

UNIVERSITY OF CALGARY

Against Naturalist Conceptions of Health: In Defence of Constrained Normativism

by

Juan David Guerrero

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES
IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE
DEGREE OF DOCTOR OF PHILOSOPHY.

DEPARTMENT OF PHILOSOPHY

CALGARY, ALBERTA

AUGUST, 2011

© Juan David Guerrero 2011



UNIVERSITY OF
CALGARY

The author of this thesis has granted the University of Calgary a non-exclusive license to reproduce and distribute copies of this thesis to users of the University of Calgary Archives.

Copyright remains with the author.

Theses and dissertations available in the University of Calgary Institutional Repository are solely for the purpose of private study and research. They may not be copied or reproduced, except as permitted by copyright laws, without written authority of the copyright owner. Any commercial use or re-publication is strictly prohibited.

The original Partial Copyright License attesting to these terms and signed by the author of this thesis may be found in the original print version of the thesis, held by the University of Calgary Archives.

Please contact the University of Calgary Archives for further information:

E-mail: uarc@ucalgary.ca

Telephone: (403) 220-7271

Website: <http://archives.ucalgary.ca>

Abstract

My aim in this dissertation is to raise the question 'Is there such a thing as a satisfactory “scientific” conception of health and disease that *could* inform important health issues?' and to argue that there is not. I argue that this result carries profound consequences—medical, legal, ethical, political, philosophical, economic, environmental, and social—that impact most (probably all) individuals, though they may not express it or appreciate it.

In Chapter 1, I build upon Searle’s dyadic objective-subjective distinction and propose a tripartite framework, which introduces some needed conceptual clarity into the core issues at play in the modern debate of health and disease. In Chapter 2, I enhance this framework and use it to provide a more detailed articulation of the fundamental difference between the two main sides in the modern debate—naturalism and normativism—than what is usually found in the literature. I conclude the chapter by arguing that we should take seriously the naturalist *modus operandi* to advance a value-free science of health and disease.

In Chapter 3, I critique the most influential naturalist theory of health, Christopher Boorse’s ‘biostatistical theory’ (BST). I argue that the BST essentially involves a *Cambridge-change* criterion, which, in fact, betrays the entire naturalist project.

Chapters 4-6 assess the naturalist’s prospects for advancing a satisfactory conception of health and disease that avoids “Cambridge-change objections”. Strategies such as understanding health and disease as “natural kinds”, saving the BST with different accounts of “normality” and of “function” occupies the focus of Chapters 4, 5 and 6, respectively. Regrettably, none of these strategies prove successful.

In Chapter 7, I anticipate and contest one final naturalist strategy. Shifting focus, I

argue that a much more serious encounter with normativism is needed. Further still, I argue that there is a decisive reason to openly embrace a normativist conception of health and disease. I sketch my own outline of such a conception of health and disease, which provides a very important bulwark against some genuine and substantial dangers. Further implications and strategies for change are proposed.

Acknowledgements

This dissertation would not have been possible without the assistance, guidance, and encouragement from some important people. First and foremost, I am deeply grateful to my parents, David and Lesley, who offered unconditional support. I would like to acknowledge my sister, Mar, and grandfather, Lester, who were both a source of inspiration and support.

I have benefited greatly from having the two supervisors I did. I wish to acknowledge my great indebtedness to John A. Baker, an ideal supervisor. I have benefited immensely from his continuing encouragement and constructive criticism of previous drafts of this dissertation. His uncanny ability to inject clarity to what I had written or said never ceased to amaze me. Walter Glannon was a source of much insight. I have benefited from his thought-provoking questions and his willingness to respond to my drafts quickly and closely.

I am grateful to Marc Ereshefsky who introduced me to the philosophical debate of health and disease.

Finally, for a variety of reasons, thanks are owed to my friends Megan, Kimberley, Rob, and Waller. I owe a special thanks to Lindsay.

Dedication

For my parents

To whom I owe much

Table of Contents

Approval Page	ii
Abstract.....	iii
Acknowledgements.....	v
Dedication.....	vi
Table of Contents.....	vii
List of Tables	x
Epigraph.....	xi
 CHAPTER ONE: Health and Disease: The Modern Debate	 1
1. Introduction	1
1.1 Two important caveats	3
2. Distinguishing between “naturalist” and “normativist” accounts of health	4
3. Following Searle to a point: Two different “senses” of the objective-subjective	11
3.1 Setting the stage.....	11
3.2 Searle’s epistemic/ontological contrast	16
3.3 Searle’s distinction between “brute” and “institutional” facts	19
3.4 What we can extract from Searle’s objective-subjective distinction	23
4. Re-orienting the objective-subjective distinction	27
4.1 An ontological contrast (O*).....	28
4.2 Epistemological contrast (E*)	30
4.3 Semantic contrast (S*).....	31
4.4 Summing up.....	36
 CHAPTER TWO: The Distinction between Naturalism and Normativism.....	 39
1. Introduction.....	39
2. The question of the semantic and ontological objectivity of health judgements is the basic issue between the naturalist and normativist	41
3. The varieties of normativism.....	48
3.1 Three broad varieties.....	48
3.2 Local normativist differences in their accounts of objectivity and subjectivity.....	58
4. Moving forward.....	62
 CHAPTER THREE: On a Naturalist Theory of Health: A Critique	 70
1. Introduction	70
2. The Biostatistical Theory (BST).....	77
2.1 Biological normality and the BST.....	79
2.2 Statistical normality and “Cambridge-changes”	81
3. On the BST advancing a Cambridge-change criterion	85
3.1 Dynamic statistical norms	85
3.2 Important implications.....	86
3.3 Impacting the theoretical normality of health (and disease).....	87
3.4 Cambridge-changes and the BST: A closer look.....	94
3.4.1 An “internal” tension.....	94

3.4.2 Medical thought and practice	97
4. Concluding remarks	100
CHAPTER FOUR: Defending Naturalism with Natural Kinds?	102
1. Introduction	102
2. Natural kinds	108
2.1 Reznek's argument that disease is not a natural kind	112
3. An important essentialist point: Specific diseases may be natural kinds	118
4. Disease <i>qua</i> disease does not constitute a natural kind of the sort traditional essentialism avers	123
4.1 An essential or microphysical structure of things common to all "diseases" eludes our best science	124
4.2 The difference between health and disease is not an all-or-nothing difference in kind	127
4.3 Whether some state is to be counted a diseased state is context-dependent..	132
4.3.1 The "vestigial organ" (VO) argument	133
4.3.2 The "evolutionary change" (EC) argument	134
4.3.3 Environmental factors	136
5. Chapter conclusion	144
CHAPTER FIVE: "Normality"	146
1. Introduction	146
2. Three conceptions of normality	147
2.1 Evaluative normality	148
2.2 Statistical normality	151
2.3 Biological normality	154
3. Does an adequate account of normality need to be at some level statistical?	157
4. Normality and homeostasis	161
4.1 Cannon and Boorse on homeostasis	162
4.2 Why Homeostasis is not sufficient for health	167
4.3 Homeostasis: A rejoinder?	169
4.4 The argument revisited: A two prong objection	170
4.4.1 The need to extend the model of homeostasis to the cellular level	170
4.4.2. Homeostasis is not a model that captures how many cells successfully function	172
4.5 The emergence of three serious problems	174
4.6 Section conclusion	178
5. Physiology	181
6. Chapter conclusion	186
CHAPTER SIX: "Normal Functioning"	189
1. Introduction	189
1.1 The rôle of statistical considerations in addressing qualitative and quantitative issues in relation to functions	189
1.2 Narrowing the focus	196
2. The BST's account of "function" and thence of "normal functioning"	199

2.1 The fundamental role of the BST's account of function.....	200
2.2 Why the BST's current account of function is open to Cambridge-change objections.....	206
2.2.1 Species design, functions, and normal functioning	207
2.2.2 Boorse's "general goal-contribution" account of function.....	213
2.2.3 Section Conclusion	224
3. Important implications	227
3.1 Backward-looking accounts of function	230
3.1.1 The distant/ancient-past looking accounts	230
3.1.2 The recent-past looking accounts	241
3.2 Forward-looking accounts of function	244
3.3 Present-looking accounts of function.....	250
4. Chapter conclusion	254
CHAPTER SEVEN: Conclusions and Prospectus	255
1. Introduction.....	255
2. On a "reverse engineering" strategy: Some doubts	257
2.1 On the basic assumption(s) required	258
2.2 On the solutions to the qualitative issues about functions.....	261
2.3 Section conclusion.....	270
3. Further implications and strategies for change.....	272
3.1 Taking naturalism seriously	272
3.2 Taking normativism seriously	278
3.3 Health: A Canadian perspective	286
3.3.1 Account of Health #1.....	287
3.3.2 Account of Health #2.....	289
3.3.3 Identifying values and norms: A proposal.....	291
3.4 A concluding challenge	294
3.4.1 Constrained normativism	296
References.....	305
Appendix A.....	324

List of Tables

7.1 Increasing Disparity in Income in Canada (1980-2005).....	282
7.2 Percentage of Change in Income in Canada (1980-2005).....	283

Epigraph

If you talk to God, you are praying;
If God talks to you, you have schizophrenia.
If the dead talk to you, you are a spiritualist;
If you talk to the dead, you are a schizophrenic.

— Thomas Szasz, *The Second Sin*¹

The philosophers have only interpreted the world, in various ways;
The point is to change it.

— Karl Marx, *Theses On Feuerbach*²

¹ Thomas Szasz (1973), *The Second Sin*. Garden City, New York: Anchor Press, p. 101.

² Karl Marx (1845), *Theses On Feuerbach*. Marx/Engels Internet Archive.

<<http://www.marxists.org/archive/marx/works/1845/theses/theses.htm>> Accessed July 4, 2011.

Chapter One

Health and Disease: The Modern Debate

1. Introduction

The purpose of this chapter is to bring to light as clearly as possible the underpinnings of two major perspectives on the nature of health and disease. These two perspectives have come to play a large role in the writings on health and disease of philosophers, health researchers, health educators, health practitioners, political theorists, health activists, health lobbyists and others, though they may not express it or appreciate it. To that end, this chapter argues that an important and useful tripartite distinction can be drawn within the modern debate between what in some circles has been referred to, perhaps misleadingly, as “objective” and “subjective” aspects of health and disease. My hope is to provide a framework from which to better recognize the way these so-called “subjective” aspects are often employed, sometimes explicitly and other times without discussion, to advance health claims that can be scientifically dubious and quite often ideologically tendentious.

The literature shows the modern debate over health and disease to be between two sides: usually this literature calls the sides respectively “naturalism” and “normativism”.¹

¹ The names “naturalism” and “normativism” are not ideal, but I will use them because they are firmly embedded in the literature, though it should be said there are notable exceptions. Dominic Murphy, for instance, employs the names “objectivism” and “constructivism” to denote the two major sides because, he writes, “both ‘naturalism’ and ‘normativism’ are used in other senses in a variety of debates”. See, Dominic Murphy (2009), “Concepts of Disease and Health”, in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. <<http://plato.stanford.edu/archives/sum2009/entries/health-disease/>>. Accessed July 4, 2011. For similar reasons Peter Schwartz instead employs the terms ‘value-requiring’ (VR) and ‘non-value-requiring’ (non-VR). See, Peter H. Schwartz (2007), “Decision and discovery in defining ‘disease’”, in H. Kincaid & J. McKittrick (Eds.), *Establishing medical reality*. Dordrecht: Springer, 47-63.

Though, as will emerge in this chapter, this formulation is very rough and in fact too rough for productive discussion. Nevertheless, as a first approximation we can take naturalists as contending that health status is both objectively and at least in principle entirely empirically discernable. The alleged upshot of this “realist” conception is that their account of health has a theoretical foundation in “value-free science”. Normativists contend that the state of health is, in some manner to be specified, essentially a function of values or of norms²—a satisfactory account of health is thus taken to be inextricably laden with values or norms.

This is the kind of rough-and-ready way the literature distinguishes normativism from naturalism. But it is important to recognize that it is by no means clear what this contrast can or should be thought of as coming to in detail, let alone what would be the implications for our understanding of the nature of health and disease from the decision to opt for either a naturalist or normativist conception. At least part of the problem is that there is a real sense in which theorists take naturalism and normativism to be abstract receptacles in which to couch quite different—and often opposing—sorts of content. This is perhaps not surprising given that one can hardly deny that there are many possible and in some cases even plausible ways to cash out what it is to be a “naturalist” and “normativist” about how to conceive health and disease. Indeed, objective/subjective,

² Where a norm is (i) a direct standard of evaluation of actions and responses of people; the standard (ii) being created by human acts intended to create norms or a function of human convention; and (iii) prescribing certain courses of action or responses (including affective responses) and supported by the prospect of ostensibly legitimate critical reactions ensuing upon the deviation from the norm which make it, *pro tanto*, a guide for people’s behaviour. See Joseph Raz (1980), *The concept of a legal system*, 2nd ed. Oxford: Clarendon Press, at pp. 122-127 & 147-166. I am indebted to John A. Baker for bringing this work to my attention.

objectivist/constructivist, descriptive/non-descriptive, discovered/created, value-free/value-laden, norm-independent/norm-relative, facts/values are just some³ of the different distinctions that disagreement between and within the two major positions has been stated in terms of. And because of this, to get a clear sense of the modern debate over health and disease, it is necessary to examine the underpinnings of the naturalist/normativist distinction (and its relations to the other abovementioned distinctions). It is to this task that we shall now turn, but first we must pause to make explicit two important caveats regarding this dissertation.

1.1 Two important caveats

(I) The broad usage of “disease”

For the purpose of this dissertation, unless otherwise explicitly stated, when I use the term “disease” I mean to invoke a very broad understanding of disease—one that is much broader than one typically finds in ordinary language-use. That is, I will be using “disease” as a technical definition that is meant to cover the multifarious conditions (e.g., cancers, physiological injuries, mental disorders, genetic disorders, traumas, and so on) that medical science counts to be inconsistent with perfect health.

In this usage, I am following the most influential naturalist theorist, Christopher Boorse, whom, as we shall see soon enough, defines health as the absence of disease. In my view, such a broad usage of disease is thus needed for a charitable interpretation of,

³ See, for instance, Bjørn Hofmann (2001), “Complexity of the concept of disease as shown through the rival theoretical frameworks”, *Theoretical Medicine* 22: 211–236, where he states there to be over 70 different different ways the concept of disease has been categorized.

and for an efficient critique of, naturalism. Furthermore, when I speak of “disease” I will, by implication, be speaking also of “health” (i.e., the absence of disease) and vice versa, which avoids some verbiage.

(II) Mental health and disease

For reasons that will become clear, it will be important to view “health” and “disease” in terms of *physiological* normal and subnormal functioning. What is to be stressed is that this understanding readily extends to neurological diseases or, to use the provocative words of Tomas Szasz, “proven brain diseases”.⁴ I think Szasz’s words are so provocative precisely because there is such genuine controversy over the relationship that exists between physiological health and disease, and mental health and disease. Here I concede, with regrets, that some of my discussions will be limited by skirting around the core issues of the controversy, but these are matters simply far too large and complex to entertain here. However, that being said, I submit that many arguments advanced in this dissertation could—and should—be extended to an understanding of mental health and disease. My hope is that the tripartite framework advanced in Chapters 1 and 2 will bring to light the extent to which our mental health and disease judgments do, in fact, depend on values and norms, especially when denied or not explicitly stated.

2. Distinguishing between “naturalist” and “normativist” accounts of health

I mentioned above that a claimed upshot of adopting a naturalist account of health is that

⁴ Thomas Szasz (1997), “Mental illness is still a myth”, *Review of Existential Psychology and Psychiatry* 23(1/2/3): 70-80.

such an account of what counts as health would have a theoretical foundation in value-free science. The purportedly value-free sciences in which the naturalist typically seeks to ground an account of health are the *hard* sciences (e.g., biology, chemistry, physics), which are generally thought to be more objective and, importantly, less subject to possibly dubious cultural influences and less a matter of personal “choice” (less subjective in that sense) than the *soft* sciences (e.g., history, psychology, political science).

To be clear: whether this characterization of the “soft” sciences is correct is not the important thing. What is important is that the aim of the naturalist is to advance an objective science-based account of health. That is to say, the naturalist aims for an account grounded in and informed by an objective and presumably empirical account of the state or the nature of organs and organisms, such that it is an account, which in some specified sense makes health status independent of value and culture.

So as a methodological point we may put aside any concerns we may have over whether the “hard” sciences are in the end a value-free or culture independent enterprise: fortunately, we need not take a stand on this controversial issue; for our purposes, it will suffice to leave this an open question. It is methodologically acceptable to do this because all the naturalist surely requires is for her account of health and disease to be no more value-laden than the hard sciences are.

This is an important point that is worth expanding upon. I suspect that the hard sciences in defending the scientific claims they want to make either explicitly or implicitly depend on so-called epistemic values such as truth, accuracy, coherence, and simplicity.⁵

⁵ Not surprisingly, philosophers disagree over what these so-called epistemic values of science are. An

But practically speaking we can still draw a principled and profitable distinction between the values that in the way just indicated do play a fundamental and defensible rôle in the hard sciences and those values that do not. What I want to suggest is not unlike Hilary Putnam's proposal for a "disinflation" of the fact-value dichotomy where we recognize:

there is a distinction to be drawn (one that is useful in some contexts) between ethical judgements and other sorts of judgements. This is undoubtedly the case, just as it is undoubtedly the case that there is a distinction to be drawn (and one that is useful in some contexts) between *chemical* judgements and judgements that do not belong to the field of chemistry.⁶

The moral I wish to draw is thus: we will do well to abandon the notion that even if one were to establish a fundamental normative dimension to the hard sciences then it would, *ipso facto*, sound the "game-over" buzzer for the naturalist project concerning health and disease.

Hence, I want to suggest the following connections: We tie naturalism to the view that, at bottom, it is possible to advance a satisfactory account of health which is an objective, empirically based, value- and culture-*independent* account; and we tie normativism to the view that, at bottom, a satisfactory account of health will perhaps inevitably be in some important sense value or culture *dependent* and maybe even perhaps in some sense subjective. This way of adumbrating the main difference between the naturalist and the normativist accounts of health shows the need for some examination of

important and influential view is Quine's five virtues of a hypothesis: *conservatism*, *modesty*, *simplicity*, *generality*, and *refutability*. See W. V. Quine & J. S. Ullian (1978), *The web of belief*, 2nd ed. New York: Random House, pp. 64-82. I will not pursue the issue of the ostensibly value-independence of science as it would unnecessarily take us too far afield. For those so inclined, especially as it pertains to medical science, an interesting discussion may be found in William E. Stempsey (1999), *Disease and diagnosis: Value-dependent realism*. Boston: Kluwer Academic.

⁶ Hilary Putnam (2002), *The collapse of the fact/value dichotomy and other essays*. Cambridge, MA: Harvard University Press, at p. 19.

the notions of objectivity, subjectivity, and value and culture independence. To this I now turn.

Perhaps the safest way to characterize what all of the normativist accounts have in common is to characterize them as abandoning (with or without regrets) the notion that a satisfactory account of health needs to be value- and culture-independent. Instead, and this can be viewed as what unites normativist theorists, the normativists argue or perhaps merely conclude that a viable account of health must in some way or another reference values or norms—and, as such, is importantly disjoint from the approach and goals adopted by the naturalists and perhaps the hard sciences.

Under a united stand against naturalism, however, lies extensive disagreement among normativists both over just what these requisite values and/or norms are, whose they are, what rôle they are to be taken as playing in characterizing health and disease, and the extent to which a satisfactory account of health can be disjoint from the sciences. In these discussions, the normativist side has introduced into the modern debate diverse theories of health determination, theories which make such determination either value or culture dependent (or indeed both): thus, for example, we find Fulford's "reverse view"; Nordenfelt's "welfare theory"; Pörn's "equilibrium model"; and Richman's "embedded instrumentalist" theory of health.⁷

So we may characterize the fundamental difference between naturalists and normativists in the modern debate of health and disease as lying in the ways in which they

⁷ See, for instance, K. W. M. Fulford (1989), *Moral theory and medical practice*. Cambridge: Cambridge University Press; Lennart Nordenfelt (2001), *Health, science and ordinary language*. Amsterdam: Rodopi Press; Ingmar Pörn (1993), "Health and adaptedness", *Theoretical Medicine* 14: 295-303; and Kenneth A.

answer the following question: Is it the case that a satisfactory account of health and disease cannot but reference human values or norms that are *beyond* what can reasonably be included in an objective science-based account in the way briefly indicated above?

Naturalists will answer in the negative; normativists will answer in the affirmative. This brings to the fore the fact that the issue between naturalists and normativists is at bottom the issue of whether an organism's properly considered health and disease status is simply an objective culture- and value-independent matter or whether it also involves essential reference to possibly values and culture-dependent norms, and perhaps even to subjective elements. Accordingly, all accounts of health and disease either will explicitly or implicitly have allegiance to one side or the other. This characterization of the contrast between the core ideas of the two approaches illuminates the sense in which the literature comfortably draws a distinction, neat and precise or not, between the two major positions in the modern debate concerning health and disease.

Once we characterize the basic differences between naturalist and normativist accounts of health and disease in this way, then it is easy to see that a significant task that remains is to persuasively articulate what a *satisfactory* understanding of health and disease will be from the perspective of either a naturalist or a normativist framework.

This is precisely what many theorists in the modern debate have attempted to do by advancing different and opposing articulations from within the major positions. But it is to be noticed that extensive disagreement *amongst* theorists of the same major camp has led to the introduction of a number of "locally" different ways of stating positions which

nevertheless are to be viewed as belonging to the same camp.

For example, normativists differ on whether the requisite norm- or value-dependence of health status is to stem from (a) its dependence on “objective norms”—i.e., norms framed by what *is*, in some important sense, binding on and in that sense “valuable for” all individuals whether or not the view that they are indeed valuable for them because and insofar as they are binding is in fact accepted by any particular individuals or (b) “subjective norms”—i.e., norms framed, in some way or another, by individuals, and binding on them only in so far as that they are self-imposed, different norms correspondingly binding on different individuals even within the same culture.

What I take to be the underlying difference between (a) and (b) is then, at bottom, a distinction between norms appropriately viewed as *applying to* or being *binding on* individuals whatever such individuals feel about the norms, and norms which apply to individuals merely because and insofar as they are *accepted by* those individuals.

Importantly, normativists using the term “objective” to denote states or properties that are held to be universally valuable (and hence valuable whether or not they are accepted as being valuable) are employing a very different conception of objectivity than the conception which a naturalist employs when she insists, “the hard sciences are objective”, where “objective” is understood to denote value, norm, and culture independence. That “objective” is employed in these different ways in the modern debate is unfortunate as it leads to unnecessary confusion. Still worse, I think it is mistakenly employed by some normativists to mean something different than what is often called “intersubjectivity” (about which more will be said below).

To be fair, it is worth stating here that these two different understandings of

“objective” that are in play in the modern debate of health and disease correspond nicely with Philip Pettit’s astute observation that in ordinary usage the word “objective” has a dual connotation. Pettit describes this dual connotation thus:

Under the first connotation, anything that is described as objective belongs with the world that we human beings confront; it is not part of our imagining or invention. Under the second, anything that is described as objective belongs equally to all of us human beings; it is common, intersubjective property.⁸

To reflect the two different usages of “objective” (or perhaps more accurately, two different conceptions of objectivity) at play in the modern debate of health and disease as well as the above more local disagreement within the normativist camp over whether to accept (a) or (b), it will suffice to make what I am calling a *local* (i.e., *within* a single camp) distinction between what it is for values or norms to count as “objective” or “subjective”. To labour the point, not only are there different usages of the term “objective” and its contrasts *within* the normativist camp, the naturalists and the normativists work with different usages of the term and its contrasts. Unless otherwise indicated, I shall use the term “objective” as the naturalist employs the term.

Correspondingly, unless otherwise indicated, when and if I use the term “subjective”, I will use it simply to mean “not-objective”. At times, when it is important to remember this point, I will be using “non-objective” and avoid using “subjective” at all. As we shall come to see, since there are various senses to the predicate “objective” there will correspondingly be several senses of “subjective”, senses corresponding to the denial

⁸ Philip Pettit (2001), “Embracing objectivity in ethics”, in Brian Leiter (Ed.), *Objectivity in law and morals*. New York: Cambridge University Press, pp. 234-286, at p. 246.

of objectivity.

I shall have much more to say on the local differences in both terminological usage and in substance between the various views which can be counted as normativist views in the next chapter. So, for now, the point to press is that there are various important distinctions (and distinctions between distinctions) at play in the modern debate of health and disease. Although normativist theorists are united in their agreement that a viable conception of health must reference values and/or norms, there is fierce disagreement not only over what these requisite values or norms are to be and what rôle they are to be taken as playing, but also over what empirical facts regarding health are relevant and to what extent, if at all, these facts are to be value and/or norm dependent.

3. Following Searle to a point: Two different “senses” of the objective-subjective

3.1 Setting the stage

If we are going to get a good grasp of both the underpinnings and commitments of the naturalist and normativist frameworks, then it is imperative we recognize that the above disagreement over (i) what rôle, if any at all, such values or norms are to be taken as playing and (ii) what empirical facts regarding health are relevant and to what extent, if at all, the facts picked out as relevant are to be counted as norm- or value-dependent is about (at least) two importantly distinct, though related, issues. To see this consider the following health judgement:

(H) Jack’s attention span and hyperactivity warrants the diagnosis of attention-deficit hyperactivity disorder (ADHD).

Suppose that there are two physicians (Smith and Jones) disagreeing over the truth of

(H). That is, Smith and Jones have different and incompatible judgements about Jack's health is it pertains to the diagnosis of attention-deficit hyperactivity disorder (ADHD).

Now it is quite easy to imagine taking issue with this diagnosis for any number of good reasons. For instance, perhaps one is troubled by the figures showing that since 2005 ADHD prescriptions have increased 50% for Canadian males; or that, in Canada, boys are two to three times more likely to be diagnosed with ADHD than girls; or that the 2007 U.S. report on the global use of ADHD drugs singling out Canada for "higher than expected consumption, and suggested our exposure to American advertising and cultural norms play a role".⁹ Let us, however, stipulate that, unbeknownst to one another, Smith follows the fourth edition of *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* while Jones appeals to the *International Classification of Diseases 10th Revision (ICD-10)*.¹⁰ This is not a trivial choice as studies have found that a physician is three to four times more likely to diagnose ADHD when following the *DSM-IV* rather than the *ICD-10*.¹¹

⁹ Carolyn Abraham, "Failing boys: Part 3: Are we medicating a disorder or treating boyhood as a disease?", *Globe and Mail*. Monday, 18, Oct. 2010. <<http://www.theglobeandmail.com/news/national/time-to-lead/failing-boys/part-3-are-we-medicating-a-disorder-or-treating-boyhood-as-a-disease/article1762859/>> Accessed July 4, 2011. My thanks to James Ash for bringing this article to my attention.

¹⁰ The International Classification of Diseases 10th Revision (ICD-10) is published and endorsed by the World Health Organization (WHO) as the international standard to report and categorize diseases, health-related conditions and external causes of disease and injury. The Diagnostic and Statistical Manual of Mental Disorders, edition IV (DSM-IV) is published by the American Psychiatric Association. The DSM-IV is used in the United States and in many other countries in varying degrees.

¹¹ Iaina Singh (2008), "Beyond polemics: The science and ethics of ADHD", *Nature Reviews Neuroscience* 9: 957–964. It is worth pointing out that what the *DSM-IV* calls "ADHD" the *ICD-10* calls "Hyperkinetic Disorder (HKD)". Simply for reasons of clarity, and perhaps familiarity, I will refer to the disorder as *ADHD*. A plausible explanation for the different *ADHD* diagnosis rates is found in Soyoung I. Lee, Russell J., Schachar, Shirley X. Chen, *et al.*, (2008), "Predictive validity of DSM-IV and ICD-10 criteria for ADHD and hyperkinetic disorder", *Journal of Child Psychology and Psychiatry* 49(1): 70-78. Here they note the following three substantive differences between *ICD-10* and *DSM-IV* with regards to *ADHD* diagnosis: (1) ICD-10 requires that the full syndrome be evident in two independent situations (e.g., home and school),

So perhaps Jones disagrees with Smith's diagnosis simply because Jones holds the view that the *ICD-10* is *objectively valid* and, as such, that health judgements *should be grounded* not in the *DSM-IV* (which Smith counts to be *objectively valid*) but in the *ICD-10*. Here we might say that the disagreement is about a second-order health judgement over what criteria or nosology (e.g., *DSM-IV* or *ICD-10*) should determine the truth or falsity of health judgements. The first-order judgement being the judgement that (H): Jack suffers from ADHD.

