposes as soon as possible. In order to sidestep this problem, we could continue to employ the DSM or ICD-X as the clinical nosology and update/alter it to fit with what we learn about MMCs. In concrete terms, we would use the research nosology as an update tool for these currently used

[193] Murphy (2006) proposes a "piecewise" approach to developing the nosology.

[194] Murphy (2006) also argues that concerns about the incomplete nature of the research nosology do not constitute good reasons to postpone the acceptance and development of this causally based nosology.

diagnostic manuals. Murphy (2006) offers a similar strategy of using novel research findings to continuously update current clinical/diagnostic nosologies. If we follow this approach, there will need to be an explicit recognition that the clinical nosology is a stand-in that is constantly being refined. And, as already stated, there would need to be recognition that the clinical nosology is not a-theoretical—rather, it is committed to SP's view of MDs. Employing this approach would insure that clinicians and theorists interested in diagnosis will have a nosology to employ. As the research nosology evolves, these findings would affect the clinical nosology with the end stage being a clinical nosology that is fully consistent with the research nosology—i.e., a clinical nosology that is constrained by the research nosology.

4.6.b. SP's revisionary nature

I now want to consider in more detail the revisionary nature of SP.[195] To do this, I employ the idea, introduced in the last section, that SP's research nosology can be used to revise current clinical/diagnostic noslogies like the ICD-X and the DSM-IV TR. The revision process begins by considering what contemporary findings from the mind-brain sciences have to say about the causes of several alleged mental disorders (hereafter, DSM disorders since they are categories taken from the DSM –IV TR): major depression, OCD, narcissistic personality disorder, and the paraphilias (see Addendum for remarks on the connection between the DSM-IV TR and the DSM-V). I will leave aside concerns about whether these DSM disorders are harmful and/or undesirable and focus instead on their underlying causes since I wish to illustrate how SP is revisionary with respect to its emphasis on causes, not its understanding of harm or desirability. I take the findings concerning the causes of these DSM disorders as evidence of whether or not SP would recognize them as constituting MMCs and, therefore, being potential MDs. The revisionary nature of SP lies in the fact that it will probably not recognize all DSM disorders as MMCs (i.e., potential MDs). The value of this investigation into the revisionary process is that it

[195] For similar but separate considerations regarding DSM revision, see Murphy (2006).

will help to illustrate concretely how SP differs from contemporary psychiatry when it comes to developing nosologies and understanding MDs.[196]

4.6.b.i. Depression[197]

The DSM-IV TR defines depression as follows:

> Depressed mood and/or loss of interest or pleasure in life activities for at least 2 weeks and at least five of the following symptoms that cause clinically significant impairment in social, work, or other important areas of functioning almost every day
>
> 1. Depressed mood most of the day.
> 2. Diminished interest or pleasure in all or most activities.
> 3. Significant unintentional weight loss or gain.
> 4. Insomnia or sleeping too much.
> 5. Agitation or psychomotor retardation noticed by others.
> 6. Fatigue or loss of energy.
> 7. Feelings of worthlessness or excessive guilt.
> 8. Diminished ability to think or concentrate, or indecisiveness.
> 9. Recurrent thoughts of death
>
> —(APA-DSM, 2000, p. 356).

[196] It is important to recognize that the DSM is primarily designed for diagnostic and not research purposes. Accordingly, one ought not to be terribly surprised if the categories of the DSM cobble together some things that nature has made to stand apart. Indeed, when we lack an understanding of the underlying causes—i.e., dysfunctional biological parts—responsible for MCs and MMCs, then the best we can hope for is often a nosology of symptom clusters. But, as we gain knowledge about relevant causal factors, we need to be ready to update and revise our nosologies in order to reflect our improved understanding. That is my goal here, not to criticize the DSM categories for being little more than symptom profiles, but to explore how SP would understand these DSM disorders given our current understanding of the mind-brain and how it breaks down.

[197] The following sources were used to develop this section: Hirschfield, 2000; Manji et al., 2001; Douma et al., 2005; Nemeroff & Vale, 2006; Terman, 2007; Haeffel et al., 2008; Lo et al., 2008; Hankin et al., 2009; Morris et al., 2009; Slavich et al., 2010; Matthews & Harrison, 2012.

If we take the DSM criteria to be definitive of depression (i.e., enjoying the symptom profile means that one has DSM depression) then a person will have DSM depression whenever they enjoy the symptom profile—i.e., 5 out of 9 of the symptoms at least one of which must be depressed mood (1) or loss of pleasure (2). To determine whether SP would recognize DSM depression as a MMC and, therefore, a potential MD, it will need to determine if all instances of DSM depression result from a common dysfunctional mental part. So let us consider what current findings from the mind-brain sciences have to say about the causes of DSM depression.

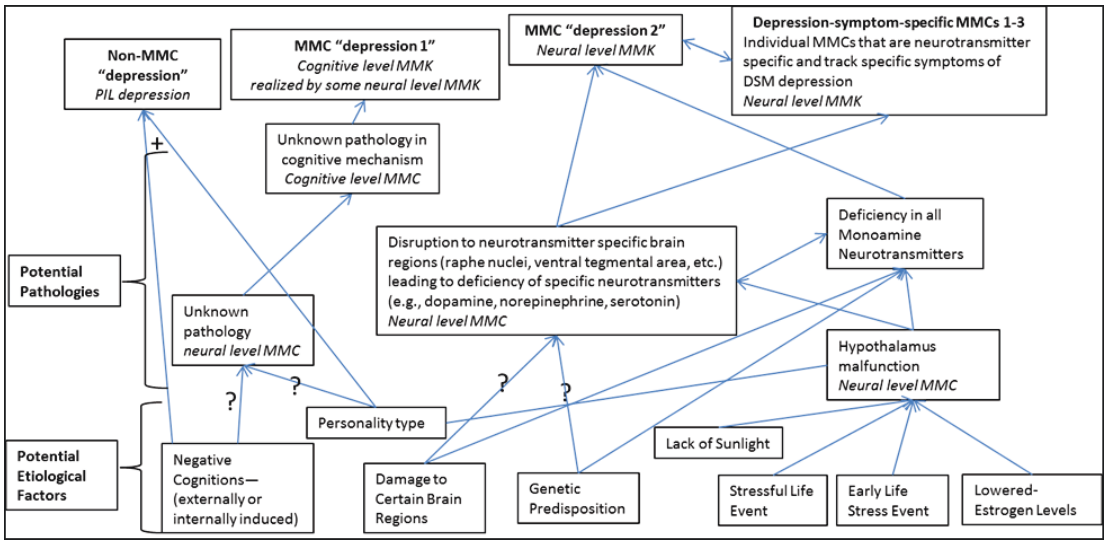

Figure 4.2. Representation of the potential pathologies and etiologies that may give rise to DSM Depression.

Perhaps the first thing to say is that it is unclear what dysfunctional mental parts(s) are actually responsible for DSM depression. There is, for instance, evidence that a deficiency in the monoamine neurotransmitters (e.g., serotonin, norepinephrine, dopamine) plays a crucial role in many instances of DSM depression. What is unclear, however, is what role this deficiency plays since SSRI interventions often restore levels of neurotransmitters in the brain to normal in a relatively short period of time, but the symptoms of DSM depression are not lessened for several weeks after SSRI intervention begins (Hirschfield, 2000). This finding raises the question of whether there might be some other dysfunctional part(s) that is responsible for the deficiency in

monoamines (Manji, et. al., 2001). If this were the case, then it would follow that *this* malfunctioning mental part which is actually responsible for the deficiency constitutes the relevant pathology. Another wrinkle in the data is that there is evidence that deficiencies in specific neurotransmitters correlate with specific signs or symptoms of DSM depression and that these specific neurotransmitter deficiencies might result from dysfunctions in neurotransmitter specific regions of the mind-brain such as the raphe nuclei (Matthews & Harrison, 2012) or the ventral tegmental area. If correct, these findings suggest that there may be several different pathologies that give rise to the several different types of neurotransmitter deficiency that in turn are responsible for certain symptoms associated with DSM depression. I have illustrated these possible connections in Figure 4.2. If this interpretation of the data is correct, then it could be that DSM depression is actually comprised of three separate, but related, MMCs that result from dysfunctions in neurotransmitter specific mental parts (labeled depression-symptom-specific MMCs in figure 4.2). As noted in the figure, the dysfunctional brain regions would count as the relevant pathologies of these depression-symptom-specific MMCs. Furthermore, these MMCs would clearly constitute MMKs because they support P-laws of interest to psychiatry and they figure in psychiatric explanations. Because there sometimes appears to be deficiencies in several neurotransmitters at once, a caveat to the above remarks might be that some sub-classes of DSM depression involve more than one MMC (i.e., some instances of DSM depression result from dysfunctions in 2 or 3 of the neurotransmitter specific brain regions). This idea is represented by the bi-directional arrow between the sub-class MMC "depression 2" and the sub-class depression-symptom-specific MMCs.

In addition to instances of depression that involve monoamine deficiency or some other unknown malfunctioning mental part, there are also likely to be some cases that result from no pathology at all. After all, there is evidence that personality type combined with a particular world-view or negative cognitions can give rise to DSM-depression (Haeffel et al., 2008; Lo et

al., 2008; Hankin et al., 2009). Cases like this are instances of Non-MMC depression—they are, perhaps, more appropriately termed "problem-in-living", or PIL, depression.[198] These sub-classes of DSM depression do not belong in SP's research nosology since they do not constitute MMCs (and, therefore, are not potential MDs) though they may appear as a potential etiological factor of a MMC.[199]

The preceding remarks suggest that SP is likely to be revisionary in its understanding of depression since contemporary empirical data suggests that the category DSM depression groups together a number of MMCs and non-MMCs that do not share a common malfunctioning mental part. Accordingly, SP's research nosology will eliminate the overarching category "Depression" (which will, in turn, eliminate it from the clinical/diagnostic nosology) and replace it with the variety of "depression" MMCs. Furthermore, SP's research nosology is likely to type the various "depression MMCs" by etiology since there are many etiological factors that can give rise to these MMCs and understanding which one is responsible for a particular case may prove useful for intervention and treatment purposes.[200] Thus, the clinical/diagnostic nosology will also type by etiology—i.e., it will include etiology-specific-"depression" MDs.

[198] "Problems in living" is a term I borrow from Tomas Szasz (1960) to refer to conditions that result from life circumstances and are not caused by malfunctioning mental parts. I discuss PIL in detail in section 4.7. I use the term throughout the remainder of this section to refer to alleged DSM mental disorders that SP would reject as MMCs.

[199] Indeed, they may even count as a scientific kind for some other science.

[200] See figure 4.2 for a representation of the etiological factors responsible for DSM depression. Lowered-levels of estrogen and lack of sunlight, for example, have both been found to affect serotonin levels in the brain (Douma, 2005; Terman, 2007). It also seems that there may be genetic factors and/or environmental factors that can cause brain changes that predispose one to these MMCs. Environmental factors such as stressful early life events like trauma or sexual abuse, for example, are believed to sensitize Corticotropic Release Factor (CRF) circuits making it likely that people who are genetically predisposed to develop MMC "depression" actually develop it (Nemeroff & Vale, 2005; Slavich, 2010).

4.6.b.ii. Obsessive Compulsive Disorder[201]

The DSM** defines obsessive compulsive disorder (OCD) as a condition in which a person experiences obsessions and/or compulsions.