The argument does not end here, however; for one might disagree over the extent to which, if at all, the criteria determining the truth and falsity of health judgements *are* objectively valid. After all, the possibility exists, we might say, that in the end the choice of criteria is not a fully objective matter but a choice which depends on a choice of values or norms. In other words, one could take issue with the belief which both Smith and Jones have that their criteria are objectively valid and confirmable as the correct criteria by empirical science and question whether in fact both Smith and Jones are merely *assuming* (perhaps uncritically) that *their* preferred criteria are objectively valid (perhaps because this is the criterion in force in *their* society).

To sum up, the crux of the disagreement here is whether there *are* objectively valid criteria to be discovered. The alternative being that there are no such criteria to be

whereas DSM-IV defines pervasiveness as 'impairment in social, academic or occupational functioning from ADHD symptoms' in two or more situations; (2) ICD-10 stipulates there is a single disorder defined by symptoms of inattention, impulsiveness, *and* hyperactivity. Whereas DSM-IV requires evidence of inattention and/or hyperactivity-impulsivity for the diagnosis; and (3) when other disorders are present, ICD-10 encourages the diagnostician to diagnose the other disorder. The DSM-IV, however, permits multiple comorbid diagnoses to co-occur with ADHD with few exceptions.

discovered because the choice of criteria is not in the case of health and disease an objective matter but instead is a choice, which is a choice driven by competing values or competing norms for health. I suggest that no one can deny that one can disagree over whether health judgements are *objectively* or *subjectively* true or false.

In sorting out the issues here, it is important not to confuse the issues just identified with a rather different issue. To see this, imagine that Jones and Smith both agree that (i) ADHD is in some sense a genuine disease; (ii) that there *are* objectively based criteria for ADHD; and (iii) that it is objectively true that Jack's behaviour meets the *DSM-IV* criteria for having ADHD; but *disagree* on (iv) the question of whether the *DSM-IV* criteria for having ADHD is correct and, as such, whether Jack's behaviour is to be properly counted as warranting the ADHD diagnosis.¹² Notice the sense in which (iv) is just (H) recast specifically in terms of *DSM-IV* criteria.

Again, however, the disagreement may run deeper and for distinctly different reason. For here one can be disagreeing over what is required to correctly state that an individual is and, by implication, is not healthy or diseased. For example, one may disagree over which empirical facts regarding health and disease claims in general and attention span/hyperactive behaviour in particular are to be counted sufficient for a proper diagnosis of health or of a particular disease. That is to say, one may disagree about which propositions about the world informs the content of a true health judgement.

Here again we might say this is a different disagreement about a second-order issue

¹² Notice that if the *DSM-IV* was the standard in force against which health judgements are made, then there is a sense in which health judgements would be *objectively* true but not in the sense of being value- or norm-independent. More on this below.

regarding a health judgement where what is at issue is the nature and status of a true health judgement and the place of health and disease in the fabric of the universe.¹³ An important issue here is whether a satisfactory account of health and disease may depend only on objective facts or whether it must also in the end depend on facts which are in some sense value- or norm-dependent and in this sense not objective facts. As we have seen, naturalists and normativists lock horns over this substantive issue. The disagreement over the kinds of facts that a satisfactory account of health and disease must reference to yield true health judgements stands to bring into play a second conception of the contrast between objectivity and non-objectivity.

By linking objectivity and non-objectivity to the truth and falsity of health judgements and to the nature of the facts upon which such health judgements depend seems to nicely track two importantly different ways an account of health and disease may be either value, norm, and/or culture independent or dependent. Although I have not thus far explicitly mentioned Searle's way of contrasting objective-subjective distinctions, I have been guided in my discussion partly by his useful distinction between two "senses" in which such distinctions may be drawn, what he refers to as an *epistemic* and an *ontological* sense.¹⁴ In particular, his suggestion that there is an *epistemic* and an *ontological* sense in which one may contrast the objective-subjective distinction has

¹³ J. L. Mackie (1977), *Ethics: Inventing right and wrong*. Harmondsworth, England: Penguin Books, at pp. 9 & 16-19, for instance, employs the terms "first order" and "second order" to distinguish between what he claims are two completely independent sorts of moral views. He describes a second order view thus: "a view about the status of moral values and the nature of moral valuing, about where and how they fit into the world". I believe this view has some purchase in thinking about the disagreement that occurs in the modern debate of health and disease.

¹⁴ John R. Searle (1995), *The construction of social reality*. New York: The Free Press, especially pp. 7-9.

helped motivate the above analysis that there are (at least) two senses of objective-subjective at play in the modern debate of health and disease.

But though this distinction is a useful place to start from, the discussion to follow will in the end lead me to draw not a bipartite distinction but a tripartite distinction as a way of contrasting the objective and the non-objective. In my view, a tripartite distinction facilitates a stronger grasp of the claims that lie at the very core of the naturalist and normativist frameworks in the modern debate of health and disease.

3.2 Searle's *epistemic/ontological contrast*

According to Searle, in the *epistemic* “sense” of the distinction, “objective” and “subjective” are primarily predicates of *judgements*. In this epistemic sense, what distinguishes “objective” from “subjective” judgements, for Searle, is whether the truth or falsity of the judgement can be settled by citing facts in the world whose status as facts is independent of all attitudes, feelings, and points of view of the judgement makers or hearers *about those facts*—as will emerge, these final three words are important to how Searle draws his distinctions here.

With this in mind, a nice example of an epistemically objective judgement is the judgement “Gold melts at 1948 degrees Fahrenheit”. Clearly the truth of the judgement that there exists a substance with 79 protons and that melts at 1948 degrees Fahrenheit does not depend on anybody’s beliefs, attitudes or feelings¹⁵ *about the existence of the*

¹⁵ This is not to deny that how we use words is a contingent matter that depends upon somebody’s attitudes or feelings. Of course, that it is possible for the words “79 protons” to designate something other than gold does not effect the present point being made. I expand on this in some detail in the beginning of the next section below.

substance or its nature. It is to be noticed that immediately after introducing this epistemic “sense” of the contrast Searle says that “we can speak not only of *objective judgements* but also of *objective facts*” and that “[c]orresponding to objectively true judgements there are objective facts”.¹⁶ So, presumably, an objective fact is what makes an objectively true judgement true. This use of the phrase “objective fact” will be important in the discussion to come.

In the *ontological* “sense” of the distinction, Searle states: “‘objective’ and ‘subjective’ are predicates of entities and types of entities, and they ascribe modes of existence”.¹⁷ In this ontological sense, what distinguishes an “objective” from a “subjective” entity, Searle suggests, is whether its mode of existence is such that it “exists independently of our representations of them”.¹⁸ Thus a piece of gold, for example, is an ontologically objective entity because its mode of existence is independent of any perceiver’s representation of it, whereas pain (to borrow Searle’s own example) is an ontologically subjective entity “because [its] mode of existence depends on being felt by subjects”.¹⁹ In other words, pain is a subjective experience of a particular sort.²⁰

It will be important for our purposes, as Searle acknowledges, that on the above account “we can make epistemically subjective statements about entities that are ontologically objective, and similarly, we can make epistemically objective statements

¹⁶ Searle (1995), *op cit.*, p. 8.

¹⁷ Ibid.

¹⁸ Ibid., p. 9.

¹⁹ Ibid.

²⁰ That this is so helps to explain not only a familiar medical view that pain might not always have any detectable pathological (injury) explanation but also why the exact injury mechanism(s) of medical disorders such as whiplash and whiplash-associated disorders (WAD), and fibromyalgia (FMS) stubbornly resist discovery, but also why they both continue to be controversial diagnoses.

about entities that are ontologically subjective”.²¹ Particularly telling is his example:

... the statement “I now have a pain in my lower back” reports an epistemically objective fact in the sense that it is made true by the existence of an actual fact that is not dependent on any stance, attitudes, or opinions of observers, [though] the phenomenon itself, the actual pain, has a subjective mode of existence.²²

But Searle’s views are a little more complicated than what I have reported so far.

For after discussing the above-quoted pain example he introduces a distinction which, he says, is “more fundamental” than those introduced to that point.²³ He says that “this [distinction] is the distinction between [a] those features of the world that exist independently of us and [b] those that are dependent on us for their existence”, and more specifically between those features of the world that do and those that do not “exist independently of our representations of them”.²⁴ He calls those features of the world which do exist independently of our representations of them “intrinsic” features of the world and those which do not “observer-relative” features of the world, amplifying this description by saying that they are features that exist “*relative to the intentionality of observers, users, etc.*”.²⁵ He illustrates the ideas here as follows:

It is, for example, an intrinsic feature of the object in front of me that it has a certain mass and a certain chemical composition. It is made partly of wood...and also partly of metal...All these features are intrinsic.²⁶

He continues:

²¹ Searle (1995), *op cit.*, p. 8.

²² *Ibid.*, p. 9.

²³ *Ibid.*

²⁴ *Ibid.*, brackets added for clarity.

²⁵ *Ibid.*, italics his. The term “observer-relative” first appears on p. 5 and Searle more formally introduces it on p. 10 thusly: “Observer-relative features are ontologically subjective”.

²⁶ *Ibid.*, p. 9.

But it is also true to say of the very same object that it is a screwdriver. When I describe it as a screwdriver, I am specifying a feature of the object that is observer or user relative. It is a screw-driver only because people use it as (or made it for the purpose of, or regard it as) a screwdriver.²⁷

And a little later:

A good rough-and-ready way of getting at this distinction is to ask yourself, Could the feature exist if there had never been any human beings or other sorts of sentient beings?²⁸

In my view, Searle's way of drawing the distinction here is less than ideal, but the basic idea is useful as will emerge more clearly below when I introduce another distinction Searle draws. As a preview, what I have in mind is the distinction Searle makes between what he calls, following Elizabeth Anscombe, "brute" facts and facts, which he says are a special subclass of "social" facts, that he calls "institutional" facts. Or as Searle puts it, "those features of the world that are matters of brute physics and biology, on the one hand, and those features of the world that are matters of culture and society, on the other".²⁹

3.3 Searle's distinction between "brute" and "institutional" facts

As Searle says, "Brute facts require the institution of language in order that we can *state* the facts, but the brute facts *themselves* exist quite independently of language or of any other institution...Institutional facts, on the other hand, require special human institutions for their very existence".³⁰ Searle then outlines what he takes (and we may take) are the features of these "special human institutions" on which the existence of institutional facts

²⁷ Ibid., pp. 9-10.

²⁸ Ibid., p. 11.

²⁹ Ibid., p. 27; for more see pp. 27-57. See also, G. E. M. Anscombe (1958), "On brute facts", *Analysis* 18(3): 69-72.

³⁰ Ibid., p. 27. Italics his.

depends. He draws the now familiar distinction between two kinds of what he calls “rules”—“regulative” and “constitutive”. A regulative rule R regulates an action the possibility of doing which does not depend on the existence of the rule R. For example, the possibility of not hitting someone’s head does not depend on the existence of a rule forbidding it and the possibility of driving on the right hand side of the road (Searle’s example) does not depend on the existence of a rule requiring it.

In contrast, a constitutive rule is a rule that, as he puts it, “create[s] the very possibility of certain activities”.³¹ Thus, to use his example, I cannot checkmate someone unless there exists a set of rules (the rules of chess) which define what counts as checkmate. As he points out, constitutive rules take the form “‘X counts as Y’ or ‘X counts as Y in context C’”;³² (e.g., moving the queen backwards to an unoccupied square (x) counts as a legal queen move (y) when playing chess (c)).

With this distinction in place, we can summarize what would seem to be Searle’s views on the difference between brute and institutional *facts*, taking a fact to be what makes true a true statement about some feature of the world.

Imagine you and I were witness to the following exchange:

Philosopher dad: “Stop running up and hitting my head, please”

Little Laura: “But all the older kids are hitting your head”

Philosopher dad: “No, the older kids are playing a game!”.

Upon observing this suppose we make two statements: (a) that the older-kids tapped

³¹ Ibid., p. 27.

³² Ibid., p. 28.

philosopher-dad's head and (b) that the older-kids played "duck, duck, goose". Clearly, both (a) and (b) state facts. And moreover, and for obvious reasons given the discussion above, it would be very tempting (and I will yield to the temptation) to say that (a) states a brute fact but that (b) states an institutional fact.

The next question is about what we ought to say is the ontological status of these two kinds of facts. Clearly it is tempting to say that (a) states an *objective fact*. But what are we to say about the fact which (b) states? It is an institutional fact; but can it and ought it to be classed as an objective fact?

At this point we need to bring to bear the distinction I earlier reported Searle as drawing between those features of the world which exist independently of our representations of them and those which don't, the former being called by him "intrinsic" features of the world and the latter "observer-relative". It will be remembered that he amplified this description by saying that the latter features are features that exist "relative to the intentionality of observers, users, etc.".

Putting these ideas together it is *tempting* to say that institutional facts would on Searle's terminology (as presented above) be classed as facts involving "observer relative" features of the world and brute facts would be classed as "intrinsic" features of the world and I think that this is what he would say.

Now we can address the question of whether not only brute facts can exist *objectively* but also whether institutional facts can. It will be remembered that I reported Searle as saying that pains are ontologically subjective because its mode of existence is such that its existence depends upon a perceiver "feeling" the pain; but that pieces gold are ontologically objective because their mode of existence is independent of anybody's

beliefs, attitudes or feelings. On this definition it is clearly at least initially tempting to say that institutional facts are surely *not* ontologically objective since, in a sense, their mode of existence is not independent of our perception of them. But I think that this response is a bit too quick—and I think that this is what Searle would *now* say as well.

In his 2010 book, *Making the social world*, Searle writes:

Institutional facts are typically objective facts, but oddly though, they are only facts by human agreement. Such facts require institutions for their existence. Typically, institutional facts are facts that exist only within human institutions. And what exactly is a human institution?...[It] is a system of constitutive rules, and such a system automatically creates the possibility of institutional facts.³³

Oddly indeed, since, facts that are only facts by human agreement are not independent of anybody's beliefs, attitudes or feelings. Or can they be?

If I am reading Searle correctly here, then he is hitting upon a point that needs to be understood precisely for what is to come and is thus worth belabouring. Returning to the above “duck, duck, goose” scenario, we may note that whether the older-kids played “duck, duck, goose” is a function of two classes of factors (i) what the rules of “duck, duck, goose” say are the conditions (specified in the constitutive rules of “duck, duck, goose”) under which a person counts as *playing* “duck, duck, goose” and (ii) whether what the older-kids did—and what Little-Laura did—under these conditions counts—and does not count—as playing “duck, duck, goose”. Surely we can say that the question of whether a certain constitutive rule is in fact in force in a certain community is a question to which we can give either positive or negative answers and those answers will be correct or

³³ John R. Searle (2010), *Making the social world: the structure of human civilization*. New York: Oxford University Press, at p.10.

not and their correctness or incorrectness *could* be independent of anyone's beliefs *about the question of whether they are in force or anyone's attitudes to them being in force*. That is, it could be "objective"—yes?

This question suggests the need for some refinement in the definition of what it is for a fact to be objective. These refinements will make it possible to distinguish two rather different versions of normativism, one that defines normativism as an ontological theory which affirms that states of health and disease are "norm-dependent" states and the other that affirms that they are "value-dependent" states.

3.4 What we can extract from Searle's objective-subjective distinction

For reasons that will become clear below, I think Searle's way of drawing the objective-subjective distinction belies an important issue that naturalists and normativists disagree over and, given our goals, is not the ideal framework to employ. Nevertheless, I do believe that Searle's dyadic objective-subjective contrast, in general, and his notion of "institutional facts", in particular, points the way towards grasping a clearer understanding of the naturalist/normativist debate in the literature.

What I want to suggest is that Searle's epistemic and ontological senses of the objective-subjective distinction lay the foundation for an important way to characterize the core differences between the naturalists and the normativists in the modern debate concerning health and disease.

Let us begin with Searle's ontological sense, or, as I would prefer to put it, Searle's ontological conception of the distinction. Suppose an account of health were to suggest that whether or not a certain state is a health state is a function of ontologically subjective

facts. What we are being asked to consider here is an account of health that makes health and disease a function of the sort of facts that would not exist without the presence of human beings and more specifically without the beliefs, attitudes and/or feelings of human beings *about these facts*. These are therefore facts that *eo ipso* depend on human beliefs, attitudes, and/or feelings about them. All this is to say that an account of health that makes it a function of ontologically subjective facts is an account of health that is value- or norm-dependent. It is reasonable to conclude that a satisfactory naturalist account of health may not involve essential reference—explicitly or implicitly, wittingly or unwittingly—to ontologically subjective facts.

We should also recognize that no naturalist account of health may allow the account of health or disease to make essential reference, either explicitly or implicitly, to what Searle calls an epistemically subjective judgement about the state. To do so, would be tantamount to making the truth (or falsity) of a health judgement a function of the possible projection³⁴ of personal and indeed possibly varying attitudes, tastes, and values about health status or societal/cultural norms governing judgements about health status. This is evident when we consider that Searle's conception of *epistemically subjective* ultimately stipulates the commitment to "subjective" values or norms. Properly understood, Searle's conception of epistemic subjectivity illuminates another way for an account of health and disease to be value- or norm-dependent.

This is an important point worth expanding upon, particularly because not only is this sort of subjectivity ubiquitous, it is often underappreciated. Suppose Lindsay, for

³⁴ See Mackie (1977), *op cit.*, Chapter 1, Section 10 for the notion of projection here.

whatever reason, has an aesthetic aversion to tarnished objects. And because of this aversion, Lindsay believes that gold is a more beautiful metal than silver. Now that silver tarnishes but gold does not is the case quite independently of Lindsay's (or anyone's) beliefs, attitudes, or values. That silver tarnishes but gold does not is thus an objective fact.

Nevertheless, whether it is true that gold is a more beautiful metal than silver because it does not tarnish is not a simple matter of objective facts. The truth or falsity of the judgement also depends on either (a) individual or personal attitudes or feelings of the person making or hearing the judgement. Or it depends on a rather different account of judgements of beauty, that is, it depends on (b) the norms or institutional facts which are in force in the community in which the judgement of beauty is being made—like whether one's society values or views the quality of remaining untarnished to be a significant criterion for being a beautiful metal. Notice if the truth or falsity of the judgement depends upon institutional facts by (b), then the matter could be settled independently or perhaps even in spite of Lindsay's particular attitudes or feelings. So the truth or falsity of a judgement can be independent of *particular* values or norms, yet be dependent on values and/or norms.

The important point to stress is that both of Searle's ontological and epistemic conceptions of "subjectivity" make reference to a component which is "subjective", i.e., a component which importantly *depends* upon at least one person's particular attitudes, values, points of view, feelings, etc., about that component. And because of this, no satisfactory naturalist account of health and disease may be a function of what Searle describes as ontological or epistemic subjectivity. It is in this way that Searle points the

way to a clearer understanding of two very different ways of underpinning a normativist account of health. Thus, what we should extract from Searle is the need to identify and distinguish between (at least) two distinct senses—a so-called ontological and epistemological—of subjectivity which a naturalist *must* avoid, for each will render an account of health and disease to be value or norm-dependent. With this in mind, we have an important way to characterize the core ideas of the two major positions—naturalism and normativism—in the modern debate concerning health and disease.

We can sum up the above by saying that Searle's dyadic account of the objective-subjective distinction provides the impetus for a clearer understanding of the variety of objectivity and subjectivity at play in the modern debate of health and disease. Sometimes the subjective nature of these health judgements are made explicitly and other times implicitly. And because of this, to get a good grip on both the underpinnings and commitments of the naturalist and normativist frameworks requires a reasonable understanding of how to draw the objective-subjective contrast.

At a minimum, Searle's particular way of describing the objective-subjective distinction, however, invites further investigation with a much more fine-grained analysis. After all, with a little careful reflection one can easily envisage a number of importantly different ways a normativist account of health may *depend* on (in the sense of being a function of) subjective attitudes, values, points of view, feelings, norms, et cetera.

One way to see this is by reflecting on a rather different,³⁵ though I submit a fairly

³⁵ Notably, Searle (1995) *op cit.*, at p. 7 maintains that the distinction between the objective and the subjective is a matter of degree. Thus I believe one way the following three-part contrast is importantly different from Searle's is that it makes the distinction between the objective and the subjective a difference

common³⁶ way of drawing a three-part (ontological, epistemological, and semantic) contrast between objective and subjective.

4. Re-orienting the objective-subjective distinction

Before we proceed to draw our tripartite contrast of the objective-subjective distinction, there are two important provisos we must make clear. Firstly, the objective-subjective distinction is a philosophical creation.³⁷ That being said, my purpose is to draw an ontological, epistemological and semantic contrast in such a way as to bring to light as clearly as possible the commitments that arise from the theoretical objectives of naturalism and normativism within the modern debate of health and disease. To that end, my concern is not to delineate all, or perhaps even many, of the ways in which one might find an objective-subjective contrast drawn in current philosophical literature.

Secondly, and relatedly, a primary aim in drawing *this* contrast is to bring to light the ways in which normativist—and wrongly categorized naturalist—accounts of health and disease depend on (what may be called “subjective”) values and norms. As I stated at the outset of this chapter, my hope is to provide a useful framework from which to better recognize the way normativist “subjective” underpinnings are often employed, sometimes explicitly and other times without discussion, to advance health claims.

in kind, not merely degree.

³⁶ For example, both Joseph Raz and Brian Leiter distinguish between metaphysical, epistemic and semantic objectivity. See, Joseph Raz (2002), *Engaging reason: On the theory of value and action*. Oxford: Oxford University Press, and Brian Leiter (2007), *Naturalizing jurisprudence: Essays on American legal realism and naturalism in legal philosophy*. Oxford: Oxford University Press, especially chapter 9.

³⁷ This view, for instance, is found in Raz (2002), *op cit.*, p. 119 and Andrei Marmor (2001), *Positive law and objective values*. Oxford: Oxford University Press, at p. 112.

4.1 An ontological contrast (O^*)

With the above in mind, we can draw an ontological contrast³⁸ in spelling out what it is by virtue of which something exists. More formally, we might say:

- (i): some object x **exists** ontologically objectively *iff* that x exists is not a function of any person A 's beliefs *about* x 's existence or attitudes *to* x 's existence. Something x exists subjectively if it does not exist objectively.

This contrast (O^*i) is what I take Searle to have in mind with his ontological conception of the objective-subjective distinction. We've seen that Searle distinguishes between constructed or "institutional" properties and facts and Anscombe's term, "brute" properties and facts; however, it will be helpful to expand upon this notion and explicitly draw an ontological contrast in what it is by virtue of which *something* has a certain kind of existence:

- (ii): some property P is an ontologically objective **property** of something x *iff* the fact that x has P is not a function of any person A 's beliefs *about* x 's having P or attitudes *to* x 's having P . Something P is a subjective property of x if it is not an objective property of x .

We can also draw a third ontological contrast in what it is by virtue of which something is a fact:

- (iii): some fact F is an ontologically objective **fact** *iff* the fact that F exists is not a function of any person A 's beliefs *about* whether F is a fact or attitudes *to* F 's being a fact. Some fact F is a subjective fact if it is not an objective fact.

Finally, I think there is a fourth important contrast to be drawn in virtue of which something is a state, which might be put as follows:

- (iv): some state S is an ontologically objective **state** of something x *iff* that x

³⁸ I owe these ways of drawing the distinction to John A. Baker.

is in that state is not a function of any person A's beliefs *about* whether x is in that state or attitudes *to* x 's being in that state. S is an ontologically subjective state of x if it is not an ontologically objective state of x .

With (O*iv) in mind, it is worth mentioning what is surely a very different kind of contrast between ontologically objective and ontologically subjective states, a contrast which in this literature has been confused with (O*iv):

(M): some state is an ontologically objective *state* of some organism (human or otherwise) x *iff* that state is **not** a *mental* state of x . Some state is a subjective state of x if it is a mental state of x .

For our purposes, it will suffice to refer, very roughly, to this objective-subjective contrast as the non-mentality/mentality contrast—contrast (M) for short. What is to be stressed is that (M) is very different from (O*iv). Now having drawn attention to (M) we may for the most part set it on one side.

To further illustrate these four ontological contrasts—(i)-(iv)—we might say, for example, Jack's being healthy exists objectively by (O*i), or Jack's being healthy is objectively a property of Jack by (O*ii), or that Jack is healthy is an objective fact by (O*iii), or that Jack's health is an objective state of Jack by (O*iv) because whether or not Jack is healthy is entirely a matter (a) of criteria (i.e., factors involving facts, properties, states of Jack's) whose (b) satisfaction or non-satisfaction are both independent of someone's beliefs about such factors and/or attitudes to his being healthy.

To the extent that one counts objects, properties, facts and/or states existing as a function of human attitudes or beliefs about them to be requisite for health, each of (O*i-iv) shows a different way in which a normativist account of health may, explicitly or covertly, *depend* on subjective attitudes, values, points of view, feelings, et cetera.

4.2 Epistemological contrast (E*)

A rather different way in which a normativist account of health might depend on particular attitudes, values, points of view, feelings, etc., would be an account which turns on the ways in which we may come to believe or know, for instance, that P: a particular person and/or state is healthy. One way to acknowledge this is to draw an epistemic objective-subjective contrast between the ways we come to believe or to know that P, thus:

(E*): someone A's belief or knowledge claim that P is epistemically objective *iff* A's belief or knowledge claim that P is **verified** or **falsified**³⁹ independently of (i.e., is a process which involves no essential reference to) anyone's beliefs, attitudes, or feelings *about P*. Otherwise, the belief or claim is epistemically subjective.

By way of illustration, we might say Jones' belief or knowledge claim that Jack is healthy is epistemically objective by (E*) because Jones' belief or knowledge claim qualifies as true or false with respect to it being verifiable independently of Jack's or anyone else's belief, attitudes and/or feelings that he is healthy and is, *pro tanto*, a claim that any reasonable person must accept. Notice that epistemic objectivity allows the possibility for everyone and even our society as a whole to be wrong about health judgements (more on this later).

Now (E*) is clearly related to Searle's epistemological conception of the objective-subjective distinction but I think subtly different. Consider that, epistemically speaking, Searle distinguishes "objective" from "subjective" judgements by whether the truth or falsity of the judgement can be settled by facts in the world that are independent of the

³⁹ Here I am using these terms widely to include not only proving true or false but also proving probably true or probably false.

attitudes, feelings, and points of view of the judgement makers or hearers about those facts. But is it clear here that Searle is not intending to enter the domain of semantics, the theory of meaning and truth, or becoming concerned with an explanation between meaning and our statements about the world? I'm not so sure. The difficulty is it is not at all clear that what is also at issue is whether the judgement, "Jack is healthy" designates (or denotes) certain entities that include "subjective" entities as defined in (O*i-iv).

With this in mind, a reasonable person might wonder whether Searle is packing into his epistemological sense of the objective-subjective distinction another sense in which one might contrast the objective-subjective distinction—a semantic sense.

4.3 *Semantic contrast (S*)*

At any rate, the suspicion is strong that people take naturalists and normativists in the modern debate to be locking horns over the semantic questions: what does it mean to say that an individual is healthy and by virtue of what are such statements true? George Khushf, for example, writes that, "many bioethicists have regarded the debates surrounding the nature of health and disease (and related concepts such as illness) as the key to such diverse issues as the meaning of basic health care, the appropriate scope of medicine, and the focus of medical care".⁴⁰ Elsewhere I have argued that the means for realizing the goals of healthy people and healthy communities are inevitably a function of what we understand "health(y)" to be.⁴¹

⁴⁰ George Khushf (1997), "Why bioethics needs the philosophy of medicine: Some implications of reflection on concepts of health and disease", *Theoretical Medicine* 18: 145-163, at p. 145.

⁴¹ J. David Guerrero (2010a), "The essential value(s) of health: Implications for Canadian population health research and policy", In T. McIntosh, B. Jeffery & N. Muhajarine (Eds.), *Redistributing health: New*

Insofar as this is correct, we would be wise to explicitly articulate a possible *semantic* account of the objective-subjective contrast, an account in terms of the kinds of things that make such statements true or false. It seems to me that the following will serve,

(S*): someone A's statement that P is semantically objective *iff* the truth conditions of the statement that P make the truth of P independent of anyone's beliefs, feelings, or attitudes about the truth of P. A's statement that P is semantically subjective (non-objective) if the truth conditions of the statement that P make the truth of P dependent on someone's beliefs *about* the truth of P and/or feelings, or attitudes to the truth of P.

Continuing with the above example, we might say that Jones' statement, "Jack is healthy" is objectively true by (S*) because what makes it true is something about the world (e.g., an object, fact, or property) such that the truth conditions of the statement are not a function of anyone's belief *about it* or attitude *to its presence*, i.e., if what makes health judgements true and false is not a function of anyone's beliefs, feelings, or attitudes towards Jack and his health. In other words, here semantic objectivity implies that that the statement "Jack is healthy" has assertoric force.

The crucial point is this: Given the above explication, a *semantic* conception of a objectivity/subjectivity contrast of the kind articulated in (S*) is clearly embedded within what Searle is calling the "epistemic" sense of the objective-subjective distinction. However, I believe that Searle's dyadic distinction between objective-subjective belies the important fact that naturalists and normativists disagree over the means by which we ought to choose the criterion terms with which we ought to reason to and verify the truth or

falsity of health judgements. Indeed, this is the sort of disagreement that (E*) sets out to explicitly capture. Consider again the above disagreement (in Section 3) over whether Jack's attention spans and hyperactivity warrants an ADHD diagnosis. There we saw that one might be disagreeing over whether Jack's behaviour is to count as ADHD simply because one holds the view that health judgements should be grounded not in the *DSM-IV* but in the *ICD-10*.⁴²

Now, it seems to me that we can plausibly view this as being a legitimate disagreement over two (set aside (O*) for the moment) importantly related but distinct issues. On one hand, the disagreement over whether we should ground health judgements in the *DSM-IV* or the *ICD-10* might be essentially an argument over the means by which we can reason to and verify the truth or falsity of health judgements. Suppose, for instance, Smith grounds health judgements in the *DSM-IV* just because his grandfather helped author it; whereas, Jones grounds health judgements in the *ICD-10* for reasons independent of her particular attitudes or feelings towards the authors involved. Here, it is easy to see the issue regarding the ways in which we ought to come to believe or to know that a particular health judgement qualifies as true or false providing the impetus for legitimate disagreement. This is precisely the sort of legitimate disagreement that (E*) sets out to explicitly capture.⁴³ Because of the obvious reference to belief and knowledge,

⁴² For the sake of brevity, it will be helpful to simply stipulate that both sides agree that Jack's behaviour warrants the *ADHD* diagnosis according to the *DSM-IV*, but not according to the *ICD-10* (more on this below in Section 5). Nothing I can see affects my argument, one way or the other, by such a concession.

⁴³ This way of thinking about the *epistemic* sense of the objective-subjective contrast falls in line with Brian Leiter's suggestion to view epistemic objectivity to occur when either (1) the cognitive processes at issue *reliably* arrive at accurate representations, or (2) the cognitive processes are free of factors that we know to produce inaccurate representations. See Brian Leiter (2001), "Introduction", in Brian Leiter (Ed.), *Objectivity in law and morals*. New York: Cambridge University Press, pp. 1-11, at p. 1.

I am strongly inclined, as is commonplace in the philosophical literature, to refer to *this* objective-subjective contrast as an *epistemological* contrast.

On the other hand, however, and this is crucial, it just might be the case that the disagreement over whether we should ground health judgements in the *DSM-IV* or the *ICD-10* is essentially an argument over exactly what criteria or nosology tracks *true* health judgements. Perhaps one disagrees about what it means to be healthy and as such over what facts regarding health in general and undesirable behaviour in particular one does and does not count to be relevant for *true* health judgements. The extensive and passionate disagreement over just what makes health judgements true and false is what (S*) means to explicitly capture.

It is worth noting here that my approach to link semantics with a concern for meaning and what makes judgements true and false is familiar under the aegis of current philosophical literature.⁴⁴ This is the sort of consideration which leads me to suggest that what Searle calls the “epistemic” sense of the objective-subjective distinction is really better understood as a *semantic* contrast. Recall, after all, Searle does say that what distinguishes, epistemically speaking, “objective” from “subjective” judgements is whether the truth or falsity of the judgement can be settled by facts in the world that are independent of certain attitudes, feelings, and points of view of the judgement makers or

⁴⁴ Brian Leiter, for example, notes: “Many philosophers working in the Anglo-American traditions also worry about *semantic* objectivity, that is, about whether or not the propositions in some realm of discourse (physics, psychology, ethics, law, etc.) can be evaluated in terms of their truth or falsity.” See Brian Leiter (2007), *op cit.*, at p. 257. A strikingly similar view of semantics is found in Joseph Raz (2002), *op cit.*, especially chapter 6; and Mark Crimmins (1998), “Semantics”, In E. Craig (Ed.), *Routledge Encyclopedia of Philosophy*. London: Routledge. <<http://www.rep.routledge.com/article/U036>>. Accessed July 4, 2011.

hearers about those facts. (S*) clearly captures Searle's concern with what makes health judgements true and false.

Most importantly, notice that (E*) is different from Searle's sense of the epistemic contrast. Stephen Darwall succinctly captures the difference between the epistemic and semantic when he says, "[t]he claim that we ought to believe what is true is not the tautology that we ought to believe what we ought to believe".⁴⁵ Because epistemic (E*) and semantic (S*) senses of the objective and subjective differ in important respects, it will prove useful for our framework to have the wherewithal to explicitly disentangle them.⁴⁶

To conclude, let us bring (O*i-iv) back into the fold by way of some brief remarks. Importantly connected to (S*) is the following closely related but distinct substantive issue: whether a satisfactory account of health and disease may depend only on objective facts or whether it must also depend on subjective facts. For one may disagree with the extent to which, if at all, the existence of the empirical facts themselves counted to be requisite for a satisfactory account of health and disease depend on values or norms. The pressure exerted by the naturalist-normativist disagreement over the nature of the facts (here broadly construed) on which a satisfactory account of health and disease depends upon is precisely the disagreement (O*) means to capture and shed light upon.

The discussion in this section has centered on formulating and advancing the view

⁴⁵ Stephen Darwall (2001), "Normativity", In E. Craig (Ed.), *Routledge Encyclopedia of Philosophy*. London: Routledge. <<http://www.rep.routledge.com/article/L135SECT6>>. Accessed July 4, 2011.

⁴⁶ With this I am in agreement with David O. Brink's contention that "An adequate semantic theory must (1) distinguish between the meaning of reference of terms and the beliefs of speakers about the extension of their terms, [...]" David O. Brink (2001), "Legal interpretation, objectivity, and morality", in Brian Leiter (Ed.), *Objectivity in law and morals*. New York: Cambridge University Press, 12-65, at p. 46.

that there is an important and useful tripartite (i.e., ontological, epistemological, and semantic) distinction that can be drawn between objective and subjective which persists within the modern debate of health and disease. The next chapter will be primarily concerned to further motivate this view. At this point, I hope the above remarks have succeeded in throwing new light on the work of Searle and in demonstrating that (O*), (E*), and (S*) are importantly different. At any rate, henceforth any discussion of an ontological, epistemic, or semantic sense of the objective-subjective contrast will be in the sense defined by (O*), (E*) and (S*), unless explicitly stated otherwise.

4.4 Summing up

The ontological, epistemological and semantic contrasts of the objective-subjective distinction are importantly interdependent. For instance, the ontological objectivity as defined in (O*) is logically required in order for our health judgements to qualify as, semantically speaking, objectively true and false. If our health judgements depend on ontologically subjective facts then the existence of at least some of the conditions by which the truth or falsity of the statement that, for example, “Jack is healthy” is determined will be dependent on someone’s beliefs, feelings, or attitudes.⁴⁷ This relation of entailment between (S*) and (O*) becomes significant insofar as we want to insist that legitimate disagreement over the truth and falsity of health judgements has a genuine place

⁴⁷ There are some tricky issues here and, in my view, one needs to be careful not to confuse being in an ontologically subjective state (on criterion O*) and being in a mental state. See my contrast between O* and M*. It is tempting to say that it can be an ontologically objective fact that someone, say X, is in some ontologically mental state. Relatedly, it is tempting to say (though I recognize there are problems here) that it only becomes an ontologically subjective state if the fact that X is in that mental state is a function of whether someone (that is not X) believes or likes it that they are in this state of facts.

in modern medical science. For legitimate disagreement here demands that our true health judgements must do more than merely report facts made possible by personal feelings or attitudes.

And, what is more, if health is an objective state by (O*), then it carries implications for (E*) and (S*). For it would surely be dangerous with respect to people's health to oppose both verifying our judgements concerning health (E*) with respect to the objective facts or properties that make health an objective state and including such facts or properties in our discourse about what it means to be healthy and what makes health judgements true and false (S*). Note that it is difficult to understate the importance of these implications when it comes to health and disease. To illustrate with a simple example, imagine if we collectively opposed grounding our health judgements and what it means to be healthy in (what I take to be uncontroversially) the ontologically objective fact that Hepatitis C is an infectious disease and, what is more, actively encouraged using blood infected with Hepatitis C for transfusions.

What I want to suggest is that (O*), (E*) and (S*) are linked to the way in which naturalists count health (and disease) as objective value-free states and normativists count them as subjective value- or norm dependent-states that are in some important sense psychological or social constructs. With this in mind, however, a qualification is in order. If (surprisingly) the hard sciences were to discover an objective value *v* or a norm *n*, such that:

v is of value, but whether it is of value is not a function of whether anyone *believes* that it is of value or any *likes* that it is of value, and

n is a norm but whether *n* is binding on individual A is not a function of whether anyone believes that it is binding on A or likes that it is binding on

A,

then I see no good reason to think that the naturalist cannot employ such values or norms so long as the objective value ν or the norm n were to be established in such a way that does not undermine its claim for objectivity as defined in (O*).

To be sure, such a value or norm would be of the most peculiar or “queer” sort.⁴⁸

However, my point here is not whether objective values or norms of this sort exist but that if one did, then the naturalist, strictly speaking, may appeal to values or norms. This being said, even if such values or norms were to exist, I am skeptical that one could convincingly demonstrate their existence simply by employing the empirical methods of the hard sciences or in some other manner that would not genuinely undermine the claim for objectivity. It may be worth noting that in this skepticism I am for obvious reasons in very good company—Moore, Hare, Mackie, all expressivists and anti-realists and maybe even some quasi-realists.

⁴⁸ I think it is clear that the postulation of an objective value and/or norm of this sort would, initially at least, run against Mackie’s persuasive argument from queerness. That is, an objective value that would be binding and, as such, serve as reasons for action independent of anyone’s beliefs, feelings, attitudes, desires, and so on would be very queer entities indeed. See Mackie (1977), *op cit.*, Chapter 1.

Chapter Two

The Distinction between Naturalism and Normativism

1. Introduction

We are now in a position to move towards putting forth a clearer articulation of the fundamental difference between naturalism and normativism than what is usually found in the literature. To that end, let us employ our tripartite framework to try to get a better grasp of what naturalists concerning health and disease are really trying to do.

It seems to me that naturalists are trying to show that one can advance a satisfactory account of health and disease, which makes health and disease a function solely of objective ‘facts of the matter’ in the sense defined by (O*), which, in turn, claims that the truth or falsity of health judgements (e.g., “that a certain organ or organism x is diseased or healthy”) is independent of anyone’s beliefs, feelings, or attitudes about the truth and falsity of P by (S*). The naturalist then takes this ontological and semantic objectivity to ground the claim for epistemic objectivity as defined by (E*) since, she claims, such health judgements are at least in principle verifiable and falsifiable by value-independent science.

To illustrate, consider the statement “Socrates is healthy”. In my view, naturalists will want to say that Socrates’ being healthy is function of at least *two* parameters: (i) that the organism is healthy in virtue of possessing some properties p_1, \dots, p_n where each relevant property p_i is a property whose existence is independent of our or anyone’s attitudes or feelings about it or beliefs about it (ontological objectivity); and (ii) that the conditions by which the truth or falsity of the claim “the set of properties p_1, \dots, p_n is requisite for health” is determined exclusively by objective facts, independent of values

and/or norms (semantic objectivity). The naturalist reasons (from the objectivity as specified in (i)) that the presence of each p_i is (or, perhaps, at least in principle is) establishable by using only the empirical methods of the hard sciences; and (from the objectivity as specified in (ii)) that the truth conditions for the claim “the set of properties p_1, \dots, p_n is requisite for health” is (or, perhaps, at least in principle is) determinable using only the empirical methods of the hard sciences. This is then taken to provide the requisite underpinnings for the naturalist to assert the epistemic objectivity of health judgements. So, to the extent that the methods of the hard sciences are value- and culture-independent, this provides a check line of the points at which the naturalist will need to track the objectivity of (O*), (E*), and (S*).

This characterization of naturalism lends itself to understanding the crux of the modern debate between naturalists and normativists as disagreement about whether the claim that an individual is healthy or diseased is just as amenable to value-free scientific inquiry as determining the truth of the claim that a particular piece of metal is gold or not. At bottom the issue is whether we simply discover the set of properties requisite for health, as we do with the properties of gold. Or whether, in some important sense, we “create” states of health, perhaps as we create legal tender or licensed physicians. This disagreement over the kinds of things that make health judgements true or false and about how such statements can be and are to be justified is, in a nutshell, the core issue facing the task of explicating the ways in which (O*), (E*), and (S*) are to be viewed as playing a central rôle in the structuring of the argument between the naturalists and the normativists—an argument which is essentially over the extent to which, if at all, a satisfactory account of health (and disease) must be value- or norm-dependent.

The naturalist holds that a satisfactory account of health and disease successfully tracks the requisite objectivity in the sense set out in (O*), (E*), and (S*). Normativists reject some or all of this view. Correspondingly, normativists insist that a satisfactory account of health and disease will be inextricably tied either (a) to claims about the existence of norms of various kinds and/or (b) to expressions of human values, attitudes, or feelings. More specifically, my claim about how to characterize the normativist position is that the normativist insists that a satisfactory account of health and disease will, in some important way, be subjective (i.e., non-objective) on the criteria spelled out in (O*) and/or (S*).

To be sure, there exists extensive disagreement among normativists over exactly what the right rôle values and/or norms are to play, but as we will see (in Section 3) we can get clear about some broad distinctions that lie within the framework of normativism by employing the above tripartite objective-subjective contrast further. First, however, I want to motivate the view that unlike ontological and semantic subjectivity, epistemic subjectivity does not *by itself* sound the death-knell for a naturalist concerning health and disease.

2. The question of the *semantic* and ontological objectivity of health judgements is the basic issue between the naturalist and normativist

This section aims to demonstrate that we can obtain a still clearer articulation of the fundamental difference between naturalism and normativism when we set the epistemic sense of objectivity as defined by (E*) temporarily on one side, and concentrate on the ontological (O*) and semantic (S*) accounts of the objective-subjective contrast. I think

we can do this because, properly considered, acceptance of a thesis affirming the *epistemic* objectivity of health judgements is neither a sufficient nor a necessary condition for a naturalist account of health and disease. To motivate the first conjunct of this claim, it will help to draw upon an example.

Consider again our two physicians, Smith and Jones, with different and disagreeing health judgements over whether a particular child merits the diagnosis of ADHD. For clarity of exposition let us make some simplifying assumptions. Let the *DSM-IV* stipulate that, if conditions D_1, \dots, D_n are satisfied in the case of patient X then X counts as having ADHD and not otherwise, and that the *ICD-10* stipulates that if conditions I_1, \dots, I_n are satisfied in the case of patient X then X counts as having ADHD and again not otherwise. Let us further stipulate that the two lists of conditions D_1, \dots, D_n and I_1, \dots, I_n do in fact contain some common members and that some of D_1, \dots, D_n and I_1, \dots, I_n make reference to factors like length of attention span, level of activity, and such like. That is to say, they make reference to what are clearly mental states.

Notice that some of conditions D_1, \dots, D_n and I_1, \dots, I_n may and indeed do make reference to what can plausibly be called *norms*—thus, assuming that each set of conditions includes reference (for example) to what is counted as *deficiency* in self-regulative capacities, an *excessive* tendency to postpone the beginning of tasks, an attention span that is *undesirably* short, we can say that each of the *DSM-IV* and *ICD-10* in effect sets standards of *normality* and *desirability* and each of the *DSM-IV* and *ICD-10* can appropriately be said *itself* to function as a norm.

Now let us stipulate that each physician examines a single patient X and further stipulate that each physician, Smith and Jones, makes no “mistakes” when checking to see

if X satisfies the conditions specified in the norm which he counts as the one which it is appropriate or required that he work with.

We can now say that Smith correctly judges (i.e., judges truly) that X suffers from ADHD [call this S1] and our statement can be counted true, *so long as we interpret it as making the compound assertion* (i) *that the DSM-IV affirms as conditions for suffering ADHD that the patient satisfy conditions D_1, \dots, D_n [S2]* and (ii) *that patient X satisfies conditions D_1, \dots, D_n [S3]*. The same holds for Jones *mutatis mutandis*. Now *that* the DSM-IV sets conditions D_1, \dots, D_n as the defining conditions for ADHD (i.e., statement S2) is a sociological claim that is true and, I suggest, on the criteria for ontological and semantic objectivity (O^*) and (S^*) ontologically and semantically objectively true. Similarly that patient X satisfied conditions D_1, \dots, D_n [S3] can be similarly ontologically and semantically objectively true.

The important lesson here is that even though some of conditions D_1, \dots, D_n and I_1, \dots, I_n concern mental states, dispositions and capacities, and moreover are expressions of what might naturally be called value judgements about what are to count as *desirable* and *undesirable* capacities and traits and as *excessive* or *acceptable* modes of behaviour, nevertheless the judgements S1, S2, and S3 are themselves epistemologically objective judgements on the definition (E^*).

Now contrast the following different statement:

[S4] The condition ADHD is present in patient X iff conditions D_1, \dots, D_n are satisfied in X .

Note that S2 and S3 are vitally different from S4. We can only decide if S4 is ontologically objective by (O^*) and semantically objective by (S^*) if we know that each of

D_1, \dots, D_n specify conditions that are themselves ontologically and semantically objective and we have not at any point stipulated or assumed that in the above. Thus the fact that judgements S1, S2, and S3 are epistemologically objective by (E*) is not a sufficient condition for the judgement S4 itself to be ontologically or semantically objective.

In summary, to the extent that one is willing to recognize these studies, it seems reasonable to contend that both physicians may be making a true health judgement that is, epistemically speaking, an objectively verifiable judgement which involves (a) implicit reference to institutional facts in the sense specified in my Chapter 1 (e.g., the *DSM-IV*, *ICD-10*) and/or (b) other ontologically subjective entities (e.g., *undesirable* attention spans, *hyperactivity*) which are value-dependent. Note, however, that here the judgement of health or lack thereof is, at least in part, a function of ontologically subjective—which is to say value- or norm-dependent—facts.

Many, and indeed probably most, people have no problem taking these sorts of statements (i.e., a statement that is an epistemically objective statement about ontologically subjective facts) to be objective.¹ The naturalist, of course, cannot allow that health judgements depend on value and/or norm-dependent facts, and hence, the naturalist cannot be content with merely epistemic objectivity, she must also insist upon ontological objectivity. Epistemic objectivity therefore is not a sufficient condition for a value-independent naturalist account of health and disease.

¹ Consider, for instance, the implicit acceptance of ontological subjectivity that a meaningful notion of epistemic objectivity in law demands.

And for similar reasons, *epistemic* subjectivity (whether value- or norm-dependent) is not a sufficient condition of a normativist account of health and disease. For either semantic or ontological subjectivity will suffice to make an epistemically objective health judgement value and/or norm dependent. If the above reasoning is sound, then what remains is to philosophically motivate the second conjunct of the above claim: that epistemic objectivity is not a necessary condition for a naturalist account of health and disease.

To that end, I want to press the view that epistemic objectivity (and subjectivity) is context- and society- and perhaps even person-relative. To appreciate this, it is important to recognize that *epistemic* objectivity does not preclude the possibility of being “wrong” about health judgements. It is always possible, for instance, that the *DSM-IV* and/or *ICD-10* are in some sense “mistaken” in its characterization of ADHD—see S4 above.

Equally clearly, is that *epistemic* subjectivity does not preclude the possibly of being “right” about health judgements. An individual may believe things for all sorts of irrelevant reasons, many of which will render a belief or knowledge claim epistemically subjective in one way or another. Yet, what these beliefs are *about* may be such that they can, when properly considered, be counted as qualifying as objectively true or false, that is, true or false quite independently of anyone’s particular beliefs, attitudes and/or feelings. They would, in other words, be semantically objectively true of ontologically objective states or affairs

To illustrate, suppose Mary is adamant that her only child does not have Tay-Sachs disease (TSD) because she simply cannot bear to face the possibility—a case of

classic self deception.² Given Mary's reasoning surely we want to say that Mary's health judgement about her child (i.e., "my child does not have TSD") is *epistemically* subjective by (E*). But the epistemic subjectivity of Mary's judgement in no way entails that a physician's health judgement that "Mary's child does not have TSD" can be properly considered objective by (E*), (S*), and (O*). Indeed, I believe a physician's health judgement could be rightly considered objective by (E*), (S*), and (O*), *if* he has used the standard TSD test for identifying the presence of the enzyme hexosaminidase A (Hex-A), for example. At any rate, ontologically objective objects, facts, properties, et cetera remain ontologically objective regardless of whether we arrive at our beliefs or knowledge claims on grounds that are epistemically subjective.

To put it bluntly, how or what we think about an ontologically objective object simply does not change the object in itself (to use Searle's terminology, it does not change the intrinsic nature of the world—see my discussion in Chapter 1). And it follows from this that epistemic subjectivity need not render the truth conditions of a semantically objective statement about ontologically objective objects to be, semantically speaking, subjective. Nor should we want to insist otherwise given that it is not at all uncommon to find people's beliefs and knowledge claims grounded in all sorts of irrelevant and irrational reasons.

We need to be careful here because, as I believe the scenario with Mary and her

² Tay-Sachs disease (TSD) is a fatal, recessive genetic disorder in children that causes progressive destruction of the central nervous system. Infants with TSD appear to develop normally but usually die by age 4. National Institute of Neurological Disorders and Stroke website, "NINDS Tay-Sachs Disease Information Page". <<http://www.ninds.nih.gov/disorders/taysachs/taysachs.htm>> Accessed July 4, 2011.

doctor shows, epistemic objectivity and subjectivity is context and society or even person relative. Thus, even if a particular health judgement by some particular person or group of people is epistemically subjective it does not entail that the same health judgement grounded in *different* evidence cannot be epistemically objective and vice versa. Moreover, that Mary has both an epistemically subjective and a true health judgement demonstrates that, to quote Joseph Raz, “[r]easons for believing that a thought is true need not be available to the person who has it, or at least need not be available to [her] as reasons for the thought”³. The conclusion to draw from this is that epistemic subjectivity *by itself* does not or need not make an objectively true statement as defined by (S*) about ontologically objective objects (O*) crucially value-dependent.

These considerations ought to persuade us that epistemic subjectivity does not or need not *by itself* sound the death-knell for a naturalist account of health and disease. And if this is correct, then it seems we ought to accept the claim that epistemic objectivity is not (or need not be) a necessary condition of a naturalist account of health and disease.⁴

All of this strongly suggests that the epistemic account of the subjective-objective distinction is not and should not be viewed as being the fundamental and central issue

³ Joseph Raz (2002), *op cit.*, p. 124.

⁴ It may be worth mentioning, if only very briefly, another reason to insist that the epistemic sense of the subjective-objective distinction is not the basic issue between the naturalist and normativist concerning health and disease. In my view, the classic epistemological “other minds” issue gains some traction here. The philosophical issue of “other minds” is, very crudely, that we can never have “direct” knowledge of what A’s beliefs, attitudes, and feelings exactly are, or so it is claimed. The connection of this problem with (E*) is that if we are to know or at least reasonably believe that A’s belief or knowledge claim that P is verified or falsified independently of anyone’s beliefs, attitudes, or feelings about P, then we are going to, at a minimum, need to be aware of the content of A’s belief or knowledge claim. At any case, I will not pursue this issue because it would take the discussion into issues of radical skepticism far beyond the scope of this dissertation.

between the naturalist and normativist. And thus, by pushing the epistemic sense (E*) temporarily on one side, we may begin to outline the fundamental difference between naturalism and normativism with some broad strokes.

In my view, ontological and semantic objectivity are two fundamental features of naturalism. Correspondingly, the naturalist insists that (O*) and (S*) objectivity yields a satisfactory account of health and disease. The normativist instead insists that a satisfactory account of health and disease will be such that true health judgements properly track the subjectivity of (O*) and/or (S*). This is the sense in which (S*) and (O*) objectivity is the basic issue between the naturalist and normativist.

3. The varieties of normativism

3.1 Three broad varieties

If it is remembered that as I pointed out in Chapter 1 there are two ways in which something can fail to be objective by O* and/or S* (either it depends for its existence and nature on the existence of some norm or it depends for its existence and nature on the existence of some attitude or belief). Using the ideas which I have now outlined we can view normativism as having three broad varieties. Normativism might be:

- (I) semantically objective but ontologically subjective
- (II) semantically subjective but ontologically objective
- (III) semantically subjective and ontologically subjective

Each division corresponds to a distinct way in which one may, either explicitly or covertly, depend on values or norms in specifying a normativist account of health.

(I) Semantically objective but ontologically subjective

Normativism of this sort provides an account of health and disease that makes health judgements a function of semantically objective statements about ontologically subjective entities. To illustrate, consider again the statement “Socrates is healthy”. A normativist of this sort will want to say that Socrates is healthy is to say something like the following: It is the case that Socrates possesses properties p_1, \dots, p_n , where each property p_i is a property whose presence or absence is true independently of Socrates’ or anyone else’s belief that he is healthy, or attitudes or feelings about his being healthy. That is to say, the statement “Socrates is healthy” reports a true health judgement that is semantically objective by (S*). And the fact that Socrates is healthy according to some criteria makes the statement “Socrates is healthy” true.

But this first kind of normativist can be an ontological subjectivist, for a normativist of this ilk can also then say that at least some of the properties that inform the content of the health judgement will be (O*) subjective properties in the following sense. A property p_i is a value- or culture-dependent property if the fact that the p_i exists is dependent on particular human beliefs about p_i or attitudes to p_i , and/or dependent on the acceptance in the community where the judgement is being made of certain norms. Note, for example, an immediate upshot of this normativist framework is its capacity to square with our strong intuitions that health is a robust state of being which is in some important sense to be pain-free and to function well in a modern society.

Suppose we were to count the state of being in serious discomfort to be an unhealthy state. To employ the above framework, the statement “Socrates is healthy” reports a semantically objective fact of the matter—Socrates possesses properties p_1, \dots, p_n —in the

sense that the truth and falsity of this (that Socrates does not have depression, carpal tunnel syndrome, high arch foot, Raynaud's disease, ADHD, for instance) is independent of Socrates' or anyone else's beliefs about these properties, or attitudes or feelings about the possession of those properties. However, the existence of the fact *that* Socrates is not experiencing any *serious* pain or discomfort—facts counted to be requisite for health—clearly is dependent on someone's attitudes and feelings. This adds the ontologically subjective component, by (O*).

Taking a step back, notice that the claim, as so far stated, is that semantic objectivity holds for the first-order issue regarding the health judgement "Socrates is healthy" in the sense that the truth conditions of the statement that "Socrates possesses properties p_1, \dots, p_n " (let us refer to this as P) make the truth of P independent of anyone's beliefs about the truth of P , feelings or attitudes to the truth of P . As it stands, however, there is no reason to think that the (S*) objectivity at this level extends to the second-order issue of the objectivity of claims to the effect that, for example, the properties p_1, \dots, p_n are the criteria for health.

The point to press here is that first-order issues regarding health judgements are distinct and importantly different from second order issues. Thus, the insistence that an individual A^i has the disease D^1 (or is healthy) is objective by (S*) and/or (O*) is perfectly compatible with the insistence that the properties p_1, \dots, p_n are the criteria for D^1 (or for health) is not objective by (S*) and/or (O*). All of this I hope became clear in my exposition of the ADHD example above (Section 2), though I do think this point is often underappreciated and, worse, sometimes ignored. At any rate, we would do well to remain keenly aware of the sense in which there are at least two levels—a first-order and second-

order—with distinct and different issues in which objectivity and subjectivity may be at play.