> Obsessions are defined as persistent, intrusive, distressing thoughts or images that are not "simple excessive worries about real life problems" which the subject attempts to suppress via other thoughts and/or behaviors and that are recognized as being "self-authored" (i.e., not believed by the subject to be "inserted from outside"). Compulsions are defined as repetitive behaviors or "mental acts" the subject feels compelled to perform in order to suppress the distressing images or thoughts (i.e., delusions) but which are excessive or incapable of "neutralizing" the supposed threat represented as the obsession. Additionally, the subject—unless a child—recognizes the "excessiveness or unreasonableness" of the obsessions or compulsions, these obsessions and/or compulsions cause distress in the subject's life, occupy a significant amount of time (at least 1 hour a day) or disrupt one's social or professional life. In the event that another "axis 1 disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g., hair pulling in the presence of trichotillomania)"
>
> (The preceding borrows, and is adopted, from the DSM-IV-TR).

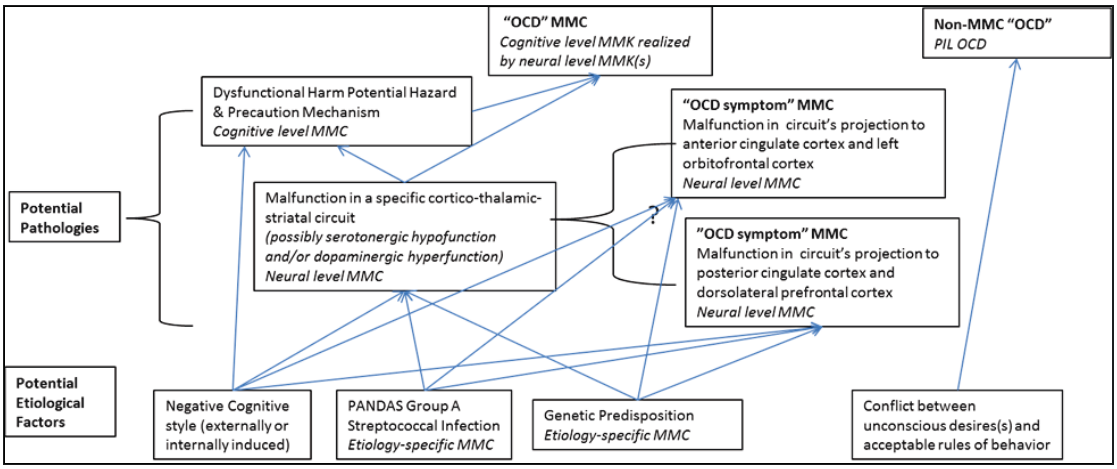

Figure 4.3. Representation of the potential pathologies and etiologies that may give rise to DSM Obsessive-Compulsive Disorder.

[201] The following sources were used to develop this section: Mataix-cols et al., 2004; Mataix-cols et al., 2005; Boyer & Lienard, 2006; Friedlander & Desrocher, 2006; Murphy (2006); Simpson et al., 2006; Gilbert et al., 2008; Harrison et al, 2009.
DSM V has clustered a number of conditions such as hoarding and skin-picking with OCD because they all share the core feature of repetitive behavior. (See APA-DSMb).

Whether or not SP recognizes DSM OCD as a MMC is unclear (see Figure 4.3). There is mounting evidence that DSM OCD actually results from a malfunction in a certain frontostriatalthalamic circuit that in turn causes a posited "Potential Hazard and Precaution Mechanism" to malfunction (Friedlander & Desrocher, 2006; Boyer & Lienard, 2006). Accordingly, DSM OCD *appears* to constitute a MMC. But, there is some debate as to whether all instances of DSM OCD involve malfunctions in the same components of this circuit as particular symptoms have been correlated with breakdowns in specific regions of the circuit (Mataix-Cols et al., 2004; Mataix-Cols et al., 2005). This finding raises the possibility that DSM OCD is actually comprised of a number of sub-classes that involve dysfunctions in specific regions of the frontostriatalthalamic circuit and, therefore, constitute specific MMCs—i.e., "OCD-symptom" MMCs. If it turns out that there is not a more global dysfunction responsible for the symptom specific dysfunctions, then it would appear that DSM OCD does not constitute a MMC—i.e., that it is only a label that picks out a number of MMCs (i.e., "OCD-symptom" MMCs) that sometimes co-occur. SP's stance on the status of DSM OCD as a MMC and, therefore a potential MD, will depend on what the science says.

It should also be pointed out that there is the possibility that some instances of DSM OCD result from no pathology at all, that they result from an internal conflict between one's desires and beliefs. These cases would not constitute MMCs and would, perhaps, be best thought of as PIL OCD. There does not appear to be much evidence that many cases of DSM OCD are of this nature so we should not let the few cases that might exist rob the DSM OCD category of its potential status as a MMC.

4.6.b.iii. Narcissistic Personality Disorder[202]

The DSM-IV TR defines narcissistic personality disorder (NPD) as a condition in which an individual enjoys low self-esteem and is unable to handle criticism but, nonetheless, exhibits a suite of character traits, likely as a defense mechanism, such as: arrogance, an "inflated sense of self-importance", an expectation of being recognized as superior, a longing for admiration, and an expectation of special treatment.[203] Given the lack of current scientific findings regarding DSM NPD, it seems unlikely that SP would recognize it as a MMC (see Figure 4.4).[204] In fact, DSM NPD seems to be a paradigmatic case of a DSM disorder that is actually best thought of as a PIL. And, while it could turn out that some instances of DSM NPD arise as the result of an as yet unknown dysfunctional mental part[205], the majority of instances likely result from personality types combined with a certain cognitive style (i.e., world-view) (Akhtar, 2001).

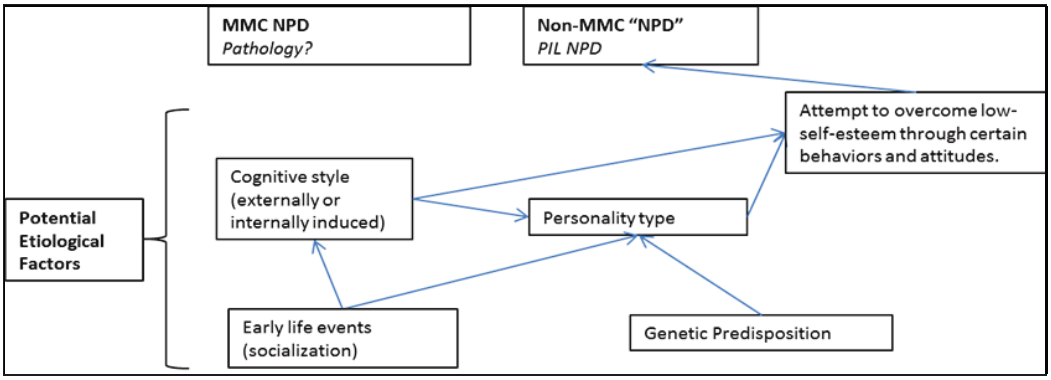

Figure 4.4. Representation of the potential pathologies and etiologies that may give rise to DSM Narcissistic Personality Disorder

[202] There is a dearth of literature on the science, especially the biology, of NPD.

[203] Definition borrowed, and adapted, from the DSM-IV-TR.

[204] See footnote 202 on the lack of scientific (esp. biological) literature on NPD.

[205] I have included the MMC NPD with unknown pathology (i.e., Pathology ?) to indicate that it is possible there may be some instances of DSM NPD that constitute MMCs even though we are not currently aware of them.

Of course, it is possible that there exists a particular gene, or suite of genes, that predisposes one to develop the personality or cognitive style that is responsible for DSM NPD. But even if this were the case, that would not necessarily mean that DSM NPD constitutes a MMC unless it turned out that the predisposing gene(s) caused some mental part to E-systemically malfunction. This is what seems to be missing in DSM NPD—no mental part seems to be E-systemically malfunctioning; rather, DSM NPD just seems to be a phenotypic expression of normal variation in the human genotype. DSM NPD may be a phenotype that society finds despicable and that causes problems for the individual that expresses it but this by itself does not make DSM NPD a MMC.[206] Accordingly, NPD is unlikely to appear in SP's research nosology and, therefore, it will not be a potential MD.

4.6.b.iv. The Paraphilias: pathologies of desire[207]

An interesting set of cases are the alleged "sexual desire" mental disorders, the paraphilias—e.g., sadism, masochism, frotteurism, bestiality, pedophilia, etc. According to the DSM-IV TR, the paraphilias are "recurrent, intense sexually arousing fantasies, sexual urges or behaviors generally involving (1) nonhuman objects, (2) the suffering or humiliation of oneself or one's partner, or (3) children or other non-consenting persons that occur over a period of 6 months [which] cause clinically significant distress or impairment in social, occupational, or other important areas of functioning" (DSM-IV TR). DSM-V moves beyond DSM-IV TR's definition and holds that paraphilias only constitute mental disorders when they "cause distress to the person or harm others". SP will definitely not recognize DSM paraphilias as MMCs since it clearly

[206] It is important to keep in mind that E-systemically functioning systems can give rise to behaviors (or personalities) that are deemed harmful or undesirable by society. The simple fact that a behavior is deemed harmful or undesirable and that there is a genetic predisposition to this behavior does not necessitate that there is a MMC present as the behavior may be the result of an E-systemically functioning system. Furthermore, just because the behavior is not caused by a MMC does not mean that society cannot or will not intervene. Indeed, we may decide that some behavior is biologically normal—i.e., does not involve an E-systemic malfunction—but is, nonetheless, unacceptable by society's standards. DSM NPD and many other personality disorders may be of this type.

[207] Wakefield (2011) discusses the paraphilias in depth and appears to reach conclusions that are, in some ways, similar to mine.

cobbles together cases that involve simple matters of sexual taste with cases that probably involve genuine dysfunctions to mental parts (see Figure 4.5). Furthermore, it is unlikely that many of the particular DSM paraphilias (e.g., bestiality, sadism, etc.) will constitute MMCs as most appear to bring together cases that probably constitute MMCs with cases that are simple matters of sexual taste.

SP would likely recognize cases where there is a malfunction in the impulse control system and, as a result, the individual acts out on his/her questionable sexual desires, as constituting MMCs. Notice, however, that the MMC in this case has to do with the inability to not act on one's desires. Even if the individual had "normal" desires, such as a desire for ice cream, but could not inhibit acting on the desire because of a malfunction in the impulse control system, then that person would still appear to enjoy this "impulse control" MMC (see Figure 4.5). This example illustrates that cases of DSM paraphilia that result from a dysfunctional impulse control system may constitute MMCs but the nature of the desire has little, if anything, to do with why these cases count as MMCs. Accordingly, they would not constitute paraphilia MMCs.