This brings to light another, rather different way in which one may inject an ontologically subjective component into their account of health. This alternative way makes health and disease to be norm-constructed. A representative example of this sort of framework to my mind is Lester King's view of health and disease:

Disease is the aggregate of those conditions which, judged by the prevailing culture, are deemed painful, or disabling, and which, at the same time, deviate from either the statistical norm or from some idealized status. Health, the opposite, is the state of well-being conforming to the ideals of the prevailing culture, or to the statistical norm.⁵

Notice that on this normativist account of health, to say “Socrates is healthy” is for the speaker to make a judgement about Socrates whose truth is a function of what the norms of the prevailing culture count as being non-painful or disabling. *A fortiori*, this is to make health claims depend on one or more cultural norms that do not exist in the world independent of human attitudes and feelings. Here we can clearly see how a normativist account of health and disease may employ ontologically subjective facts by depending on norms to construct the requisite properties for health and disease. This, however, is not the only way an account of health and disease can be norm-constructed.

(II) Semantically subjective but ontologically objective

Normativism of this sort provides an account of health and disease that makes health judgements a function of semantically subjective statements about ontologically objective

⁵ Lester S. King (1981), "What is disease?", in Caplan, Engelhardt, Jr., & McCartney (Eds.), *Concepts of health and disease: Interdisciplinary perspectives*. Reading, Mass: Addison-Wesley, 107-118, at p. 112.

entities. An important difference between this sort of normativism and (I) is thus that the existence of the properties requisite for health is independent of the beliefs, attitudes or feelings of any perceiver about the properties (O* objectivity). That is to say the content of the health judgement and what is counted to be requisite for health does not depend on the presence of any properties, facts, states, and so on that are not discovered in nature. We are, however, constructing the truth or falsity of the judgement, in that its truth function is in part at least a function and hence depends at least in part on the values and/or norms of an individual, a group, a society, a culture, et cetera, and hence is, semantically speaking, subjective.

So to say that some organism is healthy on this account is to say something like the following: That, on one hand, the organism possesses properties p_1, \dots, p_n where that the organism possesses each property p_i is a *brute* fact, but, on the other hand, that the truth or falsity of this judgement (i.e., that *these* particular properties p_1, \dots, p_n are requisite for health) is a function of at least *two* parameters: (i) there exists in the society in which health is being ascribed some *social construction*, a construction which specifies certain conditions C_1, \dots, C_n for what is to count as a *healthy* state and (ii) the particular organism said to be healthy satisfies C_1, \dots, C_n in virtue of possessing properties p_1, \dots, p_n . An example of this sort of normativism would be an account of health emphasizing a particular lens of a view about what counts as a life that is free *enough* of discomfort that is constructed, at least in part, by the beliefs, attitude or feelings of humans and, in virtue of this focus, counts the particular properties p_1, \dots, p_n to be requisite for health.

To take a prominent example from the literature, Jerome Wakefield seems to be a proponent of this sort of normativism. Consider that he proposes a hybrid value and

factual account he calls the “harmful dysfunction” (HD) analysis of the concept of disorder⁶ (instead of disease he employs the term ‘disorder’) that he describes as follows:

According to the HD analysis, a disorder is a *harmful dysfunction*, where “harmful” is a value term, referring to conditions judged negative by sociocultural standards, and “dysfunction” is a scientific factual term, referring to failure of biologically designed functioning. In modern science, “dysfunction” is ultimately anchored in evolutionary biology and refers to failure of an internal mechanism to perform one of its naturally selected functions.⁷

On this sort of normativism, states of health or disease are thus crucially like, say, the value of gold. For example, suppose I am holding a piece of gold weighing one ounce. As I type this, the value of one ounce of gold in Canadian dollars is very close to \$1,496.⁸ Now, we can settle the issue of whether what I am holding in my hand is a piece of gold weighing one ounce by *objective facts* of the matter (i.e., its weight, whether it contains 79 protons and melts at 1948 degrees Fahrenheit, and so on). That it *is* one ounce of gold is a fact independent of anybody’s beliefs, attitudes, or feelings about it. These are “brute facts”.

However, the statement that an ounce of gold is worth \$1,496 is in a very important sense—a second order sense—semantically subjective. This is because the truth or falsity of the fact that an ounce of gold is worth \$1,496 is in part a function of constructed sociocultural standards or norms. Like, for instance, the value we give to the Canadian

⁶ “Disorder in the medical sense”, Wakefield states, “is a hybrid value and factual concept. See, Jerome C. Wakefield (2005), “Biological function and dysfunction”, in David M. Buss (Ed.), *The handbook of evolutionary psychology*. Hoboken, New Jersey: John Wiley & Sons, Inc. pp. 878-902, at 891.

⁷ Jerome C. Wakefield (2007), “The concept of mental disorder: Diagnostic implications of the harmful dysfunction analysis”, *World Psychiatry* 6: 149-156, at p. 149. See also, Jerome C. Wakefield (1992), “The concept of mental disorder: On the boundary between biological facts and social values”, *American Psychologist* 47(3): 373–388, especially at p.384.

dollar and the desire we have for gold. We do not settle the truth or falsity of the monetary value of an ounce of gold simply by discovering facts that exist independent of any perceiver—norm-constructed facts are required. This makes it a value-dependent endeavor.⁹

And this same second order sense of subjectivity can be said to be at play in Wakefield's state of "*harmful dysfunction*", in that the truth or falsity of what dysfunctions (i.e., alleged ontologically objective facts) are counted to be harmful is settled by what is deemed negative by sociocultural standards. Because sociocultural standards are the kinds of facts that exist only as a matter of agreement between members of culture or society, it is clear that Searle's notion of "institutional facts" is at play here. That Wakefield's conception of harmful dysfunction depends on sociocultural standards or institutional facts is the way in which his account of health and disease is norm-constructed.

It is perhaps worth mentioning that there are as many ways to cash out this sort of normativism as there are norms or values one may accept to justify counting differing brute facts to be requisite for health. Some ways, of course, will generate more plausible accounts of health and disease than others.

(III) Both semantically and ontologically subjective

Normativism of this third sort provides an account of health and disease that makes health judgements a function of semantically subjective statements about ontologically subjective

⁸ <<http://goldprice.org/gold-price.html>> Accessed June 12, 2011.

⁹ Notice here that once the particular norms are in force to yield the requisite shared meanings there is a sense—a first-order sense—whereby the proposition that "the object in my hand is worth no less than \$1,496" is objectively true by (S*).

entities. Correspondingly, it shares the former feature with (II) and the latter feature with (I). We may therefore expect to find values and/or norms playing an active role in two distinct ways. Firstly, we may expect the truth (or falsity) of the judgement that “ $p_1...p_n$ is requisite for health” to be a function of the accepted values of an individual, a group, a society, a culture, or the like. And, secondly, we may expect the existence of at least one or more of the properties $p_1.....p_n$. to depend on particular human attitudes, beliefs, or feelings.

To illustrate this framework, consider the (in)famous account of health advanced by the World Health Organization (WHO) and, in particular, their 1946 definition of health:

Health is a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity.¹⁰

This positive view of health has been a matter of significant controversy, and I believe rightly so. An immediate worry is that the standard for health is set so high that nobody is—or likely will ever be—healthy.¹¹ If, however, some people are in fact healthy, then we need to know what a state of *complete* physical, mental and social well-being actually amounts to, in order to sensibly distinguish healthy individuals from unhealthy ones. For our purposes, it will suffice to note that whether or not an individual is properly counted to be healthy and, as such, in a “complete state of well-being” depends upon our understanding of physical, mental and social well-being.

Notice that opposing views of physical, mental, and social well-being will virtually

¹⁰ World Health Organization (1946), *Constitution of the World Health Organization*. Geneva: Author, p. 2.

¹¹ According to Roberto Mordacci & Richard Sobel (1998), “Health: A comprehensive concept”, *Hastings Center Report* 28(1): 34-37: if the hopelessly utopian WHO definition is taken literally, then it is meaningless. On this point, I agree.

guarantee there to be different criteria for what is to be properly counted as a complete state of well-being. If it turns out, as seems very likely given the inclusion of social well-being, that any plausible conception of “a complete state of well-being” is in some important sense a function of human wants and desires, then the statement “Socrates is healthy” reports a semantically subjective fact of the matter. This is because of the way in which the claim that p_1, \dots, p_n are the criteria requisite for health (i.e., complete well-being) is made true: It is made true, in part, by a particular conception of states of well-being and which conception is at work here is a function of which are the values of an individual, a group, a society, a culture, etc., that are the ones that are (in fact) accepted.

As we will see below, determining the requisite criteria for a “state of complete physical, mental, and social well-being” is not simply a matter of picking a list of discovering ontologically objective natural properties—it is much trickier than that. But the important point to be emphasized *here* is the point that in different groups, societies and cultures different values and norms may be accepted and in force, despite—and perhaps against—the accepted values or norms of any one individual.

Perhaps, however, we might spell out a normativist account for health using the WHO definition rather differently by viewing its rôle in health determinations not in terms of the fact that it is accepted by the WHO but at a more individual level, i.e., in terms of fact that it is accepted by some individual as part of his or her own personal values or norms. To illustrate, suppose if individual *A* judges that person *B* is not healthy he does so by checking if *B* satisfies the WHO criteria for health and *A* uses these criteria because and in so far as he accepts the WHO criteria for health as satisfactory criteria. Using the terminology I have outlined we can see how and why to adopt *this* version of normativism

can be a serious source of problems, practical problems, but serious problems for all that, for, given that people do have various and in cases opposing wants and desires and vastly different conceptions as to what would count (to put the point in a nutshell) as a flourishing life. Hence health determinations on this account become much too variable, indeed individualist, for use in developing guidelines for governments, for health care administrators, and even for front line health care workers.

But this last point brings out the more general problem with the WHO definition is whether the content of the WHO definition is used as a norm for health determinations by groups or by individuals. For given the diversity of wants, desires and conceptions of what counts as a flourishing life that I just mentioned, it is clear for obvious reasons that the WHO definition as it stands lacks any sort of principled standard that would distinguish “health” in some sense from what simply might reasonably—or unreasonably!—be thought to contribute to one’s well-being. The recognition of this problem is well-documented in the philosophy of health literature. The most common line of criticisms against the WHO definition have centered on the way in which health is made to include nearly all of well-being. These criticisms typically proceed by providing grounds for believing that the WHO definition is too inclusive because it erroneously embraces *any* alleged criteria of well-being as integral components of health—criteria, for instance, such as achieving happiness¹², finding a four-leaf clover,¹³ and realizing an IQ of 400.¹⁴ .

¹² Leon R. Kass (1981), "Regarding the end of medicine and the pursuit of health", in A. L. Caplan, H. T. Engelhardt, Jr., & J. J. McCartney (Eds.), *Concepts of health and disease: Interdisciplinary perspectives*. Reading, Mass: Addison-Wesley, 3-30, at pp. 5-6.

¹³ Kenneth A. Richman (2004), *op cit.*, at pp. 27-61.

¹⁴ Andrew Stark (2006), *The limits of medicine*. New York, NY: Cambridge University Press, at p. 59.

3.2 Local normativist differences in their accounts of objectivity and subjectivity

For each of these three broad families of normativist accounts of health described above, I was careful to use “objective” and “subjective” narrowly, consistently taking “objective facts” to be “brute” facts as defined in Chapter 1. Brute facts recall are those “intrinsic” facts about the world whose mode of existence is logically independent of any human representation of them. That is to say, I have been using “objective” in the sense defined in (O*iii):

(O*iii) Some fact *F* is an ontologically objective *fact* iff the fact that *F* exists is not a function of any person *A*’s beliefs *about* whether *F* is a fact or attitudes *to F*’s being a fact. Some fact *F* is a *subjective* fact if it is not an objective fact.

“Subjective” facts then are facts that are created, value-laden and/or facts that are created, value-dependent, and/or facts that are such that their mode of existence depends, in some important sense, on particular beliefs about them or attitudes or feelings to their existence.

Having said that, what must be acknowledged is that many authors within the modern debate over health and disease seem to be working with a very different sense of objectivity and subjectivity and *a fortiori* there is an importantly different sense of objective (and subjective) facts, states, judgements, values and norms at play in the literature. This different understanding of objectivity/subjectivity lends itself to yet further distinctions—distinctions which may be called “normativist” distinctions.

To illustrate, consider semantically subjective judgements, i.e., judgements where the truth or falsity of the judgement depends on certain beliefs about the facts being claimed and attitudes or feelings to these facts. At this point, we can see exactly how it is

that there are indeed different varieties of normativist theory of health. For we have one variety of normativist theory if the truth or falsity of the health judgement is, according to that theory, to be settled by taking into account people's (individual) values, attitudes or feelings, and a different variety if it is to be settled by taking into account norms in force in the community, society, or group where the health judgement is being made. I can and will put this point by saying that normativist views have various "local" differences one from the other.

Now exactly *whose* values (everyone's? at least one individual? a particular individual or group?) are to make-up the requisite values and *whose* (everyone's? at least one individual? a particular individual or group?) norms are to be factored into the health determination on the theory in question is one of the most difficult and most important issues that normativists need to decide on when developing their different theories. Fierce disagreement exists between those who insist the requisite normativity is to stem from "subjective norms"—i.e., norms framed by the individual making the health determination—and those who maintain it must stem from norms which are in some sense "intersubjectively valid" or perhaps even in some to be specified "objectively valid"—i.e., I suppose, norms framed in terms of what is valued, in some important sense, by *all* individuals and valuable in some sense *for* all individuals.

Obviously therefore depending how we mix and match these various conceptions of subjectivity so we get "local" differences in the variety of normativist theory under consideration.

For those normativists who espouse a version of normativism that is semantically subjective but ontologically objective (or perhaps which espouses "intersubjective

validity” in the sense outlined below), a fundamental problem facing them will be that there is no clear agreement even amongst philosophers about which kinds of phenomena, if any at all, are properly *generally* to be counted as constituting objectively existing or intersubjectively valid criteria. Hence, when we turn to the *local* issues that arise for normativists when trying to spell out what *they* in *their* version should say about these matters (i.e., how they should mark their specific normativist views from other normativist views), then matters ramify into complex issues in all directions.

On the face of it, for example, “having a full stomach at the end of every meal” might well be valued by all and though *prima facie* to be valuable for all. But then further reflection reveals that on the contrary *for some* people having a full stomach at the end of a meal is *not* valuable. For some having a full stomach at the end of every meal is a major cause of undesirable sequelae in old age. So it is not clear whether the normativist ought to be seeking norms for health that are in some sense objectively valid *for all* (or perhaps intersubjectively valid *for all*) or norms which are framed in ways which make them in some sense subject relative. In short, such normativist attempts to establish a persuasive conception of intersubjectively valid or (in some sense) objective norms is as contentious and unsettled as it tends to be in philosophy generally.

In my view, a useful way to capture and to appreciate the “local” sense of the subjective/objective distinction is to approach it in terms of a familiar question in the general debates in philosophy of ethics: “is *x* good because it is valued or desired (subjectivist/non-objectivist) or is *x* valued or desired because it is good (objectivist)?”¹⁵

¹⁵ The subjectivist/objectivist divide can be traced at least as far back to Plato’s dialogue the *Euthyphro*

Indeed, some writers view this familiar question to express the core disagreement between objectivist and subjectivist accounts of values.¹⁶ So, using this contrast, we might rephrase the question to be: “are the criteria p_1, \dots, p_n ultimately properly counted to be requisite for health because:

- (a) what is specified in the criteria or counted valuable by *an individual* valued or desired by an individual (a normativist theory that took this approach would be *locally*-subjectivist); or
- (b) what is specified in the criteria is adopted as something to be counted valuable *for all* (a normativist theory that took this approach would be *locally*-objectivist or, perhaps, more familiarly it would be adopting a theory that purports to be intersubjectively valid).

In this way, (a) and (b) track the two poles of a fierce disagreement that exists within the normativist camp. The upshot I propose thus: If we can show that some author has adopted as a criterion for a health judgement that is a function of what is defined above as either (a) subjectivist or (b) locally-objectivist criteria, then we can be sure he or she is operating from the normativist perspective. And this is so even if he or she suggests what purports to be an “objective” (or perhaps an intersubjectively valid) component of his or her account of health. With the realization that even normativists may adopt accounts of the components of health that are “locally” purportedly objectivist or purportedly intersubjectively valid, we have a useful framework in which to classify the many different normativist accounts. This way of putting things is important because in what would otherwise be a rather confusing way a line that has been drawn in the literature

where Socrates (10a) poses the question “Is what is holy holy because the gods approve it, or do they approve it because it is holy?”

¹⁶ For example, Andrei Marmor (2001), *op cit.*, at p. 160.

between normativist theories of health that are (a) “subjectivist” (e.g., Culver & Gert; Kovacs; Nordenfelt) and those that are (b) “objectivist” (e.g., King; Sade; Lennox).

4. Moving forward

As we move forward, we should keep firmly in mind that the crux of the modern debate concerning health and disease is at bottom the issue over the extent to which humans discover and/or, if at all, create the factors which fix states as either healthy or diseased. In what follows, guiding this dissertation will be the strong belief that we should take seriously the naturalist *modus operandi* to advance a value-and norm-independent account of health and disease. I say this for two main reasons.

First, and most obviously, when it comes to many states of health and disease it is undeniable that there are facts of the matter to be mistaken about. A primitive society, for example, might well value, and indeed worship, a child born with Hutchinson-Gilford Progeria Syndrome (hereafter, simply Progeria) because of its symptoms resembling the appearance of accelerated aging in children and the value they place on reincarnated demigods. Such a society might even declare a child with Progeria to be beyond healthy.

But such a declaration betrays a mistaken view of reality. Regardless of the accepted values and cultural norms, the biological fact remains that this child has a fatal genetic condition, where death occurs at an average age of thirteen years (with a range of about 8-21 years).¹⁷ Whatever the values and norms—and no matter how well-entrenched

¹⁷ Progeria Research Foundation, “Progeria 101/FAQ” <www.progeriaresearch.org/progeria_101.html> Accessed July 4, 2011.

—they do not *ipso facto* ameliorate ontologically objective biological dysfunction. The reality is the disease *ceteris paribus* will be fatal. As Brian Leiter recognizes and explains: ontologically objective entities make themselves felt *causally* and, as such, give us an *external* criterion for objectivity.¹⁸

To further press this important point consider diseases that are curable or, at least, treatable. It is relatively uncontroversial that there are numerous values or norms that, if operationalized, would transform our beliefs and behaviours to alter our social response to any number of diseases. Nor is it controversial that, at this point in time, it would be absurd to press for a culture shift that would have medical practice deny, for instance, the disease-status of cancer and forgo administering any treatment. Beyond the absurd, however, lies the stark reality for many individuals: their very survival depends on the capacity for medical practice (broadly construed) to engage with the world as it actually *is* and accurately assess the biological facts of the matter. What we should therefore not want is for values or norms to create factors that run against ontologically objective facts of the matter when it comes to health and disease. Probably the best (and perhaps only) way to preclude this from happening is to not allow health judgements to become a function of values or norms. This alone provides sufficient reason to take naturalism seriously.

Second, and no less importantly, the decision to opt for a normativist account of health and disease carries with it the legitimate worry that health claims will become a function of dubious values or norms, of which there exists no shortage. That simple fact is

¹⁸ Brian Leiter (2007), *op cit.*, at pp. 261-262.

precisely what prompts a healthy skepticism towards a wholesale commitment to normativism.

It is difficult to overstate the claim that enormous consequences, for both society and the individual, turns on how we negotiate between the values and/or norms that might be deemed requisite for a satisfactory account of health and disease. History is riddled with shameful instances where dubious values and norms were employed to give disease-status to behaviour viewed to be deviant and/or undesirable. The literature often-cites the fact that in the middle of the 19th Century, some doctors, most notably in southern United States, identified a slave's attempt to escape as symptomatic of the disease "drapetomania"—the disease causing slaves to run away from their masters.¹⁹

Perhaps even more troubling, Lawrie Reznick remarks that there was a time when the desire for masturbation and homosexuality were each considered conditions that constitute disease and, most unfortunately for those individuals diagnosed with such a "condition", the respective medical treatments were to cauterize the clitoris and burn out the part of the brain (the hypothalamus) thought responsible for homosexual behaviour.²⁰ Enormous consequences indeed. Consequences, I submit, that make one resistant to embrace the view that values and/or norms are, in one way or another, the *sine qua non* of a satisfactory account of health and disease.

Although drapetomania, masturbation and homosexuality no longer have disease-

¹⁹ See, for instance, Lawrie Reznick (1987), *The Nature of disease*. London: Routledge and Kegan Paul, at p. 17; and Arthur L. Caplan (1993), "The concepts of health, illness and disease", in W.F. Bynum & Roy Porter (Eds.), *Companion encyclopedia of the history of medicine, Vol. 1*. London: Routledge, 233-248, at p. 244.

²⁰ Reznick (1987), *op cit.*, pp. 6-8.

status with modern medical practice,²¹ it is probably naïve to think that questionable values and norms do not drive some of our current health claims.²² I think it can plausibly be argued there are some notable “conditions” that presently have a medical diagnosis and medical treatment—e.g., attention deficit/hyperactivity disorder (ADHD); chronic fatigue syndrome (CFS), Asperger’s syndrome; dysthymia; oppositional defiant disorder; post-traumatic stress disorder (PTSD); tobacco use disorder; multiple chemical sensitivity (MCS); erectile dysfunction (ED); and female orgasmic disorder (FOD); to name but ten—which lack clear medical pathology *qua* biological dysfunction.

That current medical practice prescribes medical treatment for these and other conditions is clearly significant for North Americans. The National Institute of Mental Health and Canadian Mental Health Association, for example respectively report that 26.2 percent of Americans ages 18 and older suffer from a diagnosable mental disorder in a given year²³ and 21.3 percent of adult Canadians will suffer a mental disorder in their lives.²⁴

²¹ Sadly, the view that homosexuality is a disease remains stubbornly resistant to change. Recently, India’s Health Minister Ghulam Nabi Azad publically derided homosexuality “as an unnatural ‘disease’ from the West” stating that, “Unfortunately this disease has come to our country too...where a man has sex with another man, which is completely unnatural and should not happen, but does”. The Associated Press, “India’s health minister calls homosexuality a ‘disease’” July 5, 2011 <<http://www.cbc.ca/news/world/story/2011/07/05/india-gay-slur.html>> Accessed July 5, 2011.

²² For an interesting and persuasive argument that powerful social forces within and outside of medicine are fuelling the medicalization of life problems see, Peter Conrad (2007), *Medicalization of society: On the transformation of human conditions into treatable disorders*. Baltimore: The Johns Hopkins University Press.

²³ See National Institute of Mental Health, “The numbers count: Mental disorders in America” <<http://www.nimh.nih.gov/health/publications/the-numbers-count-mental-disorders-in-america/index.shtml>> Accessed September 13, 2010.

²⁴ See Canadian Mental Health Association, “Statistics”. <http://www.cmha.ca/bins/site_page.asp?cid=284-285-1258-1404&lang=1> Accessed September 13, 2010.

These numbers probably should not be so high. To my mind, Ethan Watters²⁵ provides some valuable, and tremendously troubling, insight into why so many North Americans “suffer” from a diagnosable mental disorder. It is actually worth spelling it out, even though it will take a little while, because it is a perfect real-world example of the way in which a view of health and disease becomes saturated with tendentious values and is precisely the kind of scenario the naturalist aims to preclude with a value- and norm-independent account of health and disease. It is also a good example of the sort of powerful and influential stakeholders that have vested interests in changing how we think about health and disease.

Watters tells the story of how, in late 2000, the pharmaceutical giant GlaxoSmithKline (GSK) went about changing the cultural understanding of depression in Japan. What is particularly striking about Japan was that traditionally the Japanese had a deep embrace for sadness; moreover, they did not even have a word for “depression” beyond *utsubyô*, a mental illness as “chronic and devastating as schizophrenia”.²⁶ Most experiences of melancholy were simply counted to be non-pathological. In response to this, Watters writes, GSK underwent a “mega-marketing campaign” to market depression as “the cold of the soul” to be a common affliction, easily treatable—with their drug Paxil! GSK’s campaign to reshape the very consciousness of the Japanese consumer in order to accept GSK’s label of depression seems to have been successful: The *GSK Annual Review 2004* reports “the strong performance of the product [Paxil] in Japan with sales of £171 million (up 25%) and the performance of *Paxil CR* which generated sales of £396

²⁵ Ethan Watters (2010), *Crazy like us: The globalization of the American psyche*. New York: Free Press.

million (up 14%)”.²⁷

Viewed from a distance, many will undoubtedly find these jumps in profits impressive. However, beneath the increase in Paxil sales lies a simple and salient point that underscores the importance of the cultural dimension of health and disease: Japanese physical and mental functioning probably did not change, but its cultural understanding and social response to depression surely did.²⁸

This kind of mega-marketing campaign to change the definition of and social response to a particular “condition” is, of course, not unique to Japan. One cannot seriously deny that profit-seeking drug companies engage North Americans with similarly sophisticated marketing campaigns. In fact, Paxil’s product director, Barry Brand proudly states as much when he says, “Every marketer’s dream is to find an unidentified or unknown market and develop it. That’s what we were able to do with social anxiety disorder”.²⁹ Indeed GSK’s well-orchestrated marketing of Paxil in the United States was successful:

CNS [central nervous system] is our largest product sales category, led by *Seroxat/Paxil* which became number one in the US selective serotonin reuptake inhibitor market for new retail prescriptions in 2000. We expect to expand its value in 2001 from approvals to market the product to treat general anxiety disorder and post traumatic stress disorder.³⁰

²⁶ Ibid., p.193.

²⁷ GlaxoSmithKline, *GSK Annual Review 2004: New challenges, new thinking*, at p. 14. <<http://www.gsk.com/investors/reports/ar2004/annual-review-04/business.htm>> Accessed July 4, 2011. And sales would continue to grow as the *GSK Annual Review 2008* reports a 4% Paxil growth in Japan. <<http://www.gsk.com/investors/annual-reports-archive.htm>> Accessed July 4, 2011.

²⁸ Interestingly, Watters (2010), *op cit.*, pp. 200 & 249 contends that cultures are more susceptible to embrace new beliefs about mental health and disease during times of social anxiety or discord.

²⁹ Shankar Vendantam (2001), “Drug ads hyping anxiety make some uneasy”. *Washington Post*, July 16, A01. Cited in Conrad (2007), p. 18.

³⁰ GlaxoSmithKline, *GSK Annual Review 2000: It’s about you*, at p. 3. <<http://www.gsk.com/investors/>

Buried deep within GSK's stated aim to expand the market of Paxil is a conflict between values: maximizing company profits and minimizing health risks. The reality is that Paxil produces a number of serious side-effects, some of which can be deadly. One such side-effect, for example, is the increased tendency for suicidal ideation and/or behaviour.³¹ Concomitant with the increasing use of Paxil are the increasing number of serious Paxil side-effects. In the wake of GSK's successful campaign to get more people to use Paxil to treat a greater number of "conditions", there has been an increase in litigation against the company for a variety of Paxil side-effects. In response, there is a disturbing trend in the pharmaceutical industry to resolve legal issues with monetary settlements and press on. In a July 2010 press release, GSK announced that:

...it expects to record a legal charge *for the second quarter of 2010* of £1.57 billion (\$2.36 billion US)...The settlements and agreements in principle to settle include product liability and anti-trust litigation relating to Paxil... The company continues to work to resolve an investigation commenced by the US Attorney's Office for the District of Colorado into the Group's sales and promotional practices.³²

At a minimum, GSK shows a troubling disregard for the serious—and sometimes fatal—side-effects associated with their drugs. Within the pharmaceutical industry such disregard is, to be sure, limited neither to Paxil nor to GSK. In fact, five different pharmaceutical companies are reported to have recently admitted to federal charges of

[annual-reports-archive.htm](#)> Accessed July 4, 2011.

³¹ See, for example, Allan V. Horowitz (2010), "Pharmaceuticals and the medicalization of social life", in Donald W. Light (Ed.), *The risks of prescription drugs*. New York: Columbia University Press, pp. 92-115. To press the point Horowitz (p. 95) notes that, in 2004, attorney general of New York State, Eliot Spitzer filed a lawsuit against GSK on charges of fraud by failing to disclose that adolescents taking Paxil had higher rates of suicidal ideation than those taking a placebo.