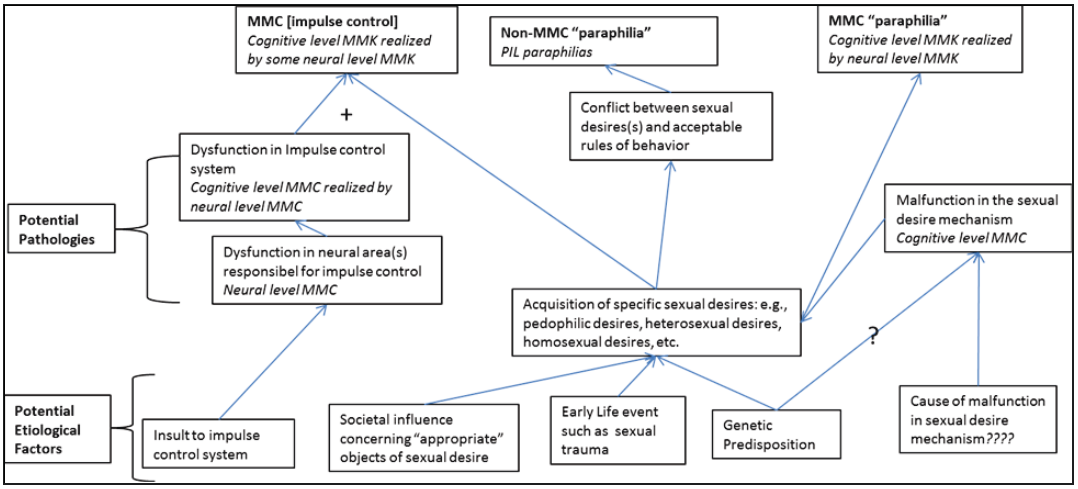

Figure 4.5. Representation of the potential pathologies and etiologies that may give rise to the DSM Paraphilias.

So what about those cases of paraphilia—or any sexual desire for that matter—that do not result from a malfunctioning impulse control system? Do any of those count as MMCs? If

people have a choice about their sexual desires, then whether or not a sexual desire is indicative a MMC will depend on whether or not one's decision-making mechanism is E-systemically functioning. If the decision-making system is malfunctioning, then SP would say that a MMC is present but it is not one that is defined by the sexual desire but by a malfunction in the decision-making system. If one has a choice and the decision-making system is E-systemically functioning, then the paraphilia will not constitute a MMC though it may constitute a PIL. If people do not have a choice about their sexual desires, then whether or not SP would say that some particular sexual desire constitutes a MMC would depend on whether or not the desire results from a malfunctioning sexual desire mechanism.[208] Determining what sorts of sexual desires are indicative of E-systemic function in the hypothesized sexual desire mechanism, however, will depend on what forward-looking evolutionary theory has to say about the contribution that sexual desires make to our ability to survive and reproduce.[209] After all, it could turn out that acting on sexual desires that many consider deviant, including those listed as DSM paraphilias, plays a role in contributing to survival insofar as doing so allows one to decompress in a "safe" manner. It is also possible that other paraphilias like pedophilia may not serve any evolutionary purpose—i.e., they do not improve one's ability to survive and reproduce—but they also do not necessarily result from a dysfunctional sexual desire mechanism since they do not necessarily impact an individual's "ability to reproduce". These may be cases where there is no MMC present but society decides they will not tolerate the behavior—e.g., pedophilia—even though it is considered within the normal biological range—i.e., it does not result from a malfunctioning sexual desire system.[210] SP would see these cases as PIL.

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[208] In this case, strictly speaking, it would be the desire mechanism that constitutes the MMC, not the "sexual desire".

[209] In many cases, such desires will not actually impact our *ability* to survive and reproduce though they might influence whether or not we actually do survive and reproduce.

[210] It is important to realize that SP's suggestion that we not think of the paraphilias as MMCs does not mean that anything goes and we must allow people to act on any and all of their sexual desires. SP has no comment on things such as this as it is simply interested in scientifically understanding break-downs in the mind-brain. Accordingly, it is

A final case worth considering—though one that is clearly not a paraphilia!—is that of homosexuality since some may think SP would be forced to recognize homosexuality as a MMC because it seems to involve a sexual desire that lowers one's chance of reproductive success and, therefore, might result from an E-systemic malfunction.[211] The first thing to point out is that homosexuality does not necessarily impact one's *ability* to reproduce though it may have an influence on whether one actually does reproduce.[212] Accordingly, we would need independent reasons to think the hypothesized sexual desire mechanism is malfunctioning in cases of homosexuality in order to show that homosexuality constitutes a MMC. Such reasons are not at all obvious. Furthermore, there are actually reasons to think that such desires could sometimes be the intended result of an E-systemically functioning sexual desire system (Bartova & Valentova, 2013). It could be, for instance, that evolution has designed the sexual desire system to "go homosexual" in some cases since doing so could help to increase the chances of reproductive

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entirely consistent with SP's findings regarding the paraphilias to devise socially constructed norms for sexual desires that hold the paraphilias to be harmful and undesirable even if they do not result from malfunctioning mental parts.

Let us consider an example. We may decide that adults should not desire to have sex with children or animals and that anyone that does want to have sex with children or animals has "bad desires". Such a claim could be justified by the idea that children and animals cannot consent and consent is an important component of any sexual interaction. Accordingly, sex with children and animals cannot be consensual and is, therefore, problematic. Notice that in this case, it does not matter whether or not the person that acts on the desire to have sex with children or animals has a MMC that is responsible for the "bad desire". What matters is that the person has acted on a desire that society has deemed to be undesirable and/or harmful.

Furthermore, just because there is no MMC present does not mean that intervention, perhaps in the form of chemical castration, is uncalled for. After all, the claim here is not that intervention is only called for when a MMC is present since we may sometimes need to intervene on states that do not constitute MMCs but that are, nonetheless, judged to be harmful and/or undesirable. In these sorts of cases, what matters are the reasons that ground the judgment that the state or condition (e.g., desire) is harmful and/or undesirable. Accordingly, we can explain why heterosexuality and homosexuality are not, and should not be, considered harmful and/or dangerous behaviors, while pedophilia and bestiality should—namely, in normal cases of homosexuality and heterosexuality there is consent (and when there is not consent, then we would judge the instance to be harmful or dangerous—e.g., rape, sexual assault, etc.), while in cases of pedophilia and bestiality there is always a lack of consent from at least one participant. The upshot of these considerations is that even if SP decides that the paraphilias are unlikely to constitute MMCs, we may still want to intervene on some of these cases. The possibility of intervention on states or conditions that do not constitute MMCs but that appear to impact one's mental life is fully consistent with SP insofar as SP recognizes that there are "problems in living" that people might experience which may require intervention.

[211] Notice that the same could be said of heterosexuals that desire to not have children—i.e., that they may enjoy a MMC since they have a desire that lowers reproductive success and, therefore, might result from an E-systemic malfunction. See also footnote 213.

[212] A homosexual person whose sexual organs are not able to contribute to reproduction would still enjoy a MMC even though they may never have sex that could be reproductively successful. The judgment that the person enjoys a MMC issues from the fact that the "ability to reproduce" is compromised.

success in kin-group members. If true, a system that gives rise to homosexual desires would not count as malfunctioning since the system has been designed to go that way in some cases and the ability of the system to reproduce has not been compromised. Of course, even if it did turn out that SP recognized homosexuality constituted a MMC this would not necessarily mean that it would be forced to recognize homosexuality as a MD since not all MMCs are MDs.[213]

In conclusion, SP is likely to eliminate the paraphilias from its research nosology since it is unlikely that SP would recognize many (or any) of the paraphilias as MMCs and, therefore, they would not be potential MDs. Furthermore, most (or all) of the cases of DSM paraphilia that would constitute MMCs would probably do so not because they involve sexual desires specifically but because they involve a breakdown in either the hypothesized sexual desire mechanism or a breakdown in the impulse control system. Accordingly, these would not be instances of MMC "paraphilia" but something else altogether that would be recognized in SP's nosology.

The previous examples were meant to illustrate how SP is revisionary with respect to both its research and clinical/diagnostic nosologies. With this section completed, I now turn my attention to responding to the challenge of "Szasz-ian Anti-psychiatry".

4.7. Challenging SP: Szasz-ian Anti-psychiatry

Up to this point, I have mainly focused on MMCs in an attempt to remain within the purely scientific confines of SP. The background assumption throughout has been that what goes for MMCs in terms of pathology, etiology, etc. also goes for MDs since SP holds that only MMCs are potential MDs. Now, however, I want to consider a potential objection to SP that

[213] Another interesting possibility is that heterosexual people that desire to not have children could also be found to enjoy a MMC if it turned out that this desire for no children resulted from a breakdown in a mental mechanism, perhaps an "offspring desiring mechanism"! The upshot is that MMCs derived from malfunctions in the mental mechanisms related to reproduction could be widespread among heterosexuals.

requires us to explicitly discuss MDs as understood by SP. Recall that for SP, MDs are simply MMCs that have been deemed harmful or undesirable by the relevant/appropriate group. Without going into specifics about which MMCs actually do constitute MDs, I propose to just assume that some do and to assess the proposed objections against these assumed cases. With this strategy in mind, let us now explore how well SP stands up to Tomas Szasz's anti-psychiatry arguments and his insistence that mental disorders are really just "problems in living" (hereafter, PIL).

Szasz's anti-psychiatry view was built around the theory that mental illness (i.e., MDs) understood as a medical condition was a confused and misleading idea. He wrote

> [t]he notion of mental illness [MD] thus serves mainly to obscure the everyday fact that life for most people is a continuous struggle, not for biological survival, but for a 'place in the sun,' 'peace of mind,' or some other human value. For man aware of himself and of the world about him, once the needs for preserving the body (and perhaps the race) are more or less satisfied, the problem arises as to what he should do with himself. Sustained adherence to the myth of mental illness allows people to avoid facing this problem [of deciding how to live one's life], believing that mental health, conceived as the absence of mental illness [MD], automatically insures the making of right and safe choices in one's conduct of life. But the facts are all the other way. It is the making of good choices in life that others regard, retrospectively, as good mental health! (Szasz, 1960, 118)

Within this passage we are given a glimpse into the motivating idea behind Szasz's theory—namely, that MD does not cause one to have a difficult life, rather, life is difficult and making bad life choices causes one to experience the symptoms often associated with MD.[214] But Szasz doesn't stop here. He goes one step further by arguing that MD is actually a myth, while PIL are real but not the subject matter of psychiatry understood as an objectivist medical practice. His argument for this position involves two central claims: 1) if MDs are brain-based illnesses, then they are not MDs but brain illnesses and, therefore, fall within the domain of neurology, not

[214] "Many people today take it for granted that living is an arduous process. Its hardship for modern man, moreover, derives not so much from a struggle for biological survival as from the stresses and strains inherent in the social intercourse of complex human personalities. In this context, the notion of mental illness is used to identify or describe some feature of an individual's so-called personality. Mental illness—as a deformity of the personality, so to speak—is then regarded as the *cause* of the human disharmony. It is implicit in this view that social intercourse between people is regarded as something *inherently harmonious,* its disturbance being due solely to the presence of "mental illness" in many people." (Szasz, 1960, 114)

psychiatry or psychology[215], and 2) what are commonly called MDs are actually just the emotional and physiological responses that humans have when faced with PIL—i.e., dilemmas that result from conflicting values and the "hardships of social interactions"—and, therefore, do not fall within the domain of objective medicine.

For Szasz, there is no need for psychiatry because MDs do not really exist since they are either brain disorders or they are PIL.[216] If the alleged MDs are really reducible to problems in human relations (i.e., if they are actually just PIL), then it would seem reasonable to question the necessity and usefulness of psychiatry (Szasz, 1960, 116). SP, however, is unlikely to be swayed by Szasz's arguments since there is no obvious reason to think that disorders of the brain cannot constitute MDs *because* they constitute brain-disorders. Indeed, insofar as it is a part of SPs theoretical basis that the mental is, in some sense at least, identical to the brain, brain disorders will fall within the realm of psychiatry. SP's approach essentially turns Szasz's objection on its head by taking the mental and the neurological to be coextensive.[217] This conclusion puts pressure on Szasz's claim that MDs are a myth since any brain disorder that is deemed harmful or undesirable will, in fact, constitute a MD.

While the general thrust of Szasz's approach is misguided according to SP, the notion of a "problem in living" (i.e., PIL) is likely to prove useful. After all, SP will need a label for those cases of *alleged* MDs that do not result from malfunctioning mental parts but, nonetheless, are deemed harmful or undesirable. These cases are what SP might refer to as PIL. The sort of cases I have in mind are ones in which a person exhibits behavior that is consistent with MMCs—what

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[215] "I use the word "psychiatry" here to refer to that contemporary discipline which is concerned with *problems in living* (and not with diseases of the brain, which are problems for neurology)." (Szasz, 1960, 116)

[216]"We may recall in this connection that not so long ago it was devils and witches who were held responsible for men's problems in social living. The belief in mental illness, as something other than man's trouble in getting along with his fellow man, is the proper heir to the belief in demonology and witchcraft. Mental illness exists or is "real" in exactly the same sense in which witches existed or were 'real.'" (Szasz, 1960, 117)

[217] I believe that Murphy makes a comment similar to this one in regards to Szasz's position though I do not recall where he does this.

I will call "problematic behavior"—even though there is no malfunctioning mental part that is responsible.[218] A quick glance at the DSM-IV reveals a number of alleged MDs that may be better described as PIL, cases such as histrionic personality disorder, negatively valued psychological (mainly affective) states that result from a conflict between values and lifestyles, many of the personality disorders, and depressive-like state resulting from existential angst.[219] All of these cases, indeed all cases that constitute PIL, share two "szasz-ian" features in common: 1) there is no malfunctioning mental part responsible for the alleged problematic state, and 2) the problematic state issues from either a social conflict with others or a moral conflict within oneself or between oneself and society (Szasz, 1960).[220] SP strongly rejects the idea, implicit in the DSM-IV, that PIL constitute MDs by holding that only those states that involve a malfunctioning mental part constitute a MMC and only MMCs are potential MDs.

Just because a state counts as a PIL and not a MD, however, does not mean that intervention will not be called for. PIL are real and they can, and often do, affect people's lives. For this reason alone, these conditions may need to be intervened on. But there is another reason that it may be important to intervene on PIL, namely, because they can take on the role of an etiological factor and eventually cause a break-down in a mental part if left untreated (i.e., PIL

[218] PIL are like MDs insofar as they often exhibit behavioral/cognitive profiles (i.e., sign & symptom profiles) similar to those associated with MDs. Given the similarity in presentation of PIL and MDs, diagnosis will often require further investigation in order to determine whether a given sign/symptom profile results from a malfunctioning mental part (and therefore constitutes a MD) or from a life-problem. Proper diagnosis in these cases may require an extensive life history in order to understand what sort of PIL is responsible, a necessary piece of knowledge if intervention is to be attempted.

[219] In many cases, PIL probably result from a value conflict (Szasz, 1960): a person's value system is at odds with the values of the society or culture and this leads to negative life situations for the person. Furthermore, the negative life situations that are experienced often depend on a given culture or society. For example, being lazy while holding a strong work ethic and living in a society that values such a work ethic may causes an individual to experience a PIL. If, however, the person lived in a society where such a work ethic was not valued, then it is unlikely the PIL would arise.

[220] One area in which the notion of a PIL may prove theoretically useful is the domain of the paraphilias (i.e., "abnormal sexual desires"). After all, it seems unlikely that we will be able to localize and mechanistically decompose the mental part responsible for sexual desire and we will need to do this if we are going to be able to determine when these desires deviate from the norm because of a malfunctioning mental part or simply because of individual differences in "taste". In fact, many instances of poor cognitive functioning, especially those which result from poor learning strategies and do not involve a malfunctioning mental part, may be best understood as PIL. The same may hold true for many behavioral and personality disorders, especially those common in early adolescence.

may function as etiological factors of MDs). To see this, one need only consider the fact that stress, a common symptom of PIL, can cause actual damage to mental parts resulting in MDs. Of course, all PIL need not give rise to MDs. In some cases, the PIL may just give rise to a symptom suite without ever causing a mental part to malfunction. Even in these cases, intervention may be called for in order to resolve the life problem. In other cases, individuals may be willing to live with the problem or they may be able to resolve the problem without the help of a therapeutic intervention.[221]

Before moving on, one more issue needs to be considered, namely, do PIL fall within the domain of psychiatry. That is to say, are PIL the sort of things that psychiatrists ought to have a theoretical interest in? The answer seems to be both yes and no. On the one hand, the fact that PIL can function as etiological factors suggests that understanding and treating PIL could fall within the purview of psychiatry since they could function as etiological factors of MDs and part of the task of SP is to improve, and maintain mental health, not just restore it once a MD is present. In this case, what brings PIL into the domain of psychiatry is the fact that psychiatry is not only tasked with restoring, but also improving and maintaining, mental health understood as freedom from MD. On the other hand, it seems unlikely that PIL must function as etiological factors since some PIL seem to not give rise to MDs. In these cases, the PIL is unlikely to fall within psychiatry's domain. Of course, as Guze has pointed out, determining whether or not a given PIL is likely to give rise to a MD and, therefore, falls within the domain of psychiatry, probably has more to do with an individual's neurobiology than with the particular life problem (1989).[222] This idea suggests that SP may have a role to play in better understanding what makes

[221] Murphy (2006) may mention this point.

[222]"The point I am making is not that psychologically meaningful experiences are irrelevant to the development of psychiatric disorders. I remain agnostic about their ultimate importance because, in the great majority of instances, these putative causes of psychiatric disorders seem to reflect only the usual range of human troubles that most people experience without becoming ill (Guze & Helzer, 1985). But even if ultimately it can be shown convincingly that these experiences play causal roles in illness, it is to the specific vulnerability that we must direct our attention if we are to hope for essential scientific understanding and effective therapeutic intervention. It appears highly unlikely that an

a person prone to the effects of PIL—i.e., what makes a person prone to develop a MD when confronted with a PIL. All of these ideas taken together suggest that PIL are the sorts of things that SP may need to consider even though it will not always be clear whether or not a given PIL actually does fall within the domain of SP. In any case, I take the above considerations to suggest that the Szasz-ian challenge to psychiatry is not lethal to SP.

4.8. SP-Relevant Therapeutic Interventions

Another aspect of SP that deserves consideration has to do with the sorts of therapies that may be relevant to its practice. This brief discussion is important as there is likely to be a misconception that SP is interested exclusively in psychopharmacological therapies and interventions. In this section I aim to show that this is an unjustified worry and that many therapeutic interventions may prove relevant to SP, even interventions that are based on mistaken psychological theories.

4.8.a. Gene-expression: what makes an intervention relevant to SP

Given my claim that SP will likely recognize many interventions as relevant, it is probably worthwhile to say a bit more about why this is the case. The obvious response in regards to whether or not an intervention is relevant has to do with whether or not the therapeutic intervention is efficacious with respect to the MMCs of SP—i.e., whether the treatment works on MMCs in order to restore proper function to the effected mental part. Answering this question will be quite easy since well controlled studies focusing on pre-treatment and post-treatment outcomes can be run to determine whether the intervention has actually improved the condition—i.e., moved one closer to a state of E-systemic function. Furthermore, given the long history of

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intervention strategy designed to reduce or eliminate the troubles, disappointments, frustrations, and pressures of daily living will prove feasible or powerful enough." (Guze, 1992, 317)

Guze's reasoning explains why two people can experience the same event (a breakup with a spouse) but only one of them will go on to develop a MD (major depression).

many therapeutic interventions, it is often the case that we have extensive data concerning how well these therapies work.

While it is important that we actually have evidence that a therapy works, a more interesting question has to do with how a treatment proves efficacious given SP's understanding of MMCs. A quick response, one suggested by Kandel, is that an intervention will prove relevant whenever it has the ability to affect gene expression (1998). After all, gene expression is responsible for protein development which is in turn responsible for other molecular and cellular level activities that eventually play a role in determining higher-level (i.e., systems level or organism level) traits such as the functioning of mental parts. Therapies that induce gene expressions that restore mental part functioning will constitute relevant intervention therapies. Accordingly, interventions that induce gene expressions that restore, maintain, or improve mental part functioning are relevant to SP.

One might feel that an approach that understands relevance in terms of a therapy's ability to induce gene expression is too reductionist and is likely to focus exclusively on psychopharmacological or biomedical treatments. This worry is unwarranted, however, since we now know that pharmacological and other biomedical interventions are not the only way to induce changes in gene expression. That gene expression can be influenced by environmental factors including interactions with other people is one of the most important findings of twentieth century biology. It has, once and for all, settled the nature vs. nurture debate by showing that phenotype (including MMCs as phenotypes) is almost always the result of both nature AND nurture (Guze, 1989; Kandel, 1998; Jablanka & Lamb, 2005). Given that gene-expression can be influenced by so many things, treating the ability to induce genetic expression as the constraint on relevant therapies is actually quite weak. This constraint is so weak that SP will likely need to include a further constraint on relevant treatments or it runs the risk of treating any sort of intervention—even ones that induce greater malfunction—as therapeutic interventions. In order

to get around this problem, SP could simply impose the further constraint that therapeutic interventions ought to induce genetic changes that lead, or contribute, to E-systemic functioning.[223] And, because SP aims to be theoretically grounded in contemporary science, it could also hold that interventions should only be counted as therapeutic when they have been shown to be therapeutically effective—i.e., when we have empirical studies showing that the intervention increases E-systemic function. The upshot of the foregoing remarks is that if an intervention is found to be relevant to SP, then it will be so in virtue of the fact that it induces the right sort of "therapeutic" gene expression. In the next section, I explore several of the more commonly employed therapeutic interventions of relevance to SP: biomedical therapies, psychodynamic psychotherapies, and cognitive-behavioral psychotherapy.

4.8.b.i. SP relevant therapies: biomedical therapies

It should be clear that the commonly accepted biomedical therapies will be relevant to SP since they meet the appropriate criteria. Biomedical therapies include "[t]reatments for psychological disorders that alter brain functioning with chemical or physical interventions such as drug therapy, surgery, or electroconvulsive therapy" (Gerrig & Zimbardo, 2002). These therapies meet the criteria of relevance for SP because they 1) induce changes in gene expression that contribute to restoring/increasing the E-systemic functions of mental parts, and 2) we have good scientific theory explaining how these interventions can induce these changes in gene expression.

4.8.b.ii. SP relevant therapies: psychotherapy

Many types of psychotherapeutic interventions will fall within the domain of SP. Psychotherapy is an umbrella term I employ to refer to a plethora of intervention strategies that

[223] Of course, we are likely to face some cases where a proposed intervention is not strictly therapeutic given the side-effects of the intervention. In these instances, decisions about whether or not an intervention counts as therapeutic may need to be made on a case by case basis, a strategy that is not uncommon in medical practice in general.

are grounded in "[a] psychological model in which behavior is explained in terms of past experiences and motivational forces; actions are viewed as stemming from inherited instincts, biological drives, and attempts to resolve conflicts between personal needs and social requirements" (Gerrig & Zimbardo, 2002). Psychotherapies aim to "treat psychological disorders, [by] focus[ing] on changing faulty behaviors, thoughts, perceptions, and emotions that may be associated with specific disorders" (Gerrig & Zimbardo, 2002). Accordingly, a unifying feature of these therapies is an emphasis on restoring mental health by changing "behaviors, thoughts, perceptions, and emotions" in non-biomedical ways. Meltzoff & Kornreich offer a more developed definition of psychotherapy:

> Psychotherapy is taken to mean the informed and planful application of techniques derived from established psychological principles, by persons qualified through training and experience to understand these principles and to apply these techniques with the intention of assisting individuals to modify such personal characteristics as feelings, values, attitudes and behaviors which are judged by the therapist to be maladaptive or maladjustive. (1970, 4)

The connection between psychotherapy and SP may seem problematic since psychotherapy has often been employed as an intervention strategy for states that SP would not count as MMCs (i.e., for states that count as PIL). Nonetheless, even if this form of therapy is often used for PIL it also seems to be used, at least sometimes, for *bona fide* MMC. Furthermore, there is now considerable evidence that some forms of psychotherapy are efficacious insofar as they can lead to gene expressions which can have a therapeutic impact on mental functioning. Getting clear on which sorts of psychotherapy are actually efficacious is a project beyond the scope of this work. It is likely, however, that some forms will prove to be useful and effective therapies, and therefore, recognized as such by SP.