³² GlaxoSmithKline, "GlaxoSmithKline legal update", July 15 2010. Italics mine. <http://www.gsk.com/media/pressreleases/2010/2010_pressrelease_10076.htm> Accessed July 4, 2011.

illegally “off-label” marketing *one of their* respective psychoactive drugs.³³ One drug each out of the hundreds and hundreds each company manufactures.

It is a thoroughly dreadful feature of our political and social systems that powerful and influential stakeholders so often seem to be able to put profits and their agenda before people and blur the boundaries between sophisticated marketing and medical science. To be sure, the drive to medicalize problems in living³⁴ is not without a vast array of powerful and influential stakeholders intent to champion the next pharmaceutical panacea.

Knowing that, let me conclude by saying this: If we are truly concerned not to advance health claims that are scientifically dubious and/or ideologically tendentious, then it follows that we have another *prima facie* reason to embrace the naturalist pursuit for a satisfactory value-free account of health and disease.

The aim of this dissertation is hence to take naturalism seriously. The next chapter examines whether the naturalist is correct to suppose that a satisfactory account of health and disease lies within the province of naturalism. To assess this view, it is necessary to examine what is generally agreed to be the most influential naturalist account of health and disease: Christopher Boorse’s biostatistical theory. To this chapter, we now turn.

³³ Marcia Angell (2011b), “The illusions of psychiatry”, *The New York Review*, July 14, 2011. <<http://www.nybooks.com/articles/archives/2011/jul/14/illusions-of-psychiatry/?page=1>> Accessed July 22, 2011.

³⁴ To my knowledge, Thomas Szasz popularized the phrase “problems in living” in his scathing attack against psychiatry. Briefly, Szasz argued that mental illness is not a disease but is instead problems in living from certain psychosocial, ethical, or legal norms. See Thomas S. Szasz (1960), “The myth of mental illness”, in A. L. Caplan, J. J. McCartney, & D. A. Sisti (Eds.), (2004), *Health, disease and illness*. Washington, D.C.: Georgetown University Press, pp. 43-50.

Chapter Three

On a Naturalist Theory of Health: A Critique¹

1. Introduction

Christopher Boorse has been over many years trying to develop a “naturalist” account of health and disease.² It is generally agreed his biostatistical theory (or, simply, BST) is the most influential naturalistic account of health and disease.³ In a nutshell, his basic idea is that a disease state is a state in which an organism functions in some sense subnormally and hence a healthy state is a state in which an organism does not function subnormally—it will be obvious that this idea could relatively easily be extended so that we can talk of not only healthy and diseased organisms but also of healthy and diseased organs. He

¹ A version of this chapter was published as J. David Guerrero (2010b), “On a naturalist theory of health: A critique”, *Studies in the History and Philosophy of Biological and Biomedical Sciences* 41(3): 272-278.

² Christopher Boorse (1975), “On the distinction between disease and illness”, *Philosophy and Public Affairs* 5(1): 49–68; (1976a), “What a theory of mental health should be”, *Journal for the Theory of Social Behaviour* 6: 61-84; (1976b), “Wright on functions”, *The Philosophical Review*, 85(1): 70–86; (1977), “Health as a theoretical concept”, *Philosophy of Science* 44: 542–573; (1987), “Concepts of health” In D. Van De Veer, & T. Regan (Eds.), *Health care ethics: An introduction*. Philadelphia: Temple University Press, 359-393; (1997), “A rebuttal on health”, in J. M. Humber, & R. F. Almeder (Eds.), *What is disease?* Towata, New Jersey: Humana Press, 3-134; (2002), “A rebuttal on functions”, in A. Ariew, R. Cummins & M. Perlman (Eds.), *Functions: New essays in the philosophy of psychology and biology*. Oxford: Oxford University Press, 63-112; and Boorse (2010), “Disability and medical theory”, in D. C. Ralston and J. Ho (Eds.), *Philosophical reflections on disability*. Dordrecht: Springer, 53-88.

³ To list just some of the authors making this claim: József Kovacs (1998), “The concept of health and disease”, *Medicine, Health Care and Philosophy* 1(1): 31-39; Ron Amundson (2000), “Against normal function”, *Studies in the History and Philosophy of Biological and Biomedical Sciences* 31(1): 33-53; Rachel Cooper (2002), “Disease”, *Studies in History and Philosophy of Biological and Biomedical Sciences* 33(2): 263-282; Lennart Nordenfelt (2004), “The Logic of Health Concepts”, in George Khushf (Ed.), *The Handbook of Bioethics*. Dordrecht: Kluwer Academic Publishers, 205-222; Kenneth Richman (2004), *op cit.*; George Khushf (2007), “An agenda for future debate on concepts of health and disease”, *Medicine, Health Care and Philosophy* 10: 19-27; Dominic Murphy (2006), *Psychiatry in the scientific image*. Cambridge, Mass: The MIT Press; Thomas Schramme (2007), “A qualified defence of a naturalist theory of health”, *Medicine, Health Care and Philosophy* 10: 11-17; Marc Ereshefsky (2009a), “Defining ‘health’ and ‘disease’”, *Studies in the History and Philosophy of Biological and Biomedical Sciences* 40(3): 221-227; J. David Guerrero (2010b), *op cit.*; and Elselijn Kingma (2010), “Paracetamol, poison, and polio: Why Boorse’s account of function fails to distinguish health and disease”, *British Journal for the Philosophy of*

wants to argue that the BST's account of health and disease are both value- and norm-independent. The upshot, Boorse contends, is that “[m]edicine has a distinctive theoretical foundation in a value-free science of health and disease”.⁴

In his most recent papers he draws upon his earlier accounts again using the notions of “normal functional ability”, “impairment”, “statistical normality”, “reference class”, “natural class of organism”, “uniform functional design” and “internal state”. The result is that an individual counts as being diseased when it performs one (or more) of the relevant functions sufficiently below the statistical norm of the appropriate reference class on typical occasions. What must be stressed, then, is that an organism's biological fitness is relative to the fitness of others—there is no notion of “intrinsic fitness” at work. That is, “intrinsic fitness” as opposed to comparative fitness (contrast the use of intrinsic in Chapter 1, Section 3.2 above, for a rather different use of “intrinsic”). This means that the BST does not understand relevant functions to be healthy or diseased simpliciter. Boorse has most recently offered the following system of formal definitions (the definitions are taken verbatim):

(D): Boorse's official definition schema:

- (1) The *reference class* is a natural class of organisms of uniform functional design, specifically, an age group of a sex of a species.
- (2) A *normal function* of a part or process within members of the reference class is a statistically typical contribution by it to their *individual* survival and reproduction.
- (3) A *disease* is a type of internal state which is either an impairment of

Science 61(2): 241–264.

⁴ Boorse (1997), *op cit.*, p. 23.

normal functional ability, i.e. a reduction of one or more functional abilities below typical efficiency, or a limitation on functional ability caused by environmental agents.

(4) *Health* is the absence of disease.⁵

Careful reflection reveals that this definition is by no means as transparent as it might have been. Moreover, as we will see below and in more detail in Chapters 5 and 6, there are elements to his theory that, though hinted at in the above schema, are not fully spelled out despite being central to his account—in particular the notion of “normal function” seems to be important.

In view of this it will, I think, be useful to make some very preliminary comments about the wording of Boorse’s definitions (i.e., what I will henceforth simply call: (D)) and then provide a rather much more explicit and detailed schema of definitions than that in (D). Without denying that my comments, at times, will be interpretative in nature, they are, for the most part, culled from Boorse’s various papers. Since I will not here try to justify my interpretations, I will merely say that I hope that in the discussions to come my justifications of my interpretations will become clear.

Here my aim is just to set out what I take to be Boorse’s main views in as clear and as sympathetic a way as I can. The wordings of some of my definitions may be a bit debatable in places and may in places leave some room for further clarification, but I do not think that this will affect the force of my critique of his ideas later in this chapter and again in Chapters 5 and 6. My definitions, I think, offer a sympathetic interpretation of Boorse’s ideas in (D).

⁵ Ibid., pp. 7-8.

Firstly, the very general comments: In (D) above, and in Boorse's usage quite generally, it seems that (a) a reference class is a class of *organisms*, i.e., the members of the reference class are organisms; (b) when Boorse talks in (D2) of a "function of a part or process *within* members of [a] reference class" he means parts of organisms or processes which organisms (or their parts down to the organelle level) engage in or which they could *ceteris paribus* engage in; (c) a (natural) function for Boorse is, given (D2), something which some part or process of an organism engages in or *ceteris paribus* could engage in. Now, assuming that these comments are accurate, here is a suggested expansion and explication of Boorse's theory.⁶ The suggestion is that Boorse's account of the BST in effect comes to the following:

(D*): The amplified version of Boorse's official definition schema:

(1*) For most species the *reference classes* for that species will be those subsets of the members of the species that have *uniform* "functional design".

In other words, the various subsets are differentiated one from another depending on the differences and similarities in their "functional design". Boorse says that reference classes must be distinguished by age (infants versus young adult versus adults, etc.,) and sex, and perhaps by race.⁷

The next step is to define "functional design":

(2*)(i) The *functional design* of the members of some reference class for a species S is that "internal functional organization" (of a physiological structure) of those members of that reference class which is (a) *statistically typical* of the members of that reference class and (b)

⁶ This particular way of amplifying Boorse's theory I owe to John A. Baker.

⁷ After proposing (D), Boorse concedes that it may be appropriate to further "subclassify by race". See Ibid., p. 8. And also Boorse (1987), *op cit.*, p. 370 & (1977), *op cit.*, p. 558.

statistically typical of the contributions that the internal functional organization in question makes to the survival and reproduction success of the *individual* members of the reference class, and

- (ii) Some function is the *normal function* of some internal physiological structure if and only if performance of that function is *statistically typical* in the ways (a) and (b).

That is, firstly, the functional design of an organism will be the way in which the *physiological structures* of the organism are organized relative to the performance of some function. Secondly, when Boorse and I when discussing Boorse speak of some internal functional organization as *functioning* we are speaking of it as *making* this contribution. And thirdly, when in this context we speak of the *function* of some physiological structure we mean what that structure does do or *ceteris paribus* could do (by what of making a contribution, according to Boorse, to the survival and reproductive success of the organism).

The next step makes explicit that what is to be counted as being a *normal* functional design (or perhaps normal function) is to be defined in terms of what is statistically typical in the ways mentioned in (2*).

- (3*) The *internal functional organization* (the functional design) of the members of some species S is *normal* if and only if it counts as being *statistically typical* in the ways mentioned (2*); and (b) an organism is *functioning normally* in respect to some aspect of its internal functional organization (its functional design) if and only if that organisms functioning is *statistically typical* in the ways mentioned in (2*).

The next clauses provide the definitions of functional *ability* and of *level* of functional ability:

- (4*) The *functional ability* of the internal functional organization (functional design) of some particular member of a species S is the ability that member's internal functional organization (functional design) to contribute to the survival and reproduction success of that individual.

(5*) The *level* of the functional ability of the internal functional organization (functional design) of some particular member of a species S is a measure of the efficiency of that member's internal functional organization's (functional design's) contribution to the survival and reproduction success of that *individual* member *as compared to efficiency of contribution of the functional design of statistically typical members of the relevant reference class* as defined in (1*).

We now turn to the task of using the above definitions to craft a definition of disease which makes perhaps clearer what Boorse had in mind in his rather brief and somewhat unclear (D3).

In my view, Boorse's core idea is that a disease is a state in which the level of the functional ability of the internal functional organization of an organism is, as he puts it, "sub-normal". That is, the internal functional organization of the organism's contribution to the survival and reproductive success of the organism is below that of the statistically typical member of the reference class for the species. Boorse mentions a couple of factors that might cause such sub-normal performance, but I think it is methodologically clearer if we set those factors aside⁸ and state the core of his definition thus:

(6*) A *disease* state of an individual member of a species is a state of the internal functional organization of that individual in which the functional ability of that internal functional organization to make a contribution to the survival and reproductive success of the individual is at a level below what is normal (i.e., statistically typical) for the relevant members of that species.

He then defines *health* as follows:

(7*) *Health* is the absence of disease.

⁸ One should note that Boorse explicitly concedes that "it is unclear that my environmental injury clause was ever worth the trouble". See Boorse (1997), *op cit.*, p. 86. I'm inclined to agree that it's not worth the trouble since whatever state the "environmental injury clause" would capture would also surely be captured by the first part of the disjunction as stated in (D2) (i.e., an internal state which is an impairment of normal functional ability).

So with this in mind, the purpose of this chapter is to argue that, despite the strengths of Boorse's account as a *naturalist* and hence *empirical* and possibly *value-free* and *norm-independent* conception of disease and health, at a deeper level the BST, in general, and in particular his account of functional ability and level of functional ability in clauses (4*) and (5*) (i.e., biological fitness, as some would call it) face some serious challenges. As they stand, I argue that these challenges fatally undermine the BST's candidacy for the rôle that Boorse has cast it to play, namely, to underpin medicine with a theoretical, value-free science of health and disease.

In particular, I argue (in Section 2) that the current framework of the BST essentially involves the use of what I will call a "Cambridge-change" criterion and as such is problematic for the reasons I will explicate later. Following the literature, I describe "mere Cambridge changes" and "real" changes in terms of the difference between those of an individual's properties that are relational and those that are intrinsic properties of that individual. The main point will be that a Cambridge-change objection presents itself because statistical norms of the BST's reference classes will not remain static; some will undergo changes—and these changes can be imagined without any corresponding change in the "internal functional organization" of the organism, despite the fact that by (D4) of Boorse's definition schema disease and health are said to be states of the "internal functional organizations" of the organism (or as Boorse puts it in (D3) "internal states" of the organism).

In Section 3, I will spell out in some detail how this is so, exploring two problems that I contend arise from the fact that the BST is to be taken as advancing a Cambridge-change criterion for health and disease. This criterion, I will argue, commits the BST to

the troubling view that, as I just said, an individual could go from being diseased to healthy, or vice versa, without any “internal” physiological change (internal functional organization) in that individual. What appears is that such a view presents the BST with two profound, perhaps insurmountable, problems: (1) it is ill-equipped to formally embrace Cambridge-changes and (2) it is in part as a consequence theoretically dubious.

As a first step in my examination of Boorse’s suggested account I need to review in a little more detail the role which Boorse has indicated he wants his definition to play out in practice, that is, I will spell out in a little more detail how Boorse intends the schema set out in D (and I submit my amplified D*) above to be interpreted.

2. The Biostatistical Theory (BST)

Firstly, it is clear from (D) and (D*) above that Boorse firmly insists that the only biological mechanisms (the “internal functional organization” of an organism) relevant to the determination of health and disease are those that contribute to the survival and the reproductive success of the individual organism.⁹ Boorse, most importantly, requires that the relevant contribution of the relevant internal functional organization be the “*actual* contribution to [the relevant] goal”.¹⁰ So unlike some other conceptions of natural

⁹ Boorse insists that his choice of goals is not normative: “The fact is that human physiologists have as yet found no functions clearly serving species survival rather than individual survival and reproduction” (1997), *op cit.*, p. 28. See also Boorse (1977), *op cit.*, p. 556; (1997), *op cit.*, pp. 9 & 25; and (2002), *op cit.*, pp. 64, 69 & 76.

¹⁰ Boorse (1997), *op cit.*, p. 66; my emphasis. See also Boorse (1976), *op cit.*, p. 80 where he defines his goal-directed theory of biological functions: X is performing the function Z in the G-ing of S at *t*, means at *t*, X is Z-ing and the Z-ing of X is making a causal contribution to the goal G of the goal directed system S. I return to discuss Boorse’s goal-directed account of biological functions in greater detail in Chapter Six.

functions,¹¹ Boorse employs a conception that is solely concerned with the actual or present contribution the relevant internal functional organization may make at the time of the health/disease ascription and not the role they may once, in the past, have served.

Secondly, for Boorse the BST includes psychology within the domain of biology: “[t]he BST does insist that all genuine disease or illness must involve biological dysfunction, on the broad view of biology as including psychology”.¹²

With this in mind, we may summarize Boorse’s account as I did above: a state of an organism counts as a disease-state when the level of the functional ability of one of the relevant internal functional structures falls below the statistical norm of the same species reference class on species-typical occasions.¹³ And because Boorse stipulates that health is the absence of disease, it would seem he commits the BST to the following positive definition of health: an individual is healthy if and only if *all* the functions that contribute to the species member’s survival and reproduction *today* are capable of performing in a way that is species-typical (i.e., the statistical norm of the relevant functions of the same

¹¹ Like, for example, Wakefield’s (1992), *op cit.*, evolutionary account of natural function or the etiological function theories respectively championed by: Wright; Millikan; & Neander. See Larry Wright (1973), “Functions”, *The Philosophical Review* 82(2): 139-168; Ruth Garrett Millikan (1984), *Language, thought, and other biological categories: New foundations for realism*. Cambridge, Mass: MIT Press; & Karen Neander (1991), “Functions as selected effects: The conceptual analyst’s defense”, *Philosophy of Science* 58(2): 168-184.

¹² Boorse (1997), *op cit.*, p. 98.

¹³ Two points need to be made clear: Firstly, the requisite sub-normal functioning may also occur when, strictly speaking, one of the relevant functions is performing at a level abnormally above the statistical norm such that the level of functioning would place the individual’s survival and/or reproduction at risk. As Boorse rightly states: “...Now the most obvious logical feature of medical normality is that most functions have a normal range of values...there is a normal range of values around a mean, with either one or two pathological tails” (2002), p. 101. See also Boorse (1977), p. 564 and (1987), p. 371. And secondly, when it comes to environmental causes, Boorse allows for sub-normal functioning to be species-typical functioning. See Boorse (1975), p. 65 and (1976a), p. 79 & (1997), pp. 83-83.

species, sex and age at time t) on species-typical occasions.¹⁴

There are two points worth explicitly noting: First, health and disease seem to be collectively exhaustive and mutually exclusive states. That is to say, an individual is either healthy or he is diseased and no individual is ever both healthy and diseased. Second, notice that the BST's demarcation of health and disease turns on the biological and statistical normality of the relevant functions of the appropriate reference class.

2.1 *Biological normality and the BST*

If Boorse's definitions of health and disease are to be naturalistic then clearly he will need a conception of normality that is not only ontologically objective (by O*) but also semantically so (by S*), and crucially a conception for which a naturalistic account can be given. It is important to notice further that insofar as Boorse wants a conception of normality that is "empirically based", and he does, it would seem to follow that he also wants a conception of health and disease that will be epistemologically objective (by E*). Boorse, of course, does not use this terminology nor does he seem to be sensitive to these distinctions.

I should point out from the outset that Boorse's purportedly naturalistic conception of normality appears to turn on the usability of several highly problematic concepts and conceptions. Indeed, it would be implausible to deny that there is a heated debate within the philosophy of biology community about the concepts of *species design, function,*

¹⁴ This would appear to be very much in line with a previous positive account of health Boorse explicitly outlined: "*Health* in a member of the reference class is *normal functional ability*: the readiness of each internal part to perform all its normal functions on typical occasions with at least typical efficiency" Boorse, (1977), p. 555.

individual survival and reproduction. Thus it has become a matter of significant controversy whether the biological concepts and conceptions that Boorse draws upon are indeed entirely empirical and, moreover, are non-normative concepts and conceptions.¹⁵

And putting this point in terms of the concepts and terminology developed in Chapters 1 and 2 above, it is not at all clear that the biological concepts and conceptions that Boorse draws on are semantically and ontologically objective conceptions, let alone entirely epistemologically objective concepts. However the extent to which, if at all, biological function in particular, and biology (and other “hard” sciences?) in general, are not objective and non-normative is a matter that clearly transcends the scope of this chapter. Thus I shall leave it an open question whether, in fact, Boorse employs a notion of biological function that is at root crucially non-normative, let alone objective in each of the three conceptions I identified in Chapter 1. However, that being said, at the end of the day all that Boorse surely requires is for the BST to be no less objective and no less empirical and no more normative than biology and physiology (read: medical science?).¹⁶

¹⁵ See, for example, W. Miller Brown (1985), "On defining disease", *Journal of Medicine and Philosophy* 10(4): 311–328; H. Tristram Engelhardt, Jr (1976), "Ideology and etiology", *Journal of Medicine and Philosophy* 1(3): 256–268 & (1986), *The foundations of bioethics*. New York: Oxford University Press; Fulford (1989), *op cit.*; Boorse (1997), *op cit.*, responds directly to these criticisms. For some more recent criticisms see Amundson (2000), *op cit.*; William E. Stempsey (2000), "A pathological view of disease", *Theoretical Medicine* 21: 321–330; Nordenfelt (2001), *op cit.*, & (2004), *op cit.*; Rachel Cooper (2002), "Disease", *Studies in History and Philosophy of Biological and Biomedical Sciences* 33(2): 263–282. Elselijn Kingma (2007), "What is it to be healthy?", *Analysis* 67: 128–13 & (2010), *op cit.*; and Ereshefsky (2009a), *op cit.*

¹⁶ Of course, that being said, Boorse is quite clear that he views biology and physiology as value-free: “If health and disease are only as value-laden as astrophysics and inorganic chemistry, I am content. I admit having no sympathy for the view that scientific concepts or knowledge is evaluative. Obviously, we do science, as we do everything, for evaluative reasons. But I do not see why our motives for information-gathering must infect the information gathered, injecting values into science, mathematics, and the Bell telephone directory. However, I leave defending the value-freedom of physics to physicists and philosophers thereof. If the BST shows that health in medicine is as objective as physics, it achieves everything I ever dreamt of for it” Boorse (1997), p. 56, & see also p. 75.

In this chapter I will leave this question on one side because I wish to draw attention to what I take to be a stronger and more radical tack against Boorse: That the BST essentially involves what counts to be a *Cambridge-change* criterion—a criterion that as such renders the BST inadequate to serve its own purposes. Or so I shall argue.

2.2 *Statistical normality and “Cambridge-changes”*

It is his conception of normality defined in terms of what is statistically typical that Boorse hopes will serve as the machinery by which the BST will forge a non-normative, (i.e., what in my terminology I would call an objective) account of “normal” functioning—it is the standard against which the BST ultimately determines an individual to be either diseased or healthy. Recall the canon of the BST: An individual will count as diseased when, roughly put, the functioning of one of her relevant internal functional organizational structures fails to be in accord with the statistical norm of the same species reference class at time *t*.

With this in mind, in application, the BST presumably will determine whether an individual is healthy by comparing the current performance level of the individual’s relevant functions (i.e., the internal functional organizational structures that contribute to survival and reproductive success) against the appropriate statistical norms of the individual’s reference class (see clauses (5*) and (6*) of (D*)). To count as being healthy, it must be the case that all of the relevant functions are found to perform at a level, given typical circumstances, that is counted as being statistically typical; when this is not the case, the individual will count as being diseased.

So the upshot of the decision to define normal functioning in terms of statistical

normality is supposed to be that this conception of “normality” will make normality an objectively determinable, i.e., purely empirical, conception and crucially allow for a naturalist definition of “normal functioning”. As an aside, statistical normality is most often defined as an average or arithmetic mean though it is important to note that there is no obvious reason that precludes viewing “statistical average” as a median or a mode.¹⁷ In any case, the point to press is what is statistically normal may fluctuate given changes in the data drawn from the organisms that compose the reference class. For example, suppose the capacity to circulate blood increased to Lance Armstrong-like levels for most humans and that the normal capacity of the human heart to circulate blood was stipulated to be the mean capacity to circulate blood. One could expect the result, *ceteris paribus*, to be that the statistically normal capacity for the human heart to pump blood would increase accordingly.

Notice here that a surprising result seems to follow: that the BST, in general, and statistical normality, in particular, facilitates what I have said are called “Cambridge changes”.¹⁸ To understand why Cambridge changes are problematic for Boorse one needs only to realize that a Cambridge change property is a property which an individual may acquire or lose not as a function of changes in the “intrinsic” properties of the individual

¹⁷ Boorse views statistical normality as an arithmetic mean. See footnote 13 above.

¹⁸ I am indebted to John A. Baker who, in conversation, brought to my attention Geach’s writings on Cambridge changes. Peter Geach (1969), *God and the soul*. London: Routledge & Kegan Paul, at pp. 71-72 distinguishes between ‘real’ and ‘mere Cambridge changes’. Geach identifies a Cambridge change to occur when a proposition about an object changes in truth-value without any *real* change in the individual object. To illustrate: if it were true that Socrates were to grow in height such that he is now taller than Theatetus then Socrates—because he physically increases in stature—undergoes a ‘real’ change; however, Theatetus—because he undergoes a change only in relation to Socrates (i.e., he is *now* shorter than Socrates—undergoes a ‘Cambridge change’. See also Peter Geach (1972), *Logic matters*. Berkeley: University of California Press, at pp. 318-27.

itself but as a function of change in *other* individuals.

To illustrate, let us return to the above example about the capacity of the heart to pump blood, and let us suppose that the statistically normal capacity for the human heart to pump blood did, in fact, significantly decrease.¹⁹ Suppose further that Socrates' heart *before* the change was *less* efficient than the average but that there was no change to Socrates' heart efficiency at the time of the change in the average—that is to say the capacity of Socrates' heart to circulate blood remained exactly the same. On these suppositions, given, *ex hypothesi*, the decrease in the capacity of the members counted to be in the relevant reference class to circulate blood, the ability of Socrates' heart to pump blood is now no longer functioning below the relevant statistical normal capacity for a heart to pump blood—it is now functioning at an average level of capacity.

What is to be stressed is while there is no “real”, no “intrinsic”, no “internal”, change to the functioning of the heart of Socrates, because his heart *now* functions no less efficiently than what is counted to be normal functioning for a human heart (i.e., Socrates undergoes a Cambridge change), he now has a statistically normal functioning heart and, *a fortiori*, on the BST Socrates' heart would now count as no longer being diseased. It would thus count as being healthy! All of this should, surely, be a source of concern for those who espouse the BST conception of health and disease. Let us look more closely at what is taking place in the generation of this worry.

What my presentation of the above example has brought to light is that there are in

¹⁹ To further motivate this scenario it may be useful to add some details here: suppose that obesity levels, in general, and hypertension levels, in particular, were to rise such that the statistical norm of the capacity of the heart to pump blood for Socrates' reference class decreased.

fact at least two different kinds of scenario in which one kind or other of a “change” would result in a change in the disease-status of an individual if we use the BST account of health and disease. One of which Boorse acknowledges, but the other of which he did not seem to notice, let alone acknowledge.²⁰

(A) *The class of scenarios which Boorse did acknowledge*: when there is physiological change in the individual under examination such that although at time t the performance of at least one of an individual’s relevant functions (i.e., those that contribute to the individual’s survival and reproduction today) was properly assessed as being statistically subnormal (i.e., a relevant function now fails to perform in accord with the statistical norm for the appropriate reference class), at some different time t' , because of a change in the individual’s capacities, it is properly assessed at time t' as performing at the statistically normal level of performance. Or vice versa.

(B) *The class of scenarios which Boorse did not acknowledge*: when there is *no* physiological change in the individual but there is a statistical change in the norm of a relevant function for a reference class such that at least one of an individual’s relevant functions would be properly counted at time t to be performing at a “subnormal” level when it was previously not or vice versa.

And if this is correct then it seems we must acknowledge that, given the current

²⁰ To be clear, I am not claiming the BST requires that a “change” occur in either the individual or the reference class to give disease-status. As an anonymous referee for *Studies in History and Philosophy of Biological and Biomedical Sciences* pointed out, with congenital pathology, an individual has always had biological dysfunction. In such cases, disease-status would not require a change. Equally clear, however, is that the BST would not correctly count these individuals to be “healthy” such that a change in disease-status would be warranted.

framework of the BST, there is an implicit Cambridge-change criterion that essentially flips the direction of fit between an individual and the relevant statistical norms of others. That is to say, there are two directions of fit and as such disease status will turn on (A) a physical or “real” change in the individual but also on (B) what I have suggested is a “mere Cambridge change” to(?) the individual where there is no physiological change in the individual to speak of—the change is with the level of functioning of other people’s relevant functions.