It may be worthwhile to take a few moments to consider the place of psychoanalysis—a particular form of psychotherapy—in SP. After all, any discussion of psychiatry risks being incomplete if it fails to say something about psychoanalysis since it dominated and influenced the

field of psychiatry for so many years (Paris, 2005) and still enjoys a certain presence in North America and Europe, though one that is definitely on the fringes of mainstream psychiatry, in the form of psychoanalytic institutes. Psychoanalysis, like other forms of psychotherapy, is broadly understood as a practice that employs dialogue with the patient as a means to treat "mental disorders" by helping the patient better understand the causes of, as well as potential strategies to deal with, the "disorder".[224] Where psychoanalysis diverges greatly from most other forms of psychotherapy is in its theoretical commitments and explanation of mental disorder.

While psychoanalysis is itself a varied practice with a long and checkered past, there are core features of the discipline that can be pinpointed:

1) the importance of events in early childhood
2) the role of unconscious (and irrational) desires and drives that are responsible for much conscious behavior and thought
3) the role that defense mechanisms play in maintaining the unconscious,
4) the idea that mental disturbances often result from conflicts that result from unconscious drives/desires and conscious views
5) the belief that the examination of "dreams, symptoms, and unintentional small acts" can reveal one's unconscious desires and drives
6) the idea that psychological treatment of the mental disturbances is achieved by making the unconscious conscious.

(adapted from Fromm, 2013)

That psychoanalysis may be relevant to SP is beyond doubt, at least to the extent that psychiatry can, has, does, and should make use of some of the concepts developed and employed by psychoanalysis. As Kandel has pointed out, concepts such as transference, the unconscious, and repression have proven useful in helping us better understand the mind-brain (1999). That these

[224] This section on psychoanalysis draws heavily from the following sources:
http://www.nimh.nih.gov/health/topics/psychotherapies/index.shtml;
http://www.mayoclinic.com/health/psychotherapy/MY00186
http://www.apa.org/divisions/div12/aboutcp.html

concepts have proven useful, however, does not say much about how the main tenets and theoretical commitments of psychoanalysis are, or are not, relevant to SP.

The exact role of psychoanalysis in SP becomes unclear once we turn our attention to the potentially conflicting ways that mental disorders are understood in these two systems. If we focus on psychoanalysis' claim that mental disturbance—i.e., mental disorder—is supposed to issue from a conflict between unconscious drives that are the result of functioning mental parts and one's "conscious experience of reality", then it seems at odds with SP's idea that mental disorders essentially involve a malfunctioning mental part. Accordingly, psychoanalysis would need to be left out of serious consideration by SP. Of course, it could be argued that the mental disturbances posited by psychoanalysis are not meant to be of the same nature as the mental disorders posited by SP. It could be, for instance, that the mental disturbances of psychoanalysis are more akin to Szaszian "problems in living" (1960). Szasz's comments on psychoanalysis seem relevant to this point. He writes

> *"[p]sychoanalysis is a moral dialog, not a medical treatment*. Psychoanalysis has nothing whatsoever to do with illness or health, medicine or treatment, or any other idea that places "professional" listening and talking within the purview of the state's licensing authority as "treatment." If the practice of psychoanalysis is not a form of treatment, what is it? It is a modern reincarnation of the age-old cure of souls as secular-existential dialogue" (Szasz, 2002).

If Szasz's understanding of psychoanalysis is correct, then it is unclear why it would have any relevance to SP since "problems in living", understood as a theoretical construct, do not result from malfunctioning mental parts. If we understand the mental disturbance (perhaps still understood as a "problem in living") that results from the unconscious drive/conscious reality conflict as an etiological factor of a MMC (i.e., as a potential cause of a malfunctioning mental part), then psychoanalysis may have a relevant role to play in SP, namely, providing insight into a certain sort of etiological factor. Besides this role, the theoretical commitments of psychoanalysis

seem to place it outside the domain of SP since it does not seem to be concerned with the malfunctioning mind-brain. Nonetheless, psychoanalytic therapies may prove useful for intervention on genuine MDs—and, therefore, relevant to SP—but not for the reasons that psychoanalysts have imagined. I will have more to say about this point—namely, how a therapeutic intervention can be effective even when the theory that grounds the therapy is incorrect—in section 4.8.c.

4.8.b.iii. SP relevant therapies: cognitive behavioral therapy

The third type of therapy that might be relevant to SP is a special type of psychotherapy, cognitive-behavioral therapy (hereafter, CBT). CBT is "[a] therapeutic approach that combines the cognitive emphasis on the role of thoughts and attitudes influencing motivations and responses with the behavioral emphasis on changing performance through modification of reinforcement contingencies" (Gerrig & Zimbardo, 2002). Leichsenring et al., describe the manner in which CBT is carried out:

> Patients and therapists work together to identify and understand problems in terms of the relationship between thoughts, feelings, and behavior. The focus lies in the here and now. Individualized, usually time-limited therapy goals are formulated. CBT intends to directly target symptoms, reduce distress, re-evaluate thinking and promote helpful behavioral responses. The therapist supports the patient to tackle problems by harnessing his or her own resources. Specific psychological and practical skills are acquired (e.g., reflecting and reevaluating the meaning attributed to a situation with subsequent behavior changes) and the therapist actively promotes change with an emphasis on putting what has been learned into practice between sessions ("homework"). The patient learns to attribute improvement to his or her own efforts (self-efficacy). A trusting and safe therapeutic alliance is viewed as an essential ingredient, but not as the main vehicle of change. (2006, 234)

CBT is a relevant intervention for SP since it meets the two requirements. First, there is good scientific understanding of how the therapy induces changes in gene expressions that can lead to improvement in the E-systemic function of mental parts (Paquette et al., 2003; Fuchs, 2004; Porto et al., 2009). Second, CBT has proven particularly effective as a means of dealing with OCD and depression (Gould et al., 1997) and there is a body of evidence suggesting that employing CBT in

conjunction with psychopharmacological therapies such as SSRI's can have a synergistic therapeutic effect (Smits et al., 2010; Potenza et al., 2011).[225]

It is important to realize that the above therapeutic interventions are not solely relevant to SP in the same way that the therapeutic interventions relevant to medicine are not solely relevant to medicine.[226] Exercise, for example, is sometimes employed as a therapeutic intervention for diseases (e.g., heart disease), but this does not mean that exercise belongs solely within the domain of medicine. Sometimes exercise is employed for purposes that have little, if anything to do, with the aims of medicine. In the same way, psychotherapy is sometimes employed as a therapeutic intervention for MDs and sometimes it is employed to deal with PIL. Biomedical approaches and CBT might also be used to deal with non-MMCs such as PIL when the employment of such interventions is believed to be worthwhile and justified. Determining what justifies the use of one sort of therapeutic intervention over another when it comes to MDs or PILs, however, is an issue that is beyond the scope of this paper. What is clear is that many sorts of interventions will likely prove useful and justified when it comes to dealing with these varied conditions.

4.8.c. How to be right (efficacious) and wrong (theoretically incorrect) at the same time

Before concluding this section, it is important to explain how it is that a therapy such as psychoanalysis could possibly prove relevant to SP given that psychoanalysis is built upon a psychological theory that is largely disproven. Put another way, the issue that needs to be

[225] For somewhat conflicting findings, see Otto et al., (2009).

[226] Whether or not we consider psychotherapeutic practices as parts of SP, or tools employed by SP, is an interesting question. It is very similar to the one that asks whether we should understand mathematics as a part of physics or as a tool that physicists employ to understand physics? I do not have an answer to either of these questions except to say that any part of SP will need to respect the core tenets of SP, in particular, the idea that MMCs and MDs are partially objective states in the sense previously discussed. Insofar as a practice accepts these core tenets, then whether or not it is considered an actual part of SP or a tool for SP seems theoretically unimportant.

discussed is how a treatment or intervention can be efficacious even though it is grounded in a theory that has been shown to be false. To understand how this can happen, we should consider the hypothetico-deductive method of theory testing. According to the H-D method, one begins with a hypothesis and then deduces a conclusion(s) that follows from the hypothesis. One then looks for evidence that is inconsistent with the deduced conclusions as a means to falsify the original hypothesis (Popper, 2014). When such evidence is not found, the hypothesis is maintained. Unfortunately, many people mistakenly think that the original hypothesis is corroborated, or confirmed, when the deduced conclusion(s) is found to hold. The problem with this sort of reasoning is that theories tend to be underdetermined by evidence (Duhem, 1914; Quine, 1951; Stanford, 2009). Evidence is said to underdetermine scientific theories insofar as two or more competing theories may be "well-supported" by the same body of evidence. (Stanford, 2009) To appreciate why underdetermination causes problems for the H-D view, we need only realize that the deduced conclusions do not function as evidence solely for the proposed hypothesis since they may also function as evidence for another competing hypothesis.[227] Furthermore, any observed conclusion that is taken as evidence can always be made to be consistent with other theories insofar as we are willing to alter auxiliary assumptions of these competing theories (Quine, 1951; Quine & Ulliman, 1978). For instance, one may think that evolution is evidence against the existence of god, but it can be made evidence in favor of the existence of god if we assume that god may have chosen to "create" humans via evolution.

The above remarks lead us to several lessons concerning theories and evidence. Lesson 1 is as follows:

[227] To assume that the deduced conclusion functions as evidence solely for the proposed hypothesis is to commit the logical fallacy of affirming the consequence:

H -> E
E_____ INVALID!!
H

1) A theory can predict a given outcome (observation) and nonetheless be false, or a single observation or set of observations can be evidence in favor of multiple, mutually exclusive theories.

A corollary of lesson 1 is lesson 2.

2) A practice may work even though the explanation for why it works is incorrect.