3. On the BST advancing a Cambridge-change criterion

3.1 Dynamic statistical norms

In order to further advance my argument, it is important to make clear that the statistical norms the BST employs will be dynamic for at least two reasons. First, as an individual ages different reference classes with crucially different statistical norms will become relevant for her. This enables the BST to distinguish between “normal” aging and disease, which is imperative because Boorse explicitly states that “normal” aging cannot be a disease.²¹ Thus there is the need to ensure that the criterion for health and disease reflect the fact that the performance levels of many of our typical physiological functioning not only will increase as we age (e.g., a typical newborn is unable to walk, talk and procreate) but will also decline as we age, which Boorse of course does in (D). If the BST is going to account for healthy babies and healthy seniors then it is imperative it draws upon age-related reference classes with crucially different statistical norms. Accordingly, the

²¹ Boorse (1997), p. 90.

statistical norms for an individual will change as he or she ages and, as such, different reference classes become relevant.²²

Second, the BST, after all, employs a conception of “normal functioning” that is concerned with “how the mechanism currently operates”²³ or, more accurately, “the actual contribution to a goal”²⁴ and not with the contribution something *may have* made but *now no longer does* make (and presumably vice versa). Hence, the relevant statistical norms may change as the various individuals who presently compose the various reference classes change individually or as the membership of the reference class changes. The extent to which the requisite statistical norms will change depends, of course, on how much there is a change in the relevant states of those who are members and/or there is a change in either the membership or the size of the reference class.

The point is that individual members and reference classes do, in fact, change. In the past century, there has been a significant increase in the average height, weight and life expectancy (to name only three) globally and amongst particular populations. A significant increase, for example, in gross obesity rates amongst the oldest female seniors—a reference class composed of few members—would surely have a substantial impact on relevant statistical norms for this age group. This demonstrates another reason statistical norms will be dynamic.

3.2 *Important implications*

²² For many years, Boorse has stressed the need to make medical normality relative to age. See, for instance, Boorse (1977), pp. 555, 558, 562 & 587; (1987), pp. 370-371; and (2002), p. 90.

²³ Boorse (1976b), p. 85.

The extent to which dynamic statistical norms actually do fluctuate clearly has important implications for the BST, for Boorse wants to insist, that “the classification of human states as healthy or diseased is an objective matter, to be read off the biological facts of nature without the need of value judgements”.²⁵ It is worth noting a related claim Boorse makes:

We have supposed that the basic notion is ‘X is a healthy Y’—that is by comparing X with its reference class Y that one distinguishes the way X does function from the way that it ought to.²⁶

So what the BST counts to be proper functioning clearly depends upon the relevant biological facts of the reference class. And because of this, changes in the statistical norms of the relevant biological facts of the reference class may result in X no longer being counted as a healthy (or unhealthy) Y. An important implication indeed.

3.3 Impacting the theoretical normality of health (and disease)

What is much less clear is the extent to which dynamic statistical norms may be said to actually impact what the BST must count to be the theoretical normality of absence of disease (i.e., health) for the various relevant functions. At least part of the problem is that what is species-typical incorporates Boorse’s conception of “a reasonable time-slice of a species” which is, as he himself explicitly concedes, “vague”.²⁷ Nevertheless, Boorse does insist, “some of the past affects what is species-typical” and speaking to this he states:

...I do see a species as extending over time as well as space, so for me some of the past affects what is species-typical. I do not take sudden or temporary

²⁴ Boorse (1997), p. 66.

²⁵ Ibid., p. 4.

²⁶ Boorse (1977), p. 562.

²⁷ Boorse (1997), p. 66.

changes in lifestyle, even if worldwide, as changes in the nature of the species. For example, if tomorrow all human beings suddenly began to live wholly inside buildings, I would not immediately say that human skin had lost its function of synthesizing vitamin D in sunlight. If the whole earth went pitch black for two days, I would not say that eyes had lost its function in the human species.²⁸

One might take this explanation to imply that Boorse requires dynamic statistical norms to reflect a reasonable time-slice of a species that is a longer than one or two days and perhaps even much longer. Though in a later work Boorse says that, “the concept of the species-typical is not overly historical”,²⁹ it is, however, neither clear nor obvious that the statistical norms for the BST’s reference classes cannot change in one day.

To see this, let us distinguish between two different issues at stake here. One is the issue of *which biological processes* are “normal” functions and, the other, *which level of functioning* is requisite for a goal of a normal function to be successfully realized³⁰—see clauses (2*ii) and (5*) of (D*). In order to avoid confusing these two importantly distinct issues, I will draw upon some terminology used by Elselijn Kingma and refer to the first issue as the “qualitative issue” about functions and the second as the “quantitative issue” about functions.³¹ With this distinction in mind, what will become clear is that even if Boorse may resist immediately saying that human eyes have lost their function (i.e., the

²⁸ Ibid.

²⁹ Boorse (2002), p. 86. See Chapter 6, Section 2.2.1 for a much fuller examination of what Boorse says regarding the requisite “time-slice” of a species.

³⁰ I am thankful to an anonymous referee at *Studies in History and Philosophy of Biological and Biomedical Sciences* for suggesting this particular distinction.

³¹ Elselijn Kingma (2010), *op cit*. This is not the only way this distinction has been drawn. Alvin Plantinga, for instance, distinguishes between a naturalistic analysis of *function* and a naturalistic analysis of *proper function*. I prefer Kingma’s terms to mark the distinction if only because the term ‘proper function’ is so often associated with Millikan’s highly influential notion of *proper function*, which she intends to be a technical term. See Alvin Plantinga (1993), *Warrant and proper function*. New York: Oxford University Press, especially Chapter 11.

solution to qualitative issues about normal function) in the human species, it therefore doesn't follow that the *level* of functioning (i.e., the solution to quantitative issues about normal function) cannot significantly and rapidly change. The implication will be that the relevant statistical norms for the reference classes may change in one day.

Let me spell out the above point in a bit more detail. Consider Boorse's example of the whole earth going pitch black for two days. Boorse seems correct to insist that the human eyes had not lost their function if the reason(s) the earth becomes no longer visible (read: goes pitch black) to humans cannot be attributed to a failure (qualitative) in the biological processes of the organisms. Perhaps here it will be helpful to imagine something like the mass unexplained epidemic of blindness that occurs in Nobel Prize winner Jose Saramago's *Blindness* (imagine it as affecting everyone one earth instead of merely those in one city as in Saramago's novel). If the earth went black because of an internal failure of function (qualitative) in human eyes, then surely the BST will have to insist that human eyes have lost their function; after all, our eyes can no longer actually see and, what is more, the eyes have universally and suddenly lost their "*normal functional ability*: the readiness of each internal part to perform all its normal functions on typical occasions with at least typical efficiency".³²

Here it seems extremely implausible to suggest that we can square (a) the suggestion that the human eyes had not lost their function in the human species even though everyone had become blind and (b) Boorse's demands (i) that we say a function of something *x* is to be the actual contribution *x* makes to some goal; (ii) Boorse's denial that there are genetic

³² Boorse (1977), p. 555 and Boorse (1997), p. 8; italics mine.

diseases that can be classed as universal;³³ and (iii) his contention that “fitness reducing effects cannot be functions”,³⁴ i.e., that if some effect reduces fitness then it cannot be classed as a function of the organism whose fitness is thus reduced.

In my view, there is a more plausible response that enables Boorse to resist saying that the human eyes had lost their function (qualitative) in the human species. Such a response, we shall see, comes at significant price. Suppose one were to offer the following sort of reply: In the above scenario, the failure to see as “normal” is not because the human eyes have lost their function but, rather, strictly speaking it is because the typical human eye is not able to see in the pitch dark. What then might be said here is that the fact that the human eyes no longer see is not due to a qualitative failure of function—the biological processes of typical human eyes are simply not able to see in pitch blackness—but only a quantitative failure in the level of functioning of the human eyes. The upshot of this response is that it squares tightly with Boorse’s view of function since it does seem correct to insist that there was no loss of the readiness of the internal processes of the human eye to perform its normal functional ability during the occasion the whole world went pitch black for two days.³⁵ In sum, I think Boorse is able to resist saying that eyes had lost its function (qualitative) in the human species because the typical human eye is not able to see in the pitch dark.

But it should be noted that if this explanation seems sufficient it is because Boorse

³³ Boorse (1977), p. 567.

³⁴ Boorse (2002), p. 85.

³⁵ Similar reasoning could account for why Boorse “would not immediately say that human skin had lost its function of synthesizing vitamin D in sunlight”.

stipulates that it was only for two days that the whole earth went pitch black.

Presumably on day three, light returned to our planet and with it our sight. The return of our sight provides very good reason to think it was the environment that changed and not, strictly speaking, the biological processes of the human eye. This is what makes it plausible to insist, for those of us with sight prior to the darkness striking, that there was no loss in the readiness of our eyes to perform its function (qualitative) in the human species.

Notice, however, it is only in hindsight, after our sight has returned, that one would be able to insist as much. For on day one or even day two of the darkness Boorse could not be sure either that (i) our sight would return or (ii) there was no loss of the readiness of the internal processes of the human eye to perform to its normal functional ability on even typical occasions. To illustrate, suppose that just as you finish reading this sentence the whole earth goes pitch black such that you cannot see anything. One would very quickly realize that the actual ability of one's eyes to function (quantitative) has significantly decreased. But at this time could one know that one's blindness would only be temporary and that both light would return and our eyes would react as before? Surely not; this simply cannot be read off from the biological facts of nature.

Here one might therefore wonder on what grounds Boorse could resist saying that the eyes had lost its function (qualitative) in the human species given our present universal blindness. The concern here is that, recall, Boorse requires a function to be an actual contribution to a goal. Add to this that one cannot be sure of either (i) or (ii) and it appears the environment has, so to speak, rendered our eyes useless. If so, one would expect the BST to view the eyes, as it does the appendix, as no longer functional. At any rate, it is

certainly not clear why the BST must resist such a view given Boorse that states:

An organ X once established by selection pressure deriving from its effect Z may cease to be functional, as did the appendix, if a change in the rest of the organism or in the environment renders Z useless.³⁶

It seems to me that Boorse could temporarily resist such a view by stipulating that the appropriate time frame between the environment having rendered the human eye's ability to see useless and the BST declaring the human eye has ceased to be functional (qualitative) will obviously be to some extent arbitrary. Yet it will not be completely arbitrary because minimally the BST will have to contend with an empirical analysis of the biological facts of nature showing that the level of function (quantitative) of the eyes has notably decreased. Thus, so long as Boorse demands that a function be an actual contribution to a goal then our persisting universal blindness will make it increasingly harder to plausibly resist saying that the biological mechanisms of the human eye have ceased to be functional (a point about the quantitative analysis of function).

Correspondingly, so long as all humans continue to remain wholly indoors and unexposed to sunlight, if not immediately, then at some point Boorse is going to have to say that the human skin has lost the actual function (a point about the qualitative analysis of function) of synthesizing vitamin D in sunlight.

But none of the above precludes dynamic statistical norms from rapidly changing. This is because significant changes to the statistical norms may occur regardless of when the BST ultimately deems there to be a loss of a qualitative function in, to continue with the above example, the human eyes. Indeed ahead of time we should expect that the

³⁶ Boorse (1976b), p. 76.

universal and sudden poor performance (a quantitative comment about function) of human eyes to significantly and rapidly lower the relevant statistical norms for some, if not all, the reference classes, perhaps even in one day. And this will especially be the case in reference classes composed of few members (e.g., the oldest female seniors). The recognition of these lower statistical norms presumably would then require the BST to cease to count many people's eyes to be functioning sub-normally and, as such, diseased.

And at the point when the BST does deem that enabling sight is no longer a current function (qualitative) of the human eyes then when it comes to health and disease it will no longer be considered a relevant function. Therefore, the poor performance of many people's eyes—that were once considered to be functioning sub-normally—will suddenly no longer be a relevant consideration. The BST will then be required to cease to count many individuals to be diseased—individuals that it would have counted to be diseased a day prior (when the mechanisms of the human eye were still a relevant function for health and disease). Notice that in both instances there need be no relevant change in the internal functional ability of a particular individual's biological processes to warrant a change in disease-status— a sufficient change in the statistical norms of the reference class will suffice. And this is a change that may occur rapidly.

One may therefore wonder what exactly follows from having to acknowledge, as it seems we must, that the leading naturalist conception of health and disease essentially involves the use of a criterion which turns out to be a Cambridge change criterion; but at least this much seems clear: (I) an individual could, for example, be correctly counted to be disease-free (i.e., healthy) yesterday and with no diagnostic change in the individual's functional ability come to be correctly (on the criterion) counted as being diseased today;

and (II) an individual could, for example, be correctly counted as being diseased yesterday and with no diagnostic change in the individual's functional ability come to be correctly counted as being disease-free today. In what is to follow, I shall try to show that these two implications travel with two intractable objections against the BST.

3.4 *Cambridge-changes and the BST: A closer look*

The striking feature of the BST is that it will count Socrates diseased if and only if there is subnormal biological functioning (i.e., dysfunction) which poses (presumably) at least the danger of a genuine physiological effect on his prospects of individual survival and/or reproduction.³⁷ Otherwise Boorse says, “[a]s long as the efficiency of all functions exceeds a minimum, any value of these traits is as healthy as any other”.³⁸ However, because what is to be counted as “normal functioning” is crucially tied to a statistical conception of functioning, as we have seen, a Cambridge-change criterion emerges; and, as such, the BST may correctly deem Socrates’ heart to be diseased when it functions at a level of, say, x , and later correctly deem the *exact same* level of functioning of Socrates’ heart not to merit disease-status.

3.4.1 *An “internal” tension*

Given the framework of the BST, the Cambridge-change criterion presents a serious challenge for Boorse’s analysis of health (and disease): How can the BST account for the presence of the requisite genuine physiological interference given the fact that (i) Socrates’

³⁷ Recall the BST stipulates that disease status requires at least one biological function relevant to the goals of individual survival and/or reproduction to be in some way interfered with such that it is unable or unready to perform as “normal”. See footnote 8 as well as (D) and (D*) above.

³⁸ Boorse (1977), p. 563.

heart functions exactly as it did before and, what is more, (ii) such functioning was previously deemed to sufficiently interfere with the biological goals of either his individual survival or reproduction? In the absence of any provisions for the BST to appeal to a change in values, surely a reasonable person might legitimately wonder why the exact same functioning of Socrates' heart now no longer counts as interfering with the biological goals of Socrates' survival and/or reproduction (read: to warrant disease-status) when it previously did so count?

Clearly an acceptable answer in this case cannot account for the change in disease-status merely by claiming that there has been some measurable increase in functional ability in the individual. After all, *ex hypothesi*, there has been no physiological change in the individual. So it would seem, the BST cannot, without some revision of the account, portray itself as claiming that a change in the disease-status of an individual must involve a change in the internal biological functioning of the individual *qua* individual.

But this is just the kind of claim that Boorse appears insistently to be making—he wants to be taken as saying that for a healthy individual to acquire a disease state (i.e., a pathological condition) is to undergo an internal physiological change. Speaking to a criticism of R. M. Hare's, Boorse says:

...the BST implies that fleas and lice are not a disease state or pathological condition because they are on, not in, the organism. Flea bites are pathological; fleas aren't. They are annoying, like flies, roaches, and teenagers; their bite is a pathological condition; but in themselves they are not a pathological condition because they are not in the organism. Penetration is all.....So I shall rule on behalf of the BST that all these organisms cause a pathological condition only when they cross the organism's boundary by penetrating living tissue or a body orifice.³⁹

³⁹ Boorse (1997), p. 68.

In a related footnote Boorse adds that “[t]he internal effects of these objects will be pathological even if the objects themselves are strictly, to the physiologist, external”.⁴⁰ Furthermore, recall that in Boorse’s most recent definition of disease he stipulates that disease “is a type of internal state” (D3). So if disease is a pathological internal state then health must be the absence of a pathological state. Hence it seems that there should be some internal biological change to plausibly warrant the BST now deeming Socrates to be healthy and no longer diseased. And, given the framework of the BST, this is surely what Boorse does have in mind when he tells us that “diseases are internal states that depress a functional ability below species-typical levels”,⁴¹ and “[t]he BST does insist that all genuine disease or illness must involve biological dysfunction, on the broad view of biology as including psychology”.⁴²

Now Boorse is, to my mind, correct to insist that the biological dysfunction is in some sense to be taken to be a necessary condition of genuine disease. But such an insistence literally creates an internal tension within the terms that together go to make the BST. The problem is that, so long as the BST essentially involves what counts to be a Cambridge-change criterion, a change in the disease-status of a particular individual may, indeed apparently will, turn on changes to the statistical norms—changes that can hardly be said to necessarily require, strictly speaking, any one particular individual to undergo *internal* physiological change(s) of any kind. Thus, in his response to Hare, Boorse cannot reasonably demand that fleas cause a pathological condition in, say, Socrates *only when*

⁴⁰ Ibid., p. 119.

⁴¹ Boorse (1977), p. 542.

⁴² Boorse (1997), p. 98.

they penetrate Socrates' living tissue or body orifice. Hence, not only is it questionable that Boorse's ruling on behalf of the BST "fits medical usage well enough"⁴³ but there is reason good reason to think the current framework of the BST, with its internal tension, is ill-equipped to formally embrace Cambridge-changes.

3.4.2 *Medical thought and practice*

The above brings to light the important issue of whether the possibility that the BST is committed to a criteria that make health and disease changes Cambridge-changes will, in effect, derail the BST from medical thought and/or practice. Any divergence should be taken very seriously since Boorse insists that the aim of the BST was to "target...the medical concept of health".⁴⁴ And what is more, Boorse states that "[t]he real threat to the BST is, of course, cases where it and medical usage clearly divide".⁴⁵ On the one hand it seems to me that the BST will notably track medical thought and practice. At any rate, if the BST is to fulfill its goals then this is going to have to be the case.

So, returning to the above scenario involving Socrates, let us say the BST properly counts Socrates to be diseased. We have seen Boorse explicitly state that (i) when it comes to health and disease, relevant functions are only those that contribute to individual survival and reproduction today; (ii) biological dysfunction is a necessary condition of disease; and (iii) health is the absence of disease. So, presumably, on (i)-(iii), Socrates will be properly deemed to be "healthy" (*i.e.*, disease-free) only if the requisite biological dysfunction, that warranted disease-status in the first place, will have dissipated enough to

⁴³ Ibid., p. 68.

⁴⁴ Ibid., p. 42.

⁴⁵ Ibid., p. 19.

reasonably contend that the current level of functioning no longer makes survival and reproduction of the individual less likely.⁴⁶ Insofar as medical usage speaks of a disease as having a cure would seem to reinforce this contention. That the BST demands that the absence of health is “simply species-subnormal part function”⁴⁷ is not then, per se, worrisome.

What is worrisome, on the other hand, is that so long as all of Socrates’ relevant biological mechanisms are counted to be functioning at a species-normal level, then the BST will have to deem Socrates to be healthy. The problem with this is if we take the BST seriously then it appears there will be Cambridge-change instances where a diseased individual, with no change in the individual’s functional ability, will have to be counted to be healthy simply on account of reduced statistical norms. Thus, in a Cambridge-change instance, one might wonder if medical thought and practice should also consider Socrates to be healthy. That it clearly cannot be the case that a biological dysfunction of Socrates’ has been ameliorated or, *a fortiori*, that a disease-state in the individual has been cured hardly gives one confidence in an affirmative answer.

Furthermore, Boorse seems to agree given that he says, “what we normally mean by curing a condition is removing it, not engineering its reclassification”.⁴⁸ And so given the fact that one’s eyes will never be able to see what one looks at more clearly, that one’s heart will never be able to pump more of one’s blood, and indeed that one’s life will never

⁴⁶ Boorse explicitly states that “To say that physiological functions are contributions to individual survival and reproduction is not to say that their failure will be fatal in any particular case...But the required contribution of a trait need only make its bearers more likely to survive than nonbearers. Nothing follows about the survival of any individual nonbearer...The most it could imply would be that diseases make people marginally less likely to leave descendants” Boorse (1977), p. 561.

be saved simply by appealing to reduced statistical norms should, it seems to me, motivate the notion that it is wrongheaded for medical thought and practice to outright wed itself to the view that what essentially matters for health (and disease) is that an individual's relevant functions conform to (diverge from) the statistical norms of the appropriate reference class. Otherwise, it seems that we are committed to saying that medicine can "cure" a diseased individual, not by removing any pathological condition but by reclassification.

Here I think it would be wise to heed Boorse's words that "[t]he main thing is to avoid false presumptions caused by calling something a disease (e.g., masturbation) which lacks the biological dysfunction on which such presumptions depend".⁴⁷ But we should also avoid false presumptions caused by calling something (or someone) healthy which lacks the biological function on which such presumptions surely depend.

In light of Cambridge-changes, however, it is difficult to see how employing the BST—with its insistence that when what essentially matters for health (and disease) is that an individual's relevant functions conform to (diverge from) the statistical norms of the appropriate reference class—would not render us more susceptible to making these false presumptions. Because appealing to statistical norms alone cannot, in fact, improve (or damage) the internal functioning of a diseased individual⁵⁰ it therefore seems that if one

⁴⁷ Boorse (2002), p. 76.

⁴⁸ Ibid., p. 99.

⁴⁹ Ibid.

⁵⁰ To be clear, I do not wish to dispute the contention that it is wholly plausible to speak of an individual becoming more (or less) healthy in comparison to others. Indeed, I would agree that such thinking is useful and often carries important implications for clinical medicine and health policy decision-making, although I think such implications will surely involve normative components. What I do want to insist, however, is that the BST is mistaken to insist that what essentially matters for health (and disease) is that an individual's

truly wants to avoid making these unwanted false presumptions then medicine should resist a wholesale commitment to the BST's prescribed value-free concept of disease.⁵¹ Hence there is legitimate reason to be troubled of the way in which the BST allows for *relational* properties to make an individual diseased or healthy. That is to say, there is good reason to think, then, that as a theory of disease and *a fortiori* health, the BST is theoretically dubious. A different approach is needed.

In summary, the implicit Cambridge-change criterion brings to light two seemingly intractable problems that the BST must come to grip with. First, the current framework of the BST appears ill-equipped to formally embrace Cambridge-changes. Secondly, the BST is beginning to look theoretically dubious. In what follows, I shall refer to these two problems as the “Cambridge-change objections”.

4. Concluding remarks

With the BST, Boorse advances, to my mind, the most promising naturalist theory of health and disease. However, it should be clear that the BST is a theory beset with some profound problems. The crux of these problems is that the framework of the BST essentially involves use of a criterion of health which turns out to be a Cambridge-change criterion: Such a criterion allows changes in *relational* properties to make the individual diseased or healthy without any seeming change in the individual's “internal” or “intrinsic” states—a problem that ultimately renders the BST an unsuitable candidate

relevant functions conform to (diverge from) the statistical norms of the appropriate reference class.

⁵¹ Boorse claims that the BST “furnishes medicine with a basic scientific concept of disease as a scaffold on which medicine, and society at large, can build clinical and social disease concepts” Boorse (1997), p. 53.

for the rôle that Boorse has cast it to play.

It is because Boorse forges his view of “normal” human functioning (biological fitness) ultimately from a “non-normative” conception of statistical normality and its concern is with the present statistical normality of a reference class, that the BST essentially involves the use of a Cambridge-change criterion. Thus an obvious response would be to remove the fundamental role the statistical normality of reference classes plays in the BST’s non-normative view of “normal” human functioning. After all, I have suggested that the Cambridge-change criterion seems to emerge because the statistical norms of the BST’s reference classes *will* undergo changes.

Still, this would of course require the naturalist to have a suitable non-normative conception of “normal” human functioning ready-at-hand. And given the failure to produce the “blueprints” for human functioning one may legitimately wonder if an alternative naturalist conception of “normal” human functioning can ever be persuasively brought to bear. If it cannot then we have hit upon a serious—and perhaps insurmountable—problem not only for Boorse’s BST but for any naturalist claim to underpin medicine with a value-free account of health and disease defined in terms of an evolutionary view of biological fitness.

Whether one can turn to an alternative naturalist conception of “normal” human functioning, which would keep the BST’s strengths and avoid the Cambridge-change objection, is what will concern us for the remainder of this dissertation.

Chapter Four

Defending Naturalism with Natural Kinds?

1. Introduction

The previous chapter argued that the most influential naturalist account of health and disease—Boorse's biostatistical theory of health and disease (BST)—is, in its current formulation, an unsuitable account of health and disease. In particular, I argued that the BST formulates its conception of normal human functioning in terms which leave it open to Cambridge-change objections. This chapter, along with Chapters 5 and 6, will provide a critical analysis of the naturalist's prospects for advancing a more persuasive non-normative conception of normal functioning that may be better able to underpin a more promising value- and norm-independent account of health and disease. As will become clear, the thrust of each of these three chapters is chiefly negative. If we cannot advance a more promising value- and norm-independent account of health and disease, then there will be good reason to side with the normativists in the debate and insist that human value-choice is required.

To start, naturalists inclined to defend Boorse's BST against the problems I outline in Chapter 3 will find there to be several different ways of responding to these objections. For convenience, the objections may be said to fall under two headings:

- (A) the BST cannot avoid being formulated in terms which involve the use of a criterion in a way which leaves the BST open to Cambridge-change objections,

and hence, because of the problems that this leaves the BST open to,

- (B) if the BST cannot avoid the use of a criterion which is open to Cambridge-change objections this fact ultimately renders the BST to be

an unsuitable candidate to solely underpin an adequate account of health and disease.

There is an immediate and response to (A): The defender of the BST should try to find a reformulation of her account of health and disease which does not use any criteria open to Cambridge-change objections. There are in fact at least two ways of trying doing this, ways which as a matter of fact were adumbrating in the last chapter (section 3.3). There I distinguished between two issues facing the BST: One was the issue of *which biological processes* are to be categorized as “normal” functions of human beings, and the other was the issue of *which level of functioning* is requisite for a goal of a normal function to be successfully realized. Using terminology from Elselijn Kingma I referred to the former as “qualitative issues” about functions and to the second as “quantitative issues” about functions. In my view, both approaches may be said to set out to employ different means to the same end: to prevent the BST from being open to Cambridge-change objections.

It is important to examine the two approaches separately, in part because otherwise there is the risk of conflating what are really two different and distinct issues, even though together they constitute the BST’s account of what is to count as normal human functioning. For our purposes, I think it is reasonable to separate and approach the issues as follows: One is the issue of which conception of “normality”, and the other, which account of “function” the BST employs. I shall examine these in turn. Chapter 5 provides an examination of different conceptions of normality (the first issue), while the second issue, occupies the focus of Chapter 6. As we shall see, some naturalist rejoinders will be more promising than others, but all will come up short.

Recall that a primary goal motivating this dissertation is to see if it is possible to formulate a promising value- and norm-independent account of health and disease. Correspondingly, we should expect the two parts of the various responses to (A) to avoid begging normative or value questions.

With this in mind, it might be thought that a promising approach to finding a value- and norm-independent account of health and disease might lie in investigating the possibility of treating disease as if it were a *natural kind* and then trying to characterize disease-states in terms of their *essential* natural properties. In this way, “natural kinds” might be thought to mark a value- and norm-independent distinction between health and disease.

Certainly, it has been a long-standing view (the view sometimes called “essentialism”¹) that the world is populated by biological objects with properties that can be viewed as forming natural kinds of the sort that, initially at least, would seem to comfortably underpin a naturalist account of health and disease. There have been some that have wanted to treat at least some properties, events, states, and processes as natural kinds, counting, along the way, diseases as natural kinds.² Now if this view about the connection between the possibility of finding a naturalistic conception of disease and

¹ Essentialism concerning natural kinds can be traced back to Aristotle, Locke, and J. S. Mill; and more recently Kripke and Putnam. See, for instance, my discussion of Locke below; Michael Ayers (1982), “Locke versus Aristotle on natural kinds”, *The Journal of Philosophy* 78(5): 247-272; Saul Kripke (1980), *Naming and necessity*. Oxford: Blackwell; and Hilary Putnam (1975), *Mind, language and reality: Philosophical papers, volume 2*. Cambridge: Cambridge University Press.

² See, for instance, T. E. Wilkerson (1998), “Recent work on natural kinds”, *Philosophical Books* 39(4): 225–33; Crawford Elder (1994), “Higher and lower essential natures”, *American Philosophical Quarterly*, 31(3): 255-265; Rachel Cooper (2005), *Classifying madness*. Dordrecht: Springer, especially Chapter 2 “Are mental disorders natural kinds?”; and Stefan Dragulinescu (2010), “Diseases as natural kinds”, *Theoretical Medicine and Bioethics* 31(5): 247-369. This is a representative sample, not an exhaustive list.

treating it as a natural kind was correct, then clearly *this* would be a promising and foundational issue for a naturalist account concerning health and disease.