As an example of lesson 2, let us consider the case of staying warm to ward off colds. For many years, people thought that colds were acquired as a result of exposure to low temperatures (Zuger, 2003). Accordingly, physicians of the time would direct people to stay warm so that they would not get a cold. The explanation behind the practice was that exposure to low temperatures caused colds in people. As it turns out, people do not get colds because of exposure to low temperature. Rather, colds result from exposure to a certain virus. Nonetheless, the practice of staying warm—i.e., protecting oneself from exposure to low temperature—actually did, and still does, help one not catch a cold (Mourtzoukou & Falagas, 2007). But the actual explanation(s) for why this practice works is quite different than that originally proposed. One explanation is that exposure to low temperature puts strain and stress on the body which, in turn, causes the immune system to be compromised, and this means the body is more vulnerable to invading organisms. By protecting oneself from exposure to low temperatures, one ensures that the immune system is as strong as possible and this increases the body's ability to fight off an invading cold virus. As you can see, the practice works even though the original explanation offered for why it works—and, perhaps even the original justification for why the practice should be implemented—was false. Psychoanalysis is a lot like the practice of staying warm to prevent a cold. Both of these "interventions" prove efficacious but the reason they do has little to do with the original theories upon which they were developed.[228]

[228] The above lessons brings up the following question: If a practice can work even if the explanation for why it works is incorrect, and if some observation can function as evidence in favor of multiple, mutually exclusive theories, how do we know when we have actually discovered a true theory. Put another way, whatever justifies accepting one theory over another? The position that I will be taking is as follows: one is justified in positing the truth of X if the truth of X

4.9. Conclusion

This chapter has attempted to spell out the theoretical commitments of SP. I have explained how SP fits with the Partial-Objectivist understanding of medicine and how it differs from contemporary psychiatry. I have also tried to say something about the sort of MCs—MMCs—in which SP is likely to be interested. In particular, I have suggested, in line with Murphy (2006), that SP is likely to have much to say about the MMCs involving perception, attention, memory, sensation, movement, and affect, while it is unlikely to have much to say at present about breakdowns in the mental parts responsible for cognitive functions like reasoning, decision-making, and desiring. I have also offered some comments on how SP's nosology is likely to be structured and explained how SP can respond to the Szasz-ian Anti-psychiatry challenge, while still making use of the Szasz-ian notion of "problems in living". Finally, I have discussed the sorts of therapies that are likely to be relevant to SP.

In conclusion, it may be worth reminding the reader that the work in this chapter was an attempt to say what psychiatry ought to be like if it is to count as a branch of partial-objectivist medicine. Given this goal, the chapter has moved beyond the more or less descriptive methodology that was employed in earlier chapters when I was getting clear on the nature of medicine and has taken on a normative bent. A result of the normative nature of this chapter is that certain features of SP may actually differ from features of psychiatry as it is currently understood. Indeed, SP is likely to appear as a revisionary account of psychiatry, primarily because of its recognition that the malfunctioning mind-brain constitutes its domain. Though SP is revisionary, one of its strengths lies in the fact that much of contemporary psychiatry is already

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is consistent with the epistemic state of our current best science at time T (i.e., combined body of scientific knowledge) and there is evidence that is consistent with X that is not consistent with the next best competitor theory P. Accordingly, we are justified in claiming the truth of the theory of evolution since it is consistent with the epistemic state of science and there is evidence in favor of evolution that is not consistent with creationism (e.g., the fossil record and theory about carbon dating). It could, of course, turn out that evolution is false if we were to uncover new scientific knowledge that altered the overall epistemic state of science or if we found new evidence that was in favor of creationism but inconsistent with evolution (i.e., if we found the dating of the fossil record was grossly inaccurate and carbon 14 and other forms of radiologic dating were horribly incorrect).

heading in its theoretical direction. That is to say, much of contemporary psychiatry seems to be committed to the idea that psychiatry is a branch of medicine that ought to be theoretically grounded in the mind-brain sciences (Kandel, 1998; Paris, 2005). Accordingly, we ought to expect much of SP to be fairly uncontroversial.

Chapter 5: SP, Mental Health Professions, and Public Policy

5.1. Introduction

In the last chapter I offered a detailed description of SP, my version of psychiatry understood as a branch of medicine. In this chapter, I consider and respond to a potential objection to SP. The objection, which takes the form of a *reductio* argument, holds that SP should be rejected since it requires that we radically reconceptualize how we think about mental health and mental disorder, and this reconceptualization would entail changes in mental health policy and our understanding of the mental health professions that most theorists would find unacceptable. I respond by explaining why this worry is unwarranted, misguided, and ultimately fails to count against acceptance of SP.

The chapter proceeds as follows. In section 2, I discuss the objection in greater detail. Section 3 introduces a response meant to defuse the objection; namely, that there are two concepts of mental health that serve different purposes in different realms of discourse. In order to explain this response, I consider two other areas of inquiry—race and health—where scientific and sociological concepts may diverge for similar reasons. The upshot of this is the idea that scientific concepts and sociological concepts are employed for different purposes and are not, therefore, competitors. In section 4, I offer further evidence in favor of the two concepts response and explain why accepting SP would not negatively impact mental health policy. I conclude with some remarks on SP's relation to other mental health professions.

5.2. The Objection

The *reductio* objection against SP runs as follows: If we accept SP, then we will be forced to accept a radical reconceptualization of mental health and mental disorder that would: 1) suggest that many mental health professions are not actually interested in mental health, and 2)

impact public policy in a negative manner. Most people (i.e., psychiatric theorists, mental health professionals, the public-at-large, etc.) would be unwilling to accept this. Hence, SP should be rejected. Let me spell out the specific aspects of the objection in more detail.

The first aspect of the objection holds that SP's understanding that mental health consists of freedom from mental disorder where mental disorders just are malfunctions in mental parts that are deemed harmful or undesirable entails that mental health consists entirely of normal—i.e., biologically normal—mental functioning. If correct, then it seems that accepting SP would force us to accept a notion of mental health that is simply inconsistent with that which must be posited if we wish to be able to explain why the various mental health professions count as mental health professions. After all, not every mental health profession is primarily interested in restoring the biological function of mental parts. Accordingly, one may think that SP's understanding of mental health must be misguided since it is a notion that seems to be irrelevant to many mental health professions.

The second aspect of the objection centers on the worry that acceptance of SP will likely impact mental health policy in a negative manner. A prime example of how policy might be affected has to do with the possibility that some sectors—especially, private insurance companies—could argue that many conditions and causes that mental health professionals believe impact mental health should not be covered by health insurance since they are not really components of mental health as understood by SP. Indeed, one could imagine a move by proponents of this view to design mental health policy to treat only those mental disorders that are recognized by SP as deserving of coverage. Such a move, allegedly made possible by SP's understanding of mental health, would negatively impact mental health policy since it would likely deprive millions of people of treatment that is needed to fully function in society. In essence, this aspect of the objection argues that if SP does not recognize things other than

biological function as relevant to mental health and, therefore, candidate issues for mental health public policy, then that would suggest that SP cannot properly understand mental health.

Answering the *reductio* objection is necessary for several reasons. First and foremost, we should be concerned if SP proves to be as problematic as the objection contests. I take it as a given that the mental health professions most certainly have something to do with mental health and that negatively impacting mental health policy does count as a *prima facie* argument against an account of psychiatry. Fortunately, as I will argue shortly, accepting SP does not entail the problems that the objection alleges. Another reason to consider this objection is that doing so will help us to better appreciate and draw out as fully as possible the ramifications of accepting my way of thinking about psychiatry. In particular, I am keen to explore SP's relationship with the other mental health professions and whether these other professions are bound by SP's notion of mental health and mental disorder. In the end, answers to these questions will play a major role in my response to the *reductio* objection.

5. 3. Responding to the objection: Step 1—The two concepts strategy

In this and the next section I respond to the preceding objection by noting that there are, in fact, multiple concepts of mental health that serve varying purposes. Two of these concepts are of particular importance to answering the objection: the biological and the sociological concepts of mental health. The biological concept of mental health is the one that SP employs since it aims to be a branch of biologically based medicine. Given SP's grounding in functional biology, it ought to be clear that its guiding theoretical concepts, including mental health, will be biologically based. What is somewhat novel and needs to be explained is the idea that there is another concept of mental health—the sociological concept—that is relevant to policy debates and, perhaps, most of the other mental health professions.

But before I say anything more about these two concepts, it is probably worthwhile to say a few words to defend my strategy from the charge of *ad hoc-ness*. As a defense, I intend to show that many theoretical terms have multiple concepts which apply to differing domains and serve multiple purposes. If true, this should lighten suspicion that my claim that we need to recognize both a biological and a sociological concept of mental health is *ad hoc* and unjustified. I begin with the case of race and then consider the case of health.

5.3.1. The Case of Race

In recent years there has been much debate centered on the question of whether races—e.g., Caucasian, Asian, African—actually constitute natural classes that are scientifically interesting in the way that species classes allegedly are. Many have assumed that the status of these classes as natural is tied to the proposed races possessing

> "biobehavioral essences: underlying natural (and perhaps genetic) properties that (1) are heritable, biological features, (2) are shared by all and only the members of a race, and (3) explain behavioral, characterological, and cultural predispositions of individual persons and racial groups" (Mallon, 2006, 528–529).

Nowadays, almost no one thinks that any of the classes traditionally considered races actually possess such "biobehavioral essences" (Appiah, 1996; Mallon, 2006; Kitcher, 2007). This fact has led many theorists to become racial skeptics and/or racial eliminativists. Racial skeptics doubt that races constitute natural classes, while racial eliminativists propose eliminating the concept RACE from scientific and/or policy discussions (Mallon, 2006, 2007). Those sympathetic with racial skepticism and eliminativism generally cite the rather strong conditions on natural racial categories, and the inability of any class to meet these conditions, as central to their position (Appiah, 1996). Not everyone accepts the skeptical/eliminativist view. Some theorists have argued for "racial population naturalism," the view that there could be "genetically significant biological groupings" which could be considered races even though these would not be discrete groups that enjoy specific cultural, mental, or physical characteristics (Kitcher, 1999).

The guiding thought behind the racial population position is that the concept RACE is much like the concept SPECIES and racial classes are much like species classes: racial classes and species classes are similar because they constitute scientifically interesting groupings that share "population-level features" even though they lack essences or discrete boundaries, while the concepts RACE and SPECIES are similar because they are scientifically useful concepts that can, and should, play a role in scientific discourse (Mallon, 2006). The "racial population naturalism" position differs from the essentialist position because it redefines the criteria that categories must meet in order to count as natural. The final position to consider is racial constructivism. Racial constructivists[229] recognize that no classes meet the natural category criteria but the concept RACE still refers to groupings of people that exist as a result of "human cultures and decisions" (Mallon 2006, 2007; Haslanger, 2008). Racial constructivists, unlike essentialists and population naturalists, deny that racial categories would exist independent of human reasoning and decision-making.

So how do these three positions on race stand up to scrutiny? First and foremost, in keeping with my earlier employment of the notion of P-scientific kinds, I want to frame the discussion of race and concepts of race in terms of scientifically useful classes. Accordingly, it could turn out that there are actually several concepts of race that are employed for different purposes and that are used to refer to distinct P-scientific kinds. Interestingly, there do appear to be at least two primary purposes for which the concept of race is employed. First, the concept is employed for biological purposes—i.e., races are posited as biologically significant classes that figure in biological theory. Second, the concept is employed for policy and sociological purposes—i.e., races are posited as sociologically (and, perhaps, culturally) significant classes that figure in sociological theory and public policy discussions.[230] Given that these purposes are

[229] This position is sometimes referred to as "constructionist anti-essentialism." See Mallon (2007).

[230] The second purpose is also scientific—it is a social science project.

somewhat divergent and are components of different sorts of scientific projects, it seems reasonable to suppose that there are actually two concepts of race at play that refer to two different P-scientific kinds: one biological and one sociological (see also Haslanger, 2000, 2008).