Furthermore, to the extent that it might be correct, it would go a long way to affirming what many seem to implicitly assume: that modern medicine is at its core a scientific, value- and norm-independent enterprise (as they sometimes put it, lumping the two together, a value-free enterprise).³

It is important to pause here to acknowledge that essentialist thinking concerning natural kinds swims against the current of the received view in the philosophy of biology. Indeed, one often hears it said in the philosophy of biology literature that there is a near consensus that traditional essentialism is incompatible with our best theories of biological kinds.⁴ But even if philosophers of biology and biologists are correct to pitch the rejection of essentialism as inimical to Darwinian theory, it does not immediately follow that the essentialist is thereby mistaken about disease being a natural kind. The notion that all disease forms a class in virtue of common, underlying natural properties is not *ipso facto* incompatible with the Darwinian tenets of variability and change.⁵ For example, while

³ See, for example, Robert D'Amico (2007), "Disease and the concept of supervenience", in H. Kincaid & J. McKittrick (Eds.), *Establishing medical reality*. Dordrecht: Springer, 35-45, at p. 36.

⁴ See, for example, Ernst Mayr (1976), *Evolution and the diversity of life: Selected essays*, Cambridge, Mass.: Harvard University Press, 5th printing 1997, pp. 26-30. Samir Okasha (2002), "Darwinian metaphysics: Species and the question of essentialism", *Synthese* 131(2): 191-213, at p. 191. Marc Ereshefsky (2010), "What's wrong with the new biological essentialism", *Philosophy of Science*. Robert A. Wilson, R., Matthew J. Barker, & Ingo Brigandt (2007), "When traditional essentialism fails: Biological natural kinds", *Philosophical Topics*, 35(1-2): 189-215 who "think that this near consensus view is correct". For an excellent overview of the consensus view and some of its leading participants see, Michael Devitt (2008), "Resurrecting biological essentialism", *Philosophy of Science*, 75: 344-382, especially at pp. 349-351.

⁵ I sympathize with Michael Devitt's observation that, "the very term 'essentialism' has become so distasteful to biologists because of its association with Aristotelian metaphysics that a biologist would doubtless be reluctant to admit to any sort of essentialism". Michael Devitt (2008), *op cit.*, at p. 347.

arguing that “the concept of disease must make necessary reference to a natural kind”, Daniel Sulmasy goes on to provide an important insight into one way the concept of a natural kind may be wedded with biological evolution. He writes:

That natural kinds have law-like principles that determine how they develop and flourish as the kinds of things that they are is simply a fact about the world as we encounter it...That living things evolve over time is simply one of the law-like generalizations that characterize them.⁶

Building upon the insights of essentialist philosophers concerning natural kinds, Jerome Wakefield contends that, what he calls, “black box essentialism” (BBE) underlies a value-independent conception of natural function.⁷ Though I seriously doubt many medical researchers are aware of the insights of philosophical discussions of natural kinds, certainly many notable medical research projects seem to simply assume an essentialist view concerning natural kinds.⁸ Consider, for instance, those research projects seeking to discover the genetic origins of homosexuality or alcoholism. Why even initially suppose the existence of a “gay gene” unless one is making heavy use of the essentialist view that one can properly characterize homosexuality in term of *essential* natural properties.⁹ It is

⁶ Daniel P. Sulmasy (2005), “Disease and natural kinds”, *Theoretical Medicine and Bioethics* 26: 487–513, at pp. 491 & 493. Though, Sulmasy denies that disease is a natural kind, he argues that the concept of human disease makes necessary reference to human beings, which is a living natural kind. Thus he concludes: “the concept of a disease must make necessary reference to a natural kind”.

⁷ Jerome C. Wakefield (1999), “Mental disorder as a black box essentialist concept”, *Journal of Abnormal Psychology* 108 (3): 465–472. Wakefield, through a series of papers, avers what he calls the “harmful dysfunction” (HD) analysis of disorder, which most notably augments a value-free concept of biological function with a value-laden requirement of “harm”.

⁸ A notable example is a new initiative by the U.S. National Institute of Mental Health (NIMH) to classify psychiatric disorders based on identifiable neural biological causes. This marks a meaningful departure from the *DSM*’s classifications based on signs and symptoms rather than causes. See Greg Miller (2010), “Beyond DSM: Seeking a brain-based classification of mental illness”, *Science* 327: 1437.

⁹ As Perlman correctly notes, disease classification historically relied on signs and symptoms but “[n]ow, we increasingly rely on laboratory criteria and define diseases according to their causes”. In this way, advances in technology seem to provide a continuing impetus for an essentialist view about disease. See Robert L.

apposite to mention that this is precisely what Kripke contends is going on:

In general, science attempts, by investigating basic structural traits, to find the nature, and thus the essence (in the philosophical sense), of the kind.¹⁰

For reasons that will become clear as we progress, the allure of essentialism concerning natural kinds seduces medical research and practice into disregarding many serious issues.¹¹ Hence, let me merely note that, given our primary goal to examine the prospects for a naturalist account of health and disease, essentialism concerning natural kinds is if only for these reasons a view we must seriously entertain. However, as it will become clear, for all the ink spilt on natural kinds, the view that disease is a natural kind fixed by a common set of underlying essential natural properties is more familiar than persuasive.

Let us now turn to this chapter's current focus: The possibility of parlaying "natural kinds" into a naturalist defense against Cambridge-change objections. It is important to be clear about my present concern. It is not to examine the merits of natural kinds beyond the possibility of it playing a pivotal role for a promising naturalist account of health and disease.¹² In this way, our analysis of natural kinds will carry significant implications for

Perlman (2005), "Why disease persists: An evolutionary nosology", *Medicine, Health Care and Philosophy* 8: 343-350, at p. 344.

¹⁰ Saul Kripke (1972), *op cit.*, p. 138. More recently, Wilkerson, also an advocate of this view, further contends: "it is strictly only members of natural kinds, and the corresponding real essences, that lend themselves to scientific investigation. Natural kind predicates are inductively projectible [sic], other predicates are not." See T. E. Wilkerson (1998), *op cit.*, at p. 227.

¹¹ H. G. Wright, for instance, persuasively argues that the overall disease category is *radial* not classical and given a failure to appreciate this, he concludes, "The art of medicine, and the character virtues on which it depends, are surviving in spite of conditions in Anglo-American medicine; not thriving because of them". H. G. Wright (2007), *Means, ends and medical care*. Dordrecht, The Netherlands: Springer, at p.161.

¹² Thus I will have little to say about many notable metaphysical criticisms that some might raise against traditional essentialism concerning natural kinds, for example, such as Lewis' argument about the possibility

the remainder of this dissertation.

2. Natural kinds

What is at issue here is whether the concept of disease can be treated as solely the object of scientific enquiry, in general, and biology, in particular. The stakes are high because, given modern medicine's fundamental concern to identify and treat disease, the very notion that modern medicine is, at its core, a scientific enterprise seems to imply a naturalist account of disease. For instance, to distinguish between states of health and disease we should expect to carve nature at its joints with a knife that is no more value-dependent or social norm-structured than the biologist employs to distinguish between tigers and lions or the chemist, to distinguish between gold and iron pyrite. For any self-respecting naturalist would surely be appalled to find that her or his analysis of disease and health was as norm-structured as any account of the difference between a weed and a garden flower, which, to quote Patrick Norwell-Smith, has to be viewed as being:

Consider...our use of the word 'weed'. The ordinary man...takes, rightly or wrongly, an uncompromisingly realistic view of [word]...such as 'dandelion' and 'yellow'. He believes that even if there were no gardeners there would still be dandelions and that they would still be yellow. But, if there were no gardeners, would there still be weeds? To say that a dandelion is a weed is not like saying that it is a member of the order *Compositae*; and the difference does not lie only in the fact that 'weed' is an ordinary-language word. To say that dandelions are weeds is not to classify them at all. For the contrast between weeds and flowers (in that sense of 'flowers' in which flowers are contrasted with weeds) depends on the interests of gardeners. If there were no gardeners we should have no use for this contrast; and if the interests of gardeners changed, if, for example, dandelions came to be admired for their

of change being a problem for any kind of essentialist account of individual identity and hence for kinds. See Brian Weatherson (2008), "Intrinsic vs. extrinsic properties", in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. <<http://plato.stanford.edu/archives/fall2008/entries/intrinsic-extrinsic/>> Accessed July 4, 2011.

beauty, rarity, or medicinal properties, dandelions would cease to be weeds. A weed is, roughly, a plant that we wish to eradicate rather than to cultivate. If a man said that he liked cultivating groundsel, we might think him odd; but if he said that he liked cultivating weeds, this would be logically odd and we should have to take him to mean that he liked cultivating those plants that others usually wish to eradicate. In this way we could remove the logical (but not the horticultural) oddness from what he says by making 'weed' into a descriptive expression.¹³

More to the point, the issue motivating this chapter can be restated: The concern is whether the fixing of states as being disease-states (or not) is solely a value-free and social norm-independent matter of the discovery of what is “given in nature”, or if it is a function of value-laden or norm-structured human choice. Thus, an important point of contention is the extent to which humans discover and/or create the factors which fix states as disease-states.

When our concern is with the nature of classification we enter into a familiar and long-standing area of debate in the history of western philosophy. As I said, in a modern incarnation the issues here have been articulated as the question of whether or not disease is a *natural kind*.¹⁴ Of course, how one answers this question depends on how one characterizes the category “natural kind”. So, let me look briefly at that question.

Several different and opposing accounts of natural kinds have been advanced over the years.¹⁵ However, given the task at hand, not every account will be a candidate for

¹³ Patrick Nowell-Smith (1954), *Ethics*. Harmondsworth, England: Penguin Books, p. 72.

¹⁴ See, for example, Khushf (2001), “What is at issue in the debate about concepts of health” in Lennart Nordenfelt (Ed.), *Health, science and ordinary language*. Amsterdam: Rodopi Press, 123-169, especially pp.133-135; Robert D’Amico (1995), “Is disease a natural kind?”, *The Journal of Medicine and Philosophy* 20: 551-569; Reznick (1987), *op cit.*, & (1995), “Dis-ease about kinds: A reply to D’Amico”, *The Journal of Medicine and Philosophy* 20: 571-584.

¹⁵ It is familiar that natural kinds has been an issue central to philosophy of science. The extent to which, if at all, science distinguishes distinct classes of objects by ‘essences’, in terms of necessary and sufficient

serious consideration. The naturalist's concern to employ a value-free and norm-independent account of health and disease will eliminate any formulation of a natural kind that is, either explicitly or implicitly, value-laden or norm-structured.¹⁶ Though acknowledging the different traditions at play in various formulations of the notion of a natural kind,¹⁷ Ian Hacking usefully suggests a principle that I take to be a general constraint that any naturalist account of the state of health and of disease must abide by *in one way or another*. As will emerge, this final italicized phrase is important. Hacking calls this principle:

Independence. It is a fact about nature, independent of psychological or social facts about human beings, that there are *kinds of things*, of substances, of organisms and so forth.¹⁸

Within the modern debate of health and disease, Hacking's suggestion seems to be almost universally implicit, if not explicit, in discussions of whether these "kinds of things"—

intrinsic properties, occupied notable 20th century philosophers of science such as Karl Popper, Rudolf Carnap, and Carl Hempel. More recently, notable philosophical arguments such as Hilary Putnam's on natural kinds and Saul Kripke's on naming have revived essentialism. See above footnote 2, for some prevalent anti-essentialist arguments. Robert Boyd has responded by viewing natural kinds, not as a set of necessary and sufficient conditions, but as essences in terms of non-unifying clusters of properties, which he calls 'homeostatic property cluster (HPC) kinds'. Paul Griffiths defends what he calls 'relational essentialism', where essences are defined not in terms of intrinsic properties but, rather, as essential relational properties. See Richard Boyd (1999), "Homeostasis, species and higher taxa", in R. Wilson (Ed.), *Species: New interdisciplinary essays*. London: MIT Press, pp. 141–186; and, Paul E. Griffiths (1999), "Squaring the circle: Natural kinds with historical essences", in R. Wilson (Ed.), *op cit.*, pp. 209–228. For a survey of the 'natural kind' literature see, Alexander Bird & Emma Tobin (2009), "Natural Kinds", in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*, <<http://plato.stanford.edu/archives/spr2009/entries/natural-kinds/>>. Accessed July 4, 2011.

¹⁶ Thus, I take it the naturalist about health and disease will dismiss out of hand natural kind accounts claiming that what counts as health and disease is crucially a function of our conceptual, cultural or social activities and that these *states* are 'created' by particular conventions or to borrow a phrase from John R. Searle, "institutional facts", not unlike the state of marriage, being Prime Minister of Canada, or legal tender is so created. See Searle (1995), *op cit.*

¹⁷ Ian Hacking, for example, distinguishes between what he calls "Mill-kinds", "Leibniz-kinds", Peirce-kinds", "finite kinds" and various "social kinds". See Ian Hacking (1991), "A tradition of natural kinds", *Philosophical Studies* 61: 109-126.

¹⁸ *Ibid.*, p. 110. Italics mine.

substances, organisms, and I might add, states and maybe processes—are “natural kinds” of the sort suitable to underpin a naturalist account of health and disease.

In a notable debate over whether “disease” is to be treated as a natural kind term, Lawrie Reznek clearly can be said to be assuming this constraint on what can be counted as a natural kind when he argues that diseases do not constitute a natural kind because there is no natural property peculiar to diseases that is lacked by all non-diseases.¹⁹ Reznek explicitly treats a natural kind as “a class of objects that shares some deep or theoretically interesting underlying nature [“independently of us”] which explains the cluster of properties each member of that class shares”.²⁰ Many have felt that this issue is a pivotal issue for discussions of the possibility of treating health and disease as natural states. If there are *no* common natural defining properties of disease, then it cannot be treated as a natural kind and hence cannot be given a naturalistic interpretation. William Stempsey emphasizes the significance of this view when he remarks:

In the absence of necessary and sufficient conditions to stipulate [sic] that a given state of affairs belongs to some classificatory set, it will be necessary to make value judgments about how much of a family resemblance is necessary to judge that one is dealing with a cluster that constitutes a disease category.²¹

A prevalent underlying assumption in the modern debate of health and disease is thus this: if disease were convincingly shown to be a natural kind, then disease classification would be tantamount to identifying the relevant empirical properties furnished by nature. Importantly, knowledge of the underlying structure common to the

¹⁹ Reznek. (1987), *op cit.* For the debate over disease being a natural kind term see: D’Amico (1995), *op cit.*; and Reznek (1995), *op cit.*

²⁰ Reznek (1995), *op cit.*, at p. 571 and 574.

²¹ Stempsey (1999), *op cit.*, p. 156.

genuine natural kind disease gives us, *eo ipso*, a principled value-independent way to distinguish disease from health. Such knowledge would then place the naturalist in the enviable position of being able to explain and predict an expected behaviour of disease. Hence a naturalist account of health, as merely the absence of disease, will have won the day. Determining whether or not a condition(s) warrants disease-status would not be unlike the way one determines a piece of metal to be gold, i.e., by some process something like identifying the relevant atomic structure. There is a question we must therefore concern ourselves with: Is disease such that its states are fixed by a common set of natural properties in virtue of which the states are diseased?

2.1 Reznek's argument that disease is not a natural kind

As mentioned above, Reznek argues that diseases and disease-states *are not* fixed by a common set of natural properties in virtue of which a state is diseased. According to Reznek, the very notion that there is some underlying common nature of disease that determines disease-status turns on the mistaken belief he calls the “essentialist fallacy”, which:

We have inherited...from Hippocrates who assumed that all diseases had the nature of being humoral imbalances. This *view* was taken up by the Germ Theory in the nineteenth century that argued that all diseases had the nature of infections. However, the disease status of a condition is settled *before* its underlying nature is known. We knew all along that Parkinson's disease was a disease even before we knew anything of its cause (the deficiency of dopamine in the *substantia nigra*). Tuberculosis was known to be a disease long before the bacillus that causes it was discovered. It is not the underlying nature of a condition that determines whether or not it is a disease—it is the consequences.²²

²² Reznek (1995), *op cit.*, p. 575.

I strongly suggest that Reznek is absolutely correct to insist that it is the consequences caused by being in a disease-state that determine the disease-status of the state. However, this in itself is not enough to establish the ontological point that disease is not a natural kind and that disease-states do not form a natural kind. To see this, let us begin by noting that Reznek's criticism is directed against a particular tradition of natural kinds that has come to be called "essentialism" in philosophy. Robert D'Amico describes it thus:

In this tradition the notion of a kind refers to the lawlike relations that hold for the essential or microphysical structure of things.²³

Complementing and further explicating this notion of natural kinds, Ereshefsky more recently writes that traditional essentialism has three main tenets:

- (i) all and only the members of a kind share a common essence
- (ii) that essence is a property, or a set of properties, that all the members of a kind must have
- (iii) a kind's essence causes the other properties associated with that kind.²⁴

What then of the claim that disease is a natural kind of the essentialist sort? It is difficult to say with precision. At least part of the problem, I believe, is that it is not at all clear how to understand the pivotal notion of "essence" in any non-question-begging sense. Fortunately, for our purposes it will suffice to employ the above as a touchstone in getting clear about traditional essentialism concerning natural kinds. Accordingly, in light

²³ D'Amico (1995), *op cit.*, at pp. 554-55.

²⁴ Marc Ereshefsky (2009b), "Natural kinds in biology", In E. Craig (Ed.), *Routledge Encyclopedia of Philosophy*. London: Routledge. <<http://www.rep.routledge.com/article/Q124SECT1>> Accessed July 4, 2011.

of the above, for *the naturalist* concerning health and disease to adopt the traditional essentialist notion that disease is a natural kind would thus seem to imply at least the following corresponding claims:

- (i*) S [a state or process] is of kind D [disease] *iff* S has E [common essence].
- (ii*) S is of kind D in virtue of its possession of unique set of natural properties E such that every S with E is of kind D and no S without E is of kind D .
- (iii*) p is a defining property of kind D *iff* p is caused by E .

More fully, these three claims portray the following sort of characterization:

- (a) A natural property or set of natural properties F_1, \dots, F_n forms the essence of items of a kind K *iff* x : if x is of kind K then x has properties F_1, \dots, F_n [From ii*].
- (b) Let natural properties G_1, \dots, G_n form the essence of items in kind D .
- (c) x : x is of kind D *iff* x has properties G_1, \dots, G_n , [From i*].
- (d) P : if P is *associated with* items of kind K [e.g., P is a sign/symptom/ marker, etc., of D] and P is not identical to any property G_i in the set G_1, \dots, G_n , then there is a property or properties G_i such that G_i caused P . [From iii*].

So, for the defender of a naturalist account of health and disease, I submit the following to be the case: The application of (i*) implies necessity as well as sufficiency, i.e., possession of E is necessary for membership of D , as well as being sufficient for it. The addition of (ii*) implies an ontological commitment that there exists a common set of natural properties (E) that is intrinsic²⁵ to all and only diseases. The upshot of the

²⁵ Just how best to define what it is to be an intrinsic property is controversial. For a survey of this literature see Weatherson (2008), *op cit*. For my purposes it suffices to say that the traditional essentialist concerning natural kinds will want to say that all members of the kind are so in virtue of a common set of intrinsic—regardless of its precise characterization—properties that are essential for membership of that kind. See

conjunction of (i*) and (ii*) is that disease-states are categorically distinct from health-states and every *S* of kind *D* (extension) can be identified—at least in principle—by an intrinsic structure *E* (intension). I take (iii*) to further imply two related explanatory commitments: That *E* is composed of essential (rather than accidental) natural properties;²⁶ and that *E* exhaustively determines the characteristic etiology of *S* and, as such, provides a foothold for science to predict and explain the properties of *S* of kind *D*.²⁷

Now, with this in mind, I would like to raise some concerns with Reznek's argument against disease being a natural kind of the sort essentialism avers. First, the crux of a knockdown argument against the essentialist variant of the view that disease-states form a natural kind cannot merely be the epistemological point that "the disease status of a condition is settled *before* its underlying nature is known". This is because this argument involves the fallacious inference that "what we do not know" therefore "cannot be". After all, before anyone identified scurvy to be a deficiency of vitamin C, any salty-sailor would have told you that one does not need to know *what* is killing an individual to know *that*

further J. Michael Dunn (1990), "Relevant predication 2: Intrinsic properties and internal relations", *Philosophical Studies* 60(3): 177-206, especially at p. 178: "Metaphysically, an *intrinsic* property of an object is a property that the object has by virtue of itself, depending on no other thing."

²⁶ For a discussion on the distinction between 'essential' and 'accidental' properties see, Teresa Robertson (2008), "Essential vs. accidental properties", in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. <<http://plato.stanford.edu/archives/fall2008/entries/essential-accidental/>> Accessed July 4, 2011. Following Robertson's modal characterization of the distinction, I take an essential property of an object to be a property that an object must have and, as such, could not lack it and still be of that kind.

²⁷ This seems to fit nicely with the views of two recent advocates of essentialism concerning natural kinds. Consider Wilkerson's Aristotelian inspired account: "certain kinds of things have a special status because they are determined by 'real essences', intrinsic properties or sets of properties which are necessary and sufficient for membership of the kinds, and which in turn underlie their causal powers." Wilkerson (1998), *op cit.*, at pp. 225 & 228. And Ellis' contention that: "The real essence of any natural kind is a set of properties or structures in virtue of which a thing is a thing of this kind, and displays the manifest properties it does...The essential properties of a kind include all of the intrinsic properties and structures that together make a thing the kind of thing it is". Brian Ellis (2001), *Scientific essentialism*. Cambridge: Cambridge University Press, at pp. 54-55.

something is. Correspondingly, Reznick's *epistemological* point hardly shows that there is no essential or microphysical underlying structure of disease to identify. At most, it shows that the classification of diseases does not require one to identify an underlying essence or nature or cause of the disease.

But in many ways this is a modest achievement. It is hardly ground-breaking. Consider the fact that in the 17th century John Locke was well aware that it is a mistake to think that what we do not know cannot be and that disease classification (or anything else for that matter) is settled *before* the intrinsic underlying nature of disease is known. Locke notably distinguished between *real* (the intrinsic underlying microphysical structure) and *nominal* (the observable characteristics by which we stipulate the abstract idea) essences and famously argued that real essences are unknowable. Thus, on Locke's view, it isn't that there is no underlying microphysical structure (i.e., the real essence) upon which the nominal essence depends but that this fact of the matter will always be hidden from us.²⁸

The upshot is that if we wanted to know whether something was gold or not we have no choice but to appeal to the nominal essence of gold, which for Locke was "a body yellow, of a certain weight, malleable, fusible, and fixed".²⁹ As such, it may strike the attentive reader that the force of Reznick's epistemological claim that it is the consequences of a disease—and not any underlying nature of disease—that determine its disease-status vindicates Locke's essentialist view. That this could be so is because

²⁸ Here I concede Locke's claim that real essences are unknowable seems untenable given that modern science tells us that the underlying structure of gold is identifiable by the atomic number 79 but this does not impact the force of the present point.

²⁹ John Locke (1690), *An essay concerning human understanding*. Kitchener: Batoche Books, 2001, at III, vi, 2.

Reznek's epistemological claim does not, in itself, stand incompatible with any of the essentialist's three tenets or even the more specific ontological claim that disease-states are fixed by a common set of natural properties in virtue of which the states are diseased.

There is good reason to think that Locke's view concerning the role of nominal essences has some traction today. It would be absurd to deny that ordinary medicine successfully identifies disease-states by identifying *observable* properties malfunctioning (i.e., signs of disease).

To illustrate the force of this point, consider the fact that Hippocrates is said to have described scurvy as a disease.³⁰ That Hippocrates was able to recognize *that* something is pathological long before others were able to identify *what* the underlying pathological condition is is the stronger epistemological point I think Reznek should have explicitly pressed further. I take this to be a stronger epistemological point not only because it persuasively motivates Reznek's insistence that it is the consequences of a disease that determine its disease-status—how else might have Hippocrates identified scurvy to be a disease? It also strongly supports the view that if disease is a natural kind then it is, to use Jasper Reid's phrase, "the macroscopic, observable properties of things"³¹ that determine whether a condition truly is a disease.

An even stronger case can be made, I believe, by recognizing that tremendous advances in technology have made many once unobservable underlying natural properties, observable. Consider, what many take to be the leading exemplar of natural kinds—the

³⁰ Irwin Stone (1966), "On the genetic etiology of scurvy", *Acta Geneticae Medicae et Gemellologiae* 15(4): 345–50. <http://www.seanet.com/~alexs/ascorbate/196x/stone-i-acta_genet_med_et_gemell-1966-v15-p345.htm> Accessed July 4, 2011.

elements. It is significant that science has identified that all and only gold atoms have 79 protons. The ability to observe a fundamental difference in properties gives us overwhelming scientific evidence countenancing the view that gold constitutes a natural kind that is categorically distinct from other elements. The naturalist concerning health and disease must take seriously that our best scientific technologies have utterly failed to identify any set of intrinsic natural properties that *all and only* diseases have. This strikes against essentialism, which, we saw, claims that the lawlike relations that hold for a distinct “essential” underlying microphysical structure of things ultimately fix the notion of a kind.

Further evidence against the view that disease is a natural kind will surface in what follows. But first, let us deviate slightly to acknowledge an important point about which I believe essentialism is correct.

3. An important essentialist point: Specific diseases may be natural kinds

I concede the above reasoning hardly shows that essentialism itself is false, even in relation to diseases. And, what is more, I believe the essentialist may be right about a very important point: *specific* diseases may possibly be properly classed as forming genuine natural kinds.³²

For the sake of clarity, I will henceforth use the term “disease *qua* disease” to name

³¹ Jasper Reid (2002), “Natural kind essentialism”, *Australasian Journal of Philosophy* 80(1): 62-74.

³² Crawford Elder, for instance, is an advocate of essentialism whom advances a similar view about disease. He contends that the diseases hepatitis A, hepatitis B, hepatitis C, and hepatitis D each form a distinct “lower” genuine natural kind. See, Elder (1994), *op cit.* For an interesting argument that some types of diseases—including some types of mental disorder—form natural kinds see, Rachel Cooper (2005), *op cit.*, especially Chapter 2.

the single class of all diseases which contains specific diseases as its members.

Here what I mean by “specific diseases” may perhaps best be illustrated by various states that the World Health Organization identifies and classifies as different three-character code disease entries in its *International Statistical Classification of Diseases and Related Health Problems* (ICD).³³ The ICD assigns a unique three-character code to each disease, with additional characters used to further specify the disease. The tenth and most recent edition (*ICD-10*), replaces the purely numeric coding of previous versions with an alphanumeric code having a letter always as the first character. For instance, Type I Diabetes Mellitus (E10), Thalassemia (D56), Cystic Fibrosis (E84), and Huntington's disease (G10) are each examples of what I am calling a specific disease. Each is a specific disease that constitutes the larger class “disease *qua* disease”.

The point the essentialist should press is that it is significant that the classification of specific diseases and other morbid conditions in *ICD-10* is explicitly guided by:

The principal objective...to provide, for each moribund entry, a single recommended name. The main criteria for selection of this name are that it should be specific (applicable to one and only one disease), unambiguous, as self-descriptive as possible, as simple as possible, and (whenever feasible) based on cause.³⁴

In light of its stipulated criteria for selection, it is difficult to see how the *ICD-10* classification can be considered a success unless it is identifying an underlying nature of the specific disease fixed by a unique set of natural properties. After all, a classification

³³ Nordenfelt (2001), *op cit.*, at p. 21, contends that “almost all contemporary national classifications of diseases and related problems are based on the *ICD*”.

³⁴ World Health Organization. (2004), *ICD-10: International statistical classification of diseases and related health problems: tenth revision (2nd Ed.)*, Geneva: Author, p. 9.

that is specific, as self-descriptive as possible and (whenever feasible) based on cause will not be fixed by accidental signs and symptoms or shared intrinsic properties. To that extent, the *ICD-10* classification seems to be strongly reminiscent of the essentialist criteria (i*-iii*).

Moreover, it is not difficult to provide empirical evidence to support this view. Consider that for many diseases, it is now possible to identify an underlying nature of the specific disease fixed by a unique set of natural properties, which explains the cluster of properties. For example, Huntington's disease (G10) is caused by mutations in the Huntingtin gene (HTT) on chromosome 4 in which DNA bases, composed of the sequence CAG, are abnormally repeated many, many times. In the inherited autosomal recessive disease thalassemia (D56), the genetic defect results in reduced rate of synthesis of either α or β globin. More specifically, we identify α -thalassemia (D56.0) as the genetic defect of two closely linked genes HBA1 and HBA2 on chromosome 16 resulting in decreased alpha-globin production. β -thalassemia (D56.1) is fixed by mutations in the HBB gene on chromosome 11 which results in decreased beta-globin production. Cystic fibrosis (E84) is an autosomal recessive disease fixed by a mutation in the cystic fibrosis transmembrane conductance regulator (CFTR).