When it comes to biology, it seems highly unlikely that races constitute scientifically useful classes—i.e., biological P-scientific kinds (Appiah, 1996; Mallon, 2006). This follows not just because it is unlikely that races possess the "biobehavioral" essence that allegedly must exist if races are to constitute natural categories but also because it is unlikely that races, understood as classes, will prove to be scientifically useful for any branch of biology. Recall that one of the upshots of the discussion of scientific kinds was that some particular class may constitute a scientific kind for one science but not for others. Species, for instance, may not constitute P-scientific kinds for functional biology or genetics even though they may constitute P-scientific kinds for evolutionary biology. The justification for this claim was that species will likely support the sort of P-laws of interest to evolutionary biology but not those of interest to other branches of biology. When it comes to race, it is unlikely that any of the alleged classes of race will support biological P-laws. One might argue that races understood as genetically similar populations—i.e., the racial populationist position—could support the relevant sort of P-laws to justify treating races as biological P-scientific kinds. The problem with this move, however, is that it is unclear that we would gain any explanatory footing by doing so since it seems that racial classes do not support any biological P-laws that would not be supported by the larger class *homo sapien* which subsumes all racial classes.[231] Furthermore, the social constructivist notion of race doesn't offer a notion of races as biological P-scientific kinds at all since the social or cultural component enjoys theoretical primacy in determining what counts as a race. Note that this fact about social constructivism does not mean that races do not constitute P-scientific kinds of any

[231] Kitcher (2007) seems to recognize this fact when he notes that there may no longer be any races in the population naturalist sense even though there were in the past. In the same paper, he seems to suggest that racial classes may still constitute scientifically interesting classes.

sort—only that they do not constitute biological P-scientific kinds since biological P-scientific kinds are not socially constructed.

The upshot of these considerations is skepticism concerning biological concepts of race. Put another way, the upshot is denial of races as P-scientific kinds and, therefore, a rejection of the concept of race as a biologically useful theoretical construct. The point here isn't that there is no biological concept of race; rather, it is that the biological concept RACE does not refer to anything suggesting that this notion of race—i.e., the idea that races area biological scientific kinds—needs to be eliminated (Appiah, 1996; Mallon, 2006).

When it comes to sociological purposes such as policy decisions, however, we may nonetheless have a reasonable concept of race available. Indeed, the notion of races as socially constructed classes seems entirely appropriate for policy issues and other sociological matters. After all, insofar as policy is sometimes designed to deal with socially and culturally originated problems, it seems entirely possible that we may need to discuss classes that are also primarily, and in the first order, socially constructed (Appiah, 1996; Haslanger, 2000, 2008). Race is, it seems, just such a class. Consider, for example, the fact that much has been done to negatively impact people of different races throughout American history. For many centuries in America, racial categories were employed as a means of sorting people into groups which enjoyed varying amounts of freedom and protection under the law. Even though the case can be made that races understood as biological classes did not exist even when race was used as a means to classify and sort people in the past, the employment of these racial categories still had a very real impact on people's lives. These categories may still be relevant and the sociological concept RACE may still refer even if we now realize that what the concept was originally intended to pick out—i.e., a unique group of people that enjoy a biobehavioral essence or a population level essence—does not exist (Mallon, 2006). Our reasons for employing the sociological concept RACE may issue solely from the fact that we wish to make amends for the wrongs that were imposed upon people

as a result of the faulty biological concept RACE. In this case, the sociological concept RACE does not pick out a natural class; rather, it picks out a group of people that share the property of being grouped by what are now known to be faulty biological concepts of race.

There are both potentially negative and potentially positive consequences of endorsing the employment of the sociological concept of race. The potential negative upshot of employing this concept is: 1) that some people are likely to misunderstand this endorsement to suggest that races exists as a biological reality even though we have every reason to think this is not true, and 2) that it will entice people to follow race-specific "scripts" that might be damaging and harmful (Appiah, 1996). The potential positive role of employing this concept is that it provides a means to attempt to right previous wrongs against certain groups via the use of policies such as affirmative action (Haslanger, 2000, 2008). For affirmative action to work, we need a concept of race that respects the divisions—i.e., "racial categories"—that we now know to be scientifically suspect (Haslanger, 2000, 2008). Accordingly, we can decide to construct a concept—i.e., the sociological concept RACE—that respects those divisions that we used to believe tracked racial categories. By employing such a concept, we are able to have a positive impact on public policy by implementing affirmative action and other related policies.

The employment of two concepts of race can go a long way to handling many of the challenges that arise when we attempt to deal with issues concerning race in the biological sciences and the public policy arena. We can deal with the fact that we will need to employ racial categories in order to right past wrongs by noting that the relevant concept of race is one that is either historically or sociologically grounded—i.e., that what determines race are historical matters of fact or sociological convention. And, we can handle the potential negative consequences of employing and endorsing the sociological concept of race by noting that questions concerning the reality of race as a biological category are posed at the biological level. To this question, the answer is resolutely that there is no such thing as race. The upshot is that

making sense of the many ways that race is considered probably forces us to posit multiple concepts of race that serve different purposes. Understanding which concept applies when and what that tells us about the issue under discussion is a task that might require much work. Having two concepts will prove useful.

5.3.2. Two Concepts of health

In chapter 2 of this work, I argued that even though there may be some broad, vague notion of health understood as well-being, there is a more narrow understanding that is central to core medical practices which takes health to involve freedom from disease. The problem with the broader notion of health, I proposed, was that it was too vague to properly characterize medicine since it likely includes things within its domain that are intuitively not part of medicine—i.e., relaxing trade barriers, education reform, etc. The more concise notion of health, on the other hand, seemed to do a better job of respecting the actual boundaries of medicine. The take home message of the second chapter was that medicine is likely interested in health understood as freedom from disease.

But we must be careful not to think that the broader notion of health is without merit. In many cases, it appears that the relevant notion of health may be the one that is understood as having to do with well-being of some sort. The World Health Organization (WHO), for instance, defines health as "…a state of complete physical, mental, and social well-being and not merely the absence of disease or illness."(WHO, 1946/2006, 1) This concept of health is likely to play an important role in public policy since it takes such a broad, encompassing approach to understanding what it means to be healthy. This is also likely to be the notion of health that explains why practices such as exercise, a healthy diet, education, sanitary living conditions, appropriate sewer systems, healthy economies, etc. are believed to be components and factors of health.

Interestingly, by noting that health is "not merely the absence of disease or illness", the WHO appears to recognize that health understood as well-being is not the only relevant concept of health. Accordingly, WHO seems to recognize two concepts of health that need to be posited and that serve somewhat different, but also overlapping, purposes. The biological concept HEALTH, the concept relevant to medicine, holds that health just is freedom from disease. The sociological concept HEALTH, the concept relevant to public policy, holds that health is a state of physical, mental, and social well-being. The biological concept is primarily employed for scientific/therapeutic purposes, while the sociological concept is employed for sociological/policy purposes. The two concepts share some overlap insofar as biological health is often a pre-requisite for sociological health though it does not guarantee it. After all, simply being free from disease does not insure one is living in a state of "physical, mental and social well-being".

Maintaining these two concepts of health is important for they serve different purposes. The purpose of health understood as freedom from disease is to offer a biologically based way to think about the practice of medicine. This concept of health is scientific—i.e., it is a biologically grounded concept. The purpose of health understood as well-being is to offer a way to think about what is necessary for people to live fulfilling human lives understood multi-dimensionally. This concept of health is also scientific though it is probably best represented as a sociological concept of health since determining what counts as well-being is likely to depend on societies and/or cultures understanding of well-being. This is not to discount the scientific nature of this concept (indeed, there might turn out to be a proper science of well-being); it is only to point out that it is different than the functional biological concept.

5.3.3. Lessons Learned

Several lessons can be taken from the previous examples of race and health. First, we should be willing to recognize that there are often multiple concepts at play when it comes to

theoretical terms that figure in multiple scientific and non-scientific projects. Accordingly, we should constantly keep an eye out for hidden multiple concepts. Second, we should be wary of letting a concept have too much influence outside of its domain. This is just to say, we shouldn't allow a concept's inadequacy to apply to all domains be grounds to reject the legitimacy of the concept; rather, we need to recognize that concepts may have limited and circumscribed domains of application. The biological concept HEALTH—i.e., the notion of health as freedom from disease—for instance, seems inadequate as an account of health as it is employed in policy debates. This does not mean the biological concept HEALTH is incorrect; rather, it simply means it may not be the concept that is employed in sociological contexts. Furthermore, we need not maintain that concepts from differing domains should be considered irrelevant outside of their own area—this claim is not supported. My point has simply been to offer a reminder that we need to be careful of allowing ourselves to be held hostage by any particular concept. For the present case, we should be careful not to hold policy hostage to the biological concept of health, or let the inadequacy of the biological concept of health for policy issues be grounds to question its usefulness as a biological concept, especially one that is focused on understanding the appropriate domain of medicine. Let us now turn our attention to mental health.

5.4. Responding to the Objection: Step 2—Two Concepts of Mental Health

Recall that the first aspect of the objection was that SP's understanding of mental health would force us to concede that only those professions and practices that are interested in mental health, understood in the manner of SP, should count as mental health professions and that this is a concession that most theorists would be unwilling to make. Hence, SP and its notion of mental health should be rejected. What I will now suggest is that the objection fails because what holds for health also seems to hold for mental health—that there are at least two concepts of mental

health that, in conjunction, explain why SP is a branch of medicine and why the mental health professions are mental health professions.

For psychiatry understood as a branch of medicine, the concept of mental health appears quite specific; namely, it is biologically based and it essentially involves freedom from malfunctioning mental parts that are deemed harmful or undesirable. But we have reason to think that this concept of mental health does not properly capture the sense of mental health relevant to many other mental health professions since some of these professions do not seem to be focused on mental health understood as freedom from mental disorder. A summary description of the various mental health professions will help us to better appreciate these practices and how they might understand mental health.

- Psychiatrists are mental health professionals that possess either an MD or an OD. They are medically trained and must perform a residency and then pass a board exam in neurology and psychiatry. These aspects of the psychiatrist's training indicate the intimate connection between psychiatry and medicine, a connection that acts as further evidence for the claim that psychiatry is generally recognized to be committed to the medical model or biomedical approach (NAMI).

- Clinical psychologists integrate "science, theory, and practice to understand, predict, and alleviate maladjustment, disability, and discomfort as well as to promote human adaptation, adjustment, and personal development. Clinical Psychology focuses on the intellectual, emotional, biological, psychological, social, and behavioral aspects of human functioning across the life span, in varying cultures, and at all socioeconomic levels." (APA, div12) These practitioners generally hold either a PhD or PsyD.

- Counselors and Psychotherapists may possess anyone of a number of degrees including, but not limited to, a PhD, MA, or MS. These degrees could be from psychology departments, psychotherapy institutes, or counseling programs. The field of "counseling psychology encompasses a broad range of culturally-sensitive practices that help people improve their well-being, alleviate distress and maladjustment, resolve crises, and increase their ability to function better in their lives. With its attention to both normal developmental issues and problems associated with physical, emotional, and mental disorders, the specialization holds a unique perspective in the broader practice-based areas of psychology." (APA, div17).

- Clinical social workers possess either a Master of Social work, Doctorate of Social Work, or a PhD. These professionals often play a role by collaborating with other mental health professionals, but they can also offer treatment services, often in the form of psychotherapy (NAMI).

The first point to appreciate about these professions is that much of their focus seems to center on optimizing and improving people's mental lives, a service that does not necessarily involve "fixing" malfunctioning mental parts. Of course, improving one's mental life may sometimes require fixing or dealing with a mental disorder (and this explains why psychiatry is considered a mental health profession), but this is not always the case. Typical reasons one may visit a mental health professional such as a counselor, clinical psychologist, or social worker include, but are not limited to, relationship problems, self-esteem problems, grieving, difficulty coping with stress, etc. These problems, which do not necessarily issue from any sort of malfunctioning mental part are, nonetheless, recognized to be relevant to mental health insofar as the professions that deal with them are understood to be mental health professions. Taken together, these points raise the following question: what is the concept of mental health that is relevant to the mental health professions as a whole—i.e., what concept of mental health, if any, unifies the mental health professions?

The answer to this question, I propose, is found in the WHO definition of Mental Health:

> *'... a state of well-being in which the individual realizes his or her own abilities, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to his or her community.'*
>
> In this positive sense mental health is the foundation for well-being and effective functioning for an individual and for a community. This core concept of mental health is consistent with its wide and varied interpretation across cultures. It is more than the absence of mental illness, for the states and capacities noted in the definition have value in themselves (WHO, 2004, 2005, 23).[232]

The above definition offers a clear conception of mental health that ably unifies the mental health professions; namely, mental health professions are such because they are all interested, in some way and in some form, in mental health understood as the proposed state of well-being. Importantly, this concept by itself fails to capture all that a concept of mental health should since

[232] The 2004 summary report leaves out the line "It is more than the absence of mental illness, for the states and capacities noted in the definition have value in themselves" and the 2005 full report leaves out the line "This core concept of mental health is consistent with its wide and varied interpretation across cultures." I have included both lines since they help to flesh out the idea I am after.

it does not allow us to make sense of SP as a medical practice that is scientifically grounded. For this, it seems we need the biologically based concept of mental health, the one that sees mental health as freedom from mental disorder. If we heed the lessons learned from the examples of health and race, then the obvious next move is to hold that there are at least two concepts of mental health that need to be posited in order to deal with the relevant issues. One concept is biological and helps us understand psychiatry understood as a branch of medicine. The other is sociological and helps us make sense of the unity of the mental health professions.

Let us consider these concepts in a bit more detail. The biological concept MENTAL HEALTH holds that mental health consists of freedom from mental disorder—i.e., freedom from breakdowns in mental parts that are deemed harmful or undesirable. This concept is the one of primary relevance to psychiatry. The sociological concept MENTAL HEALTH holds that mental health is a state of well-being in which one can cope with daily life and contribute to one's community. Employing the terminology of chapter 4, while the biological concept of health is intimately connected with mental disorder (MDs), the sociological concept of mental health seems to be connected, primarily, with problems in living (PIL). After all, a variety of issues that are classic cases of PIL (e.g., relationship problems, self-esteem issues, normal life stresses such as job loss that people may have trouble coping with) are exactly the sort of things that seem relevant to the sociological concept of mental health. Furthermore, the sociological concept of mental health seems to be the one that unifies the mental health professions since the variety of practices employed by these professions—practices ranging from restoring biological mental health to grief counseling, existential guidance, spiritual guidance, behavioral problems, retraining of cognitive styles, positive thinking, etc—all seem to be aimed at maintaining, restoring and improving mental health understood as the proposed state of well-being. The biological concept does not unite the mental health professions; instead, it unites psychiatry with core medical practices.

It is worth pointing out that these two concepts enjoy a relation of subsumption: the biological concept of mental health subsumes the sociological concept insofar as sociological mental health (i.e., well-being) requires biological mental health (i.e., no dangerous or harmful malfunctioning mental parts). Because the biological concept subsumes the sociological concept, it seems to follow that the mental health professions besides psychiatry will also be interested in biological mental health though this will not be their sole focus. Importantly, this is what we find when we look at these professions: at least some of the services they offer appear to be focused on maintaining, improving, and restoring biological mental health, while others are focused on improving, maintaining, and/or restoring sociological mental health. It also worth pointing out that SP is interested in the sociological concept of mental health insofar as the sorts of conditions relevant to sociological mental health—namely, PIL—are recognized by SP as potential etiological factors of MDs. By recognizing two concepts of mental health, we are able to see that the first aspect of the objection is unwarranted and misguided and, therefore, fails to count against SP.

The recognition of two concepts of mental health also helps us deal with the second aspect of the objection which holds that SP's understanding of mental health will negatively impact public policy. The first thing to note is that this worry is primarily misguided as it is entirely possible that the mental health concept of relevance to public policy is the sociological, not the biological, one. If the sociological concept is the one relevant to the policy arena, then we need not worry that SP's understanding of mental health will negatively impact public policy since it will be irrelevant, for the most part, to such issues. Not only is it possible that the sociological concept is the one of relevance to the policy arena, it seems quite clear that it is designed with the purpose of playing a primary role in this area. That this is the case, and that it should be the case, does not speak negatively of SP or SP's understanding of mental health. Indeed, SP can fully embrace and endorse the use of the sociological concept of mental health in

policy debates and the public arena since the project of attempting to understand SP was never motivated by a desire to redefine the domain of the mental health professions; rather, it was carried out in order to place psychiatry on a firmer scientific footing.[233]

Not only does the acceptance of SP not negatively impact mental health policy, it may actually have a positive impact by illuminating the role of PIL in MDs. SP's recognition that bad input and PIL can function as the etiological factors of MDs opens the door for proponents of mental health policy to argue that a more serious policy approach should be taken toward these conditions (i.e., PIL). After all, if we know that PIL are especially apt to cause (i.e., as etiological factors) MDs, then there is a good case to be made that properly dealing with MDs will include dealing with PIL. By making the connection between PIL and MDs clear, SP offers scientific theory that explains why PIL should be taken seriously. This theory provides a good argument that mental health can be improved by developing policy that extends coverage to the treatment of PIL.

Before concluding this chapter, it may be worthwhile to consider a quick objection that might arise in response to positing two concepts of mental health that enjoy a subsumption relation. The objection asks why both concepts are necessary if one subsumes the other. As already noted, the major impetus for positing two concepts has to do with the fact that the biological concept of mental health is probably not going to prove useful for policy debates or for explaining the unity of the mental health professions since it was devised with the narrow focus of clarifying the domain of psychiatry understood as a branch of medicine. We also have noted that it is quite common for there to be multiple concepts of important ideas, especially when we are dealing with differing domains of inquiry and separate, even if related, projects—e.g., domain-describing vs. policy projects. That this is the case seems to be the motivation for the

[233] Of course, if a particular policy issue calls explicitly for input from the biological sciences, we can always employ the biological concept of mental health. However, when this is not explicitly called for, it seems that the better concept to employ in the social and policy arena—indeed, the one that actually is employed—is the sociological concept.

WHO's proposal of multiple concepts of health and mental health (WHO, 2004, 2005, 1946/2006). Finally, the employment of both concepts allows us to make sense of psychiatry as a branch of medicine and the existence of the so-called mental health professions.

5.5 Conclusion

In this chapter I have dealt with a potentially pernicious objection to SP. In order to deal with this objection, I have shown that accepting SP does not threaten the status of other mental health professions as *mental health* professions and that it will not obviously negatively impact mental health policy. In arguing for this, I have proposed that there appear to be two concepts of mental health that serve different purposes—one is sociological, the other is biological. In thinking about these various concepts of mental health, I also attempted to offer a clearer picture of the relationship between SP and the other mental health professions. I close this chapter with several remarks on SP and the mental health professions:

- All mental health professions—psychiatry included—are interested in improving, maintaining, and restoring Mental Health where Mental Health is understood as *'... a state of well-being in which the individual realizes his or her own abilities, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to his or her community.'*(WHO, 2005, 23)
- Psychiatry is interested in the restoration, maintenance, and improvement of mental health where mental health is understood as freedom from MDs.
- Psychiatry as a mental health profession focuses on understanding and treating MDs. The domain of psychiatry and what counts as a MD should be confined to MMCs that are deemed harmful or dangerous. Psychiatrists may be interested in PIL as etiological factors.
- Problems in Living (PIL), even those that do not function as an etiological factor of a MD, may still be of interest to other mental health professions since they affect mental health understood as well-being.
- Other mental health professions may also aim to treat MDs, but, in addition to this, they also aim to treat other conditions/things that may impact mental health (namely, PIL).

Chapter 6: Conclusion

In this work I have developed and discussed Scientific Psychiatry (SP). Achieving this goal required that I fulfill three sub-goals. These sub-goals were:

1. develop a greater appreciation of the medical model that psychiatry is supposed to adhere to by investigating actual medical practices in order to determine the theoretical and methodological commitments of medicine (chapters 2 & 3).
2. interpret psychiatry in terms of my proposed view of medicine, Partial-Objectivism, in order to develop the SP account (chapter 4).
3. explain how SP is related to other mental health professions and public policy (chapter 5).

By meeting these three sub-goals, I have successfully reached my over-arching goal of developing and discussing SP, my medical model account of psychiatry. SP, however, is not a completely novel account. Indeed, it essentially borrows from, and further develops, existing medical model accounts of psychiatry (e.g., Guze, 1989, 1992; Andreasan, 1997; Kandel, 1999; Hohwy & Rosenberg 2005; and Murphy, 2006). The upshot of employing this strategy to develop SP is that it has provided a position that is broadly consistent with, but also better performing than, its theoretical predecessors.

In the process of developing the SP account, I have had to leave out many topics that would have been interesting to discuss. For instance, I was not able to spend much time discussing the clinical aspect of medicine since my project was essentially centered on appreciating the theoretical foundations and commitments of medicine in order to devise a framework to interpret psychiatry. Nor have I been able to discuss all the potential components of SP. I have not, for example, offered SP's assessment of the current catalogue of alleged mental disorders as found in the DSM-IV TR and the DSM-V though I have suggested that SP is likely to be revisionary with respect to its nosology. What I have tried to do in this work is offer the principles to which SP seems to be committed. By making these principles clear and by

offering examples, I hope to have made it easier to see how the account may handle specific issues such as constructing a nosology and understanding causes for intervention purposes. An added bonus of clarifying these principles is that it should make it easier to critically assess and identify shortcomings in the SP account. Much work remains to be done. I leave that for future theorists to pursue.

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Addendum

This project discusses the DSM-IV TR and a number of mental disorders/conditions from this manual. In the spring of 2013, the DSM-V was published and replaced the DSM-IV TR. The DSM-V, unlike the DSM-IV TR, attempted to be more theoretical insofar as there was a drive to classify mental disorders in terms of their underlying causal mechanisms. Though this was the intent of much of the committee tasked with developing the DSM-V, there is very little mention of causal mechanisms in this newest version of the manual (Gever, 2009, Grohol, 2009). Indeed, the DSM-V, like its predecessor, continues to primarily offer a symptomatic (i.e., phenotypic) description of mental disorders. Because 1) the DSM-V and the DSM-IV TR are similar in how they understand and classify mental disorders and 2) the cases of mental disorder I discuss changed very little from the DSM-IV TR to the DSM-V, my remarks on the DSM-IV TR are still relevant (Ghaemi, 2013). Accordingly, it seems unnecessary to change my discussion to the DSM-V since the changes are not relevant to my project. If anything, DSM-V seems to embody an even more confused state than its predecessor (Francis, 2012, 2013). When there are differences between these two manuals that are relevant to this project, I have mentioned it in the text or a footnote.

Curriculum Vitae

Bryan Miller was born in Spartanburg, South Carolina. Before pursuing his doctorate in philosophy at Johns Hopkins University in Baltimore, Marlyand, he received a BA from Georgia Southern University, an MA in Religious Studies (esp. Indological Studies) from the University of Georgia, and an MA through the Philosophy and Brains & Behavior program at Georgia State University. His areas of specialization are philosophy of science and philosophy of psychology.