The traditional essentialist concerning natural kinds is right, I think, to insist that each of the above specific disease can intelligibly be classed as a genuine natural kind in its own right. Each forms a class “that shares some deep or theoretically interesting underlying nature which explains the cluster of properties each member of that class shares”. Or put slightly differently, each specific disease is such that its states are fixed by a common set of underlying molecular or genetic properties in virtue of which *that* state

is diseased.³⁵

And it is just in this sense that medical practice is accurately able to identify the presence of a particular disease (e.g., Down Syndrome, Tay-Sachs, and Sickle Cell Anemia) from other states *in utero*. The extent to which modern medical science is successfully able to identify, predict, and distinguish between specific diseases in virtue of a common set of underlying molecular or genetic properties provides empirical evidence that the ontological and explanatory commitments implied in the main tents (i*-iii*) of traditional essentialism are attained.

I readily admit that there are numerous disease classifications in the *ICD-10* where medical science has yet to identify an underlying common set of natural properties, which explains the cluster of properties each member of that class shares. Indeed, the exact cause of Type I Diabetes Mellitus (E10) is unknown. Nevertheless, I believe it is reasonable to think there is an underlying common set of natural properties, given that medical science has identified autoimmune destruction of insulin-producing beta cells of the pancreas. The pancreas becomes unable to secrete the requisite insulin in most people with Type I Diabetes Mellitus.³⁶ Other diseases, like Fibromyalgia (M79.7)—whose code was recently revised in 2006 from ‘Rheumatism, unspecified (M79.0)’³⁷—is such that not only is the cause unknown but there is also no specific cluster of natural properties specified.

³⁵ I read Rachel Cooper to be arguing for something very similar to this when she writes, “I argued that the best account of natural kinds is one according to which members of a natural kind possess similar, although not necessarily identical, important properties. These important properties determine many of the other properties possessed by the member of the kind”. See Cooper (2005), *op cit.*, at p. 72.

³⁶ Mayo Foundation for Medical Education and Research. <<http://www.mayoclinic.com/print/type-1-diabetes/DS00329/DSECTION=all&METHOD=print>> Accessed July 4, 2011.

³⁷ World Health Organization (2009), *Official ICD-10 WHO updates combined 1996-2008, Volume 3*. <<http://www.who.int/classifications/icd/icd10updates/en/index.html>> Accessed July 4, 2011.

I am inclined to think that this merely demonstrates an epistemological shortcoming rather than a problem against the ontological view that disease-types are natural kinds fixed by a unique set of natural properties. After all, identifying the underlying properties that are responsible for the signs and symptoms correctly associated with a specific disease is a complex, difficult and time-consuming matter. Mistakes can be made in drawing inferences from signs and symptoms to correct diagnosis of a disease. Also, some diseases may overlap in a number of their observable properties. Most important, at one time or another, we have lacked our current knowledge of any given disease.

Again, the corresponding point is that the present lack of empirical knowledge to substantiate essentialist claims about diseases does not demonstrate their falsity. Thus we should not rush to conclude that the underlying properties of a particular disease do not exist (an ontological matter) from our failure to identify the requisite underlying properties (an epistemological matter).

However, I want to be clear: My claim is not suggesting that if the *ICD-10* assigns a unique three-character code to a disease then that specific disease is, in fact, a natural kind. The *ICD-10* (and future versions) can be mistaken with their disease classifications. And, *a fortiori*, the World Health Organization explicitly acknowledges their classification of moribund entities is to some extent arbitrary:

The ICD has developed as a practical, rather than a purely theoretical classification, in which there are a number of compromises between classification based on etiology, anatomical site, circumstances of onset, etc.³⁸

³⁸ World Health Organization. (2004), *op cit.*, p. 10.

What is to be stressed is that the essentialist need not share the same interests of classification. So, for example, while *ICD-10* assigns five unique three-character codes to Tuberculosis (A15-A19), to distinguish between means of confirmation, location and accompanying signs, the essentialist need not insist that each unique three-character code names a distinct disease (i.e., natural kind). So there may be one specific disease, Tuberculosis, which the essentialist could say formed a natural kind, even though *ICD-10* assigns greater than one three-character code to the disease. For the *ICD-10* may assign unique three-character codes for a variety of interests and purposes.

At any rate, I believe that many of the disease-states to which the *ICD-10* assigns a unique three-character code may be properly thought to constitute natural kinds. That we can identify and differentiate between disease-states at the DNA coding stage provides very good reason to think that *specific diseases* are natural kinds fixed by a unique set of natural properties which explains the cluster of properties each member of that class shares. The notion that specific diseases are natural kinds is not merely a vestige from a once-influential philosophical view.

4. Disease *qua* disease does not constitute a natural kind of the sort traditional essentialism avers

None of the above, however, lessens the force of Reznick's argument against counting disease *qua* disease as a natural kind. Reznick is very clear that his argument is against the view that all diseases, or if you like the super-class disease, constitutes a single natural kind. To be clear, he is taking issue with the view that, what I am calling, disease *qua* disease forms a natural kind—not with the issue of whether or not a specific disease may

form a natural kind. He writes:

We will be looking at the question whether diseases as a group constitute a natural kind and not the question whether a single disease (say, multiple sclerosis) [sic] is a natural kind. We are looking at the question whether there is some common underlying nature to *all* diseases and not the question whether all patients with multiple sclerosis [sic] have a common nature. It is only the former question that will help us decide issues of disease status. If all diseases belong to a natural kind, we can regard membership to this class as deciding the question of whether any condition is a disease.³⁹

I think Reznick is correct to reject the essentialist view that diseases form a natural kind that shares some distinct “essential” underlying natural property in virtue of which a state is diseased (though who knows what future medical discoveries might reveal?). For the remainder of this chapter, I will argue that the view that disease is a natural kind fixed by some shared distinct “essential” underlying natural property does not stand up to serious scrutiny. There are compelling reasons to insist that disease *qua* disease does not constitute a natural kind of the sort traditional essentialism avers.

4.1 An essential or microphysical structure of things common to all “diseases” eludes our best science

Here is another arrow in the anti-essentialist’s bow. T.E. Wilkerson suggests that natural kinds would need to be counted inductively projective. He states:

If I know that a lump of stuff is gold, or that the object in front of me is an oak, I am in a position to say what it is likely to do next, and what other things of the same kind are likely to do. I know for example that the gold cannot turn into water, and that the oak will not in due course produce tomatoes. And I know that no other piece of gold could be persuaded to turn into water, and no other oak could be persuaded to produce tomatoes. Certain outcomes are ruled in, and others are ruled out, by the real essences of gold or oaks.⁴⁰

³⁹ Reznick (1995), *op cit.*, p. 573.

⁴⁰ T. E. Wilkerson (1988), “Natural kinds”, *Philosophy* 63:29-42, at p. 30.

To begin with, let us set aside the controversial matter of whether or not ‘oak’ is a natural kind.⁴¹ I think there is a more important issue at stake here. To see this, remember (iii*) which claims: *p* is a defining property of kind *D* iff *p* is caused by *E*. Thus, if disease *qua* disease is a natural kind and the notion of a natural kind is, as is standard in the literature, defined in essentialist terms, then the defender of the BST must insist that the essence of disease rules in a certain outcome namely, that a disease will (*ceteris paribus*) produce a reduction of normal functional ability. Recall, Boorse explicitly stipulates: “*Health* in a member of the reference class is *normal functional ability*: the readiness of each internal part to perform all its normal functions on typical occasions with at least typical efficiency”.⁴²

Here it is worth pausing to mention that genetic diseases, such as Tay-Sachs, Huntington’s, and Progeria⁴³ (to name but three), seem to stand as exemplars of *specific* diseases that are inductively projective. But is there any compelling evidence for the view that there is an essential or microphysical structure of things common to all states of disease that “rules in” the vast and diverse reductions of biological functional ability that medical science identifies with disease-states and also “rules out” states of non-disease?

I do not think so. As shall emerge below, my reasons, in part, are those that led Reznek to claim, “that diseases have too diverse a collection of explanatory natures to enable us to find a common explanatory nature”. He goes on to describe these as follows:

Some are due to viral infections, some bacterial infections, some fungal

⁴¹ For instance, John Dupré claims that it is not “remotely plausible” that the term “oak” names kinds with real essences. See John Dupré (1995), “Wilkerson on natural kinds”, *Philosophy* 64: 248-251.

⁴² Boorse, (1977), *op cit.*, p. 555.

⁴³ This disease is also known as Hutchinson-Gilford Progeria syndrome (HGPS).

infections, and some due to protozoal infections. Some are vitamin deficiencies, others due to vitamin excesses. Some are the result of inborn errors in metabolism, and others are the result of structural abnormalities. Some are due to the multiplication of the body's own cells, others are due to the death of the body's own cells. Some are due to chemical irregularities, others are due to plumbing difficulties. And so on. My challenge here is: Show me what is common to all these diverse individual natures that enables us to see all these conditions as falling into the single natural kind of diseases.⁴⁴

An immediate point to press with the above list of various (and seemingly unrelated) disease etiologies is that Reznick's challenge remains open. Our best scientific theories have failed to identify any distinct intrinsic natural properties that would confirm disease *qua* disease is a natural kind.⁴⁵ And let us not forget, as we saw in Section 2.1, it is nothing short of astonishing the extent to which recent advances in scientific technology has afforded us the ability to observe previously unobservable underlying structures of things. It is therefore significant that the pivotal essentialist claim (i.e., there exists intrinsic natural properties common to all and only "diseases") continues to elude our best science. *A fortiori*, our best scientific evidence provides strong reason to doubt that disease is a natural kind of the essentialist sort.

Again, this does not convincingly show that essentialism concerning natural kinds *is* false. But I am skeptical that one could ever produce a knockdown argument for *that* claim. After all, there is the epistemological matter that just because science has not identified an underlying essence or common cluster of properties of disease *qua* disease proves neither that such an entity does not exist nor that disease *is* not a natural kind.⁴⁶

⁴⁴ Reznick (1995), *op cit.*, pp. 576-577.

⁴⁵ Ereshefsky (2009a), *op cit.*

⁴⁶ Lawrie Reznick (1995), *op cit.*

And this is surely correct. No reasonable person would accept either that microscopic living creatures did not exist long before they were first observed by Leeuwenhoek in 1674, or that the failure of the once-dominant humoral theory of disease to identify infectious diseases proves there were no infectious diseases to be identified.⁴⁷

Nevertheless, it would surely be wrongheaded for the naturalist to think that a more promising account of health and disease would be underpinned by pivotal claims unsubstantiated by science.

4.2 *The difference between health and disease is not an all-or-nothing difference in kind*

With that said, I believe one can bolster Reznick's argument and advance a very persuasive (though again not conclusive) argument that disease is not in principle a natural kind of the essentialist sort. That is, in the terminology I introduced earlier, that disease *qua* disease is not a natural kind. Recall Reznick's list of disease etiologies. He is surely correct that bacterial and fungal infections, structural abnormalities, and the death of the body's own cells are all explanatory causes of diseases. But the mere identification of each of these explanatory causes neither rule in disease nor rule out health.

To illustrate, consider that modern science recognizes that the human body contains trillions of "healthy bacteria" or pro-biotics (e.g., *lactobacillus* and *bacillus*) without which death is almost certain. *Candida albicans* yeast (fungal) is found in low concentrations in healthy intestinal systems, where it assists in the maintenance of an ideal intestinal flora level. In addition, the genetically controlled death by apoptosis of many

⁴⁷ Paul Thagard (1998), "The concept of disease: Structure and change", In: Philip Van Looke (Ed.), *Nature of concepts: Evolution, structure and representation*. London: Routledge, 215-242.

body cells (e.g., skin, intestinal, and the endometrium lining cells) is a healthy condition; they are programmed to die for normal regeneration. Also, the pregnancy of a large fetus is a structural abnormality that interferes with the bladder's functional ability, the body's ability to circulate blood and may even push the stomach out of its normal position. Yet pregnancy is not a disease.

These examples strongly suggest that health and disease is not an all-or-nothing difference in kind. In the face of these examples, the essentialist must nevertheless insist that diseases are categorically distinct from health because this is precisely what the conjunction of (i*) and (ii*) implies. So there is a real sense in which modern science seriously undermines the most basic essentialist claim i.e., that all and only disease states share a common set of natural properties in virtue of which a state is diseased. The above demonstrates that the traditional essentialist concerning natural kinds will have to look beyond the disease etiologies modern medicine typically identifies to find the empirical marker of the requisite kind to give countenance to (i*-iii*).

But those inclined to look deeper will find that the essentialist view that health and disease is an all-or-nothing difference in kind is rooted in a mode of thought to which modern medicine no longer subscribes. It is significant that modern medical practice classifies many conditions as “diseases”—not because of any difference in kind—but because of the *degree* to which some state or process or chemical, etc., is present and the *risk* to the normal functioning and the continued life of that organism which this fact poses. High cholesterol, osteoporosis, diabetes, and hypertension are some of the most

prevalent “diseases” of modern medicine.⁴⁸

Notice here that there seems to be no marked difference in kind between the states denoted by the terms “health” and “disease”. Instead, the defining mark of a disease-state is that it is a state in which there is a *quantitative* deviation from a healthy state. It is a deviation in the level of functioning requisite for a goal of a function to be successfully realized. After all, cholesterol is a requisite structural component of the human body; bone density loss is typical of “normal aging”; Type I diabetes results from the body’s inability to produce insulin. Notably, modern medicine counts the production of too much insulin (hyperinsulinemia) also to be pathological; and so too do pathological conditions surround both ends of “healthy” blood pressure.⁴⁹

Reznek takes this to show that modern medicine must not accept the view that disease constitutes a natural kind. He argues if we want to claim there are specific diseases that are only quantitatively different (i.e., not qualitatively different in kind) from “healthy” conditions, then we must draw the following conclusion:

Therefore, not all diseases are natural kinds: some have explanatory natures that vary only in degrees from the nature of normal individuals.⁵⁰

Reznek’s conclusion seems correct. That there are diseases that differ only in degree from

⁴⁸ Peter Schwartz (2008), “Risk and disease”, *Perspectives in Biology and Medicine*, 51(3), 320-334.

⁴⁹ To illustrate: Recently, as of 2003, the *Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* has defined blood pressure as follows: ‘normal’ if it is below 120/80 mmHg; ‘prehypertension’ 120/80 mmHg to 139/89 mmHg; ‘Stage I hypertension’ 140/90 mmHg to 159/99 mmHg; and Stage II hypertension as greater than 160/100 mmHg. See Joint National Committee. (2003), “The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure”, *Journal of the American Medical Association*, 289(19): 2560-2571. Here it should be noted that although a blood pressure below 120/80 mmHg would not be considered ‘high’ if it were low enough it would be considered by modern medicine as pathological.

⁵⁰ Lawrie Reznek (1987), *op cit.*, p. 181.

health is surely an incompatible fit with traditional essentialism's main tenets (i*-iii*).

Admittedly, one might insist (unpersuasively in my view) that the essentialist thesis that diseases are a natural kind fixed by a common and distinct set of intrinsic natural properties is correct and that current medical practice is mistaken to count these clinical conditions as "diseases". This rejoinder, however, is rather implausible. After all, if one insists that states differing only in degree from health are not diseases then one has to insist that modern medicine has mistakenly identified a great number of states as diseases. But it is difficult to see on what plausible grounds medical practice could be so mistaken given both the success of modern medicine and that the abovementioned states clearly reduce biological functional ability. Quite frankly, it is not at all clear to me how anyone can be familiar with stage II hypertension and Type I diabetes (to give but two examples) yet remain insistent that each is not a diseased state. At any rate, and most important, quantitative differences from healthy states may be states that preclude our ability not only to function "normally" but also to survive. Such states are pathological and medical practice is correct to insist they are diseased states.

Perhaps this might suggest that the essentialist should simply concede that some states that are only quantitatively different from healthy states, such as Stage II hypertension and Type I diabetes, are diseases. The problem with this response, however, is that the essentialist is going to have to insist that these diseases are natural kinds. Reznick forcefully illustrates the problematic implication that follows from the essentialist advancing such a view. He writes:

If we wanted to argue that essential hypertension was a natural kind, holding on to the view that all diseases are natural kinds, we would have to say that even normal people had essential hypertension, though of course very much

less of it! But this view also seems absurd—it is plainly mistaken to argue that Einstein had very much less of the disease of mental retardation than anybody else!⁵¹

The key issue here is that the essentialist requires a common set of natural properties (*E*) that is intrinsic to all and only diseases (recall ii*). Yet the difference between a diseased individual with essential⁵² hypertension and a healthy person doesn't seem to be any *qualitative* difference in kind but, rather, a *quantitative* difference in blood pressure.

This quantitative difference is significant because it is familiar and uncontroversial that a natural kind through transmutation can change into another kind. Ernest Rutherford famously showed this by changing the element nitrogen into the element oxygen.⁵³ It is dubious, however, that the essentialist can properly account for the change in disease-status here on the grounds that a state *S* of kind *H* [health] changed into a different kind, *D* [disease]. After all, blood pressure clearly seems to be a paradigm example of a level of functioning having a normal or healthy distribution with pathological tails at both ends (i.e., hypertension and hypotension).

What is more, our ability to function successfully requires our blood pressure to rise and fall. Essential hypertension thus seems to be an example of a disease that differs only in degree from health. But if the difference is only quantitative, then medical science runs

⁵¹ Ibid., p. 181.

⁵² To be clear, medical practice distinguishes between 'essential' hypertension (also referred to as 'primary' or 'idiopathic' hypertension) from 'secondary' hypertension. The former is defined as having no identifiable cause and is most common, accounting for 95% of all cases of hypertension; the latter is defined as having been caused by (i.e., secondary to) another condition. Oscar A. Carretero & Suzanne Oparil (2000), "Essential hypertension: Part I: Definition and etiology", *Circulation* 101: 329-335.

⁵³ For a lucid discussion on the history and science behind Rutherford's demonstration of transmutation see Chapter 21 in, William H. Cropper (2001), *Great physicists: The life and times of leading physicists from Galileo to Hawking*. New York: Oxford University Press, Inc.

against the essentialist notion that a once-healthy individual that is now counted as having hypertension has acquired (?) the requisite set of natural properties (*E*) of the kind *D*. Accepting the antecedent of this conditional, therefore, is tantamount to denying the main tenets of traditional essentialism. Accordingly, Reznek correctly argues that the essentialist is going to have to dig in his heels and absurdly insist that the individual had hypertension all along, though much less of the disease!

What the above shows is that insofar as current medical practice is correct to insist that some diseases differ only in degree from health, then one can advance a very persuasive argument that diseases do not constitute a natural kind of the essentialist sort. That bacterial infections, fungal infections, structural abnormalities and the death of the body's own cells are all explanatory causes of diseases *and necessary features of some healthy conditions* provides further reason to think that diseases do not in principle constitute a natural kind of the sort traditional essentialism describes. With this in mind, I will now draw one final arrow from the quiver.

4.3 Whether some state is to be counted a diseased state is context-dependent

Many uncontroversial disease-states are to be classed as disease only on grounds that inextricably involve one or more context-dependent variables. Undeniably, whether or not a condition is a disease condition depends upon how the organism actually functions. It is significant that the authoritative *Cecil Textbook of Medicine* does not identify any conditions that affect the function of the appendix as diseases.⁵⁴ Nor does it identify the

⁵⁴ Lee Goldman & Dennis Ausiello, (Eds.) (2007). *Cecil Textbook of Medicine* (23rd ed.). Philadelphia: Saunders. It does, of course, list acute appendicitis (which it refers to as inflammation of the appendix) and

presence of Heartworm (*Dirofilaria immitis*) as a disease. Instead, it merely mentions that *dirofilaria immitis* can infect humans. This is because although *dirofilaria immitis* can infect any dog—and causes severe disease even death in dogs—it is only under very rare circumstances that humans are infected. In the unusual event that one is, the majority of infected humans are asymptomatic. In the few symptomatic cases, the most common symptom is cough.⁵⁵ Thus, heartworm disease is not a human disease crucially because of the way the human body functions.

Equally clear is that the converse is true. Many common nonhuman zoonotic diseases—including rabies, ringworm, Lyme disease, tuberculosis, and H5N1 avian influenza—are identified as human diseases because each is pathogenic, given how the human body currently functions.

4.3.1 The “vestigial organ” (VO) argument

Clearly, how the human body currently functions is not how it always did. In addition to the vermiform appendix, Darwin and others have identified Jacobson’s Organ, “junk” DNA, *cutis anserina* (goose bumps), *plica semilunaris* (third eyelid), ear muscles, wisdom teeth, and pharyngeal arches in human embryos as vestigial organs.⁵⁶ The issue of whether or not these so-called vestigial organs have a function turns out to be very

pathological conditions, including carcinoid tumors, which may occur in the appendix.

⁵⁵ John W. McCall, Claudio Genchi, Laura H. Kramer, Jorge Guerrero, & Luigi Venco (2000), “Heartworm disease in animals and humans”, *Advances in Parasitology*, 66: 193-285.

⁵⁶ Charles Darwin. (1872). *The Origin of species*. 6th ed. London: John Murray, and (1874). *The descent of man and selection in relation to sex*. 2nd ed. Chicago: Rand, McNally Co. Richard Dawkins (2009), *The greatest show on earth*. New York: Free Press. G.B. Muller (2002), “Vestigial organs and structures”, in Mark Pagel, (Ed.), *Encyclopedia of evolution*. New York: Oxford University Press, 1131-1133.

complicated.⁵⁷ Regardless of which side of the debate one falls on, all probably should accept that these so-called vestigial organs no longer function as they once did. And if one accepts this, then one implicitly accepts that the human body does not currently function as it always has.

Indeed, for our present purposes, the relevant conclusion to be drawn from what I call the “vestigial organ” (VO) argument is that how the human body currently functions is not how it always did. Thus, it is not unreasonable to think that what was once a human disease may no longer reduce relevant human functioning. Conversely, what is now a human disease may not have reduced human functioning in the past. If this is correct, then the essentialist tenet (ii*) which, I have suggested, implies an ontological commitment to a common set of natural properties (*E*) that is intrinsic to all and only diseases appears misguided. For, it may be the case that the human body has changed such that every *S* with *E* is no longer, in fact, of kind *D*.

4.3.2 The “evolutionary change” (EC) argument

What I hope to show with the “evolutionary change” (EC) argument is that the force of the (VO) argument can be extended further because the human body did not merely change—it *is* changing. Richard Dawkins nicely emphasizes this point when he says:

⁵⁷ S.R. Scadding nicely describes a deep problem when he says: “The ‘vestigial organ’ argument uses as a premise the assertion that the organ in question has no function. There is no way however, in which this negative assertion can be arrived at scientifically. That is, one can not prove that something does not exist (in this case a certain function), since of course if it does not exist one cannot observe it, and therefore one can say nothing about it scientifically.” See, S.R. Scadding (1981), “Do ‘vestigial organs’ provide evidence for evolution?”, *Evolutionary Theory* 5:173-176, at 175. For some arguments suggesting the some of the so-called vestigial organs do have functions see, for example, Faye Flam (1994), “Hints of a language in junk DNA”, *Science* 226: 1320 and Alyia Zahid (2004), “The vermiform appendix: Not a useless organ”, *Journal of the College of Physicians and Surgeons*, 14: 256-258.

You cannot separate out the obvious changes in the body and treat them in isolation. To say that there are ramifications of every change is an understatement. There are hundreds, thousands of ramifications, and ramifications of ramifications. Natural selection is forever tweaking, adjusting the trim, “tinkering” as the great French molecular biologist François Jacob put it.⁵⁸

One of these ramifications of the changes the human body undergoes is whether something will be pathogenic and, as such, a human disease. It is not unreasonable to think, for example, that at one time there may have been a parasitic roundworm that caused death in humans by attacking the functioning of the appendix. But now, because of changes in the human body, insofar as it would be asymptomatic it has more in common with *dirofilaria immitis*. In that case, it would not be a human disease.

I concede this is a hypothetical scenario. Yet it finds its plausibility in the reasoning advanced by Dawkins and other evolutionary biologists that natural selection does not adhere to any “essentialist” species design.⁵⁹ Indeed, there is a considerable mass of evidence in support of Darwinian evolutionary theory of health and disease.⁶⁰ Moreover, the extensive scientific evidence that the human species is constantly changing at both the phenotypic structure and the microphysical structure provides very good reason to think that there can be no guarantee that what was once a human disease always will be

⁵⁸ Richard Dawkins (2009), *op cit.*, p. 368.

⁵⁹ Richard Dawkins (1986), *The blind watchmaker: Why the evidence of evolution reveals a universe without design*, New York: W. W. Norton & Co. and Dawkins (2009), *op cit.* Indeed, one of the main arguments against traditional essentialism is that its view of species is incompatible with Darwinian theory. See, for instance, Okasha (2002), *op cit.*, p. 191; Wilson *et al.*, (2007), *op cit.*; Paul E. Griffiths (2002), “What is innateness?”, *Monist* 85: 70–85, at p. 72; and Mayr (1976), *op cit.*, p. 27.

⁶⁰ For lucid and persuasive discussion of the evidence for evolution see Dawkins (1986), *op cit.*, & (2009), *op cit.* For an interesting and deft discussion linking Darwinian evolutionary theory to health and disease see, Randolph M. Nesse and George C. Williams (1996), *Why we get sick: The new science of Darwinian medicine*. 1st ed. New York: Vintage Books.

pathogenic. Thus the important implication of the (EC) argument is that we must be open to the possibility that a present disease-state may not be one in the future.

To sum up: The (VO) argument contends that how the human species functions *has* changed. The (EC) argument contends that relevant physiological structures of the human species *is* changing. Taken together, the (VO) and (EC) arguments make for an excellent case to endorse Reznick's claim that it is the consequences, not some common underlying nature of states, that all and only disease-states have in virtue of which a state is diseased. And, *a fortiori*, that the consequences of disease-states are not independent of how the human body currently functions—a context-dependent variable—provides a *prima facie* clear case in which essentialist claims (i*-iii*) are wrongheaded.

Two key issues therefore arise: (1) the extent to which disease-states involve context-dependant variables, and (2) whether essentialism is compatible with (1). A proper perspective on both these issues can be had, I believe, by examining the impact the external environment necessarily has on what counts as a disease-state.

4.3.3 *Environmental factors*

(I) Evolutionary change revisited

There is another major reason for thinking that what is to count as a disease is context-dependent—the profound affect the external environment has on not only on how our body functions but also whether or not a condition is a disease.⁶¹

⁶¹ Randolph Nesse goes as far as to contend that exposure to novel environments cause the majority of health problems today. I think Nesse over-extends the role of novel environments though I certainly agree that novel environmental factors cause *many* health problems today. See, Randolph M. Nesse (2001), "On the difficulty of defining a disease: A Darwinian perspective", *Medicine, Health Care and Philosophy* 4: 37-46. In this article Nesse also gives an interesting list of possible evolutionary explanations for why the human

We saw above that whether or not conditions are human diseases depend upon how the human body currently functions. And it is widely accepted among modern biologists that both the organism's genotype and the external environment crucially influence how the body currently functions. To reflect this, Marion Blute aptly proposes that we update the familiar definition of evolution (as a change in gene frequencies in a population) with an ecological–evolutionary–developmental (eco-evo-devo) definition of natural selection to acknowledge the recent accumulation of new knowledge. Among other things, this definition emphasizes that evolutionary change “is always wrought by inductive and/or constructive causal interactions between ecology and morphology, physiology or behavior”.⁶²

For our purposes, what is to be stressed is that there is an overwhelming mass of evidence showing that environmental factors are of cardinal importance in explaining the development of the phenotypic structure.⁶³ For example, there is extensive evidence to associate carcinogenesis with environmental toxins, such as nickel and inorganic arsenic, upsetting DNA methylation.⁶⁴

Thus, recent advances in our understanding of phenotype plasticity provide compelling evidence to insist that the environment influences the onset and progression

body is not better.

⁶² Marion Blute (2008), “Is it time for an updated ‘Eco-Evo-Devo’ definition of evolution by natural selection?” *Spontaneous Generations: A Journal for the History and Philosophy of Science*, 2(1): 1-5.

⁶³ See, for example, Scott F. Gilbert & David Epel (2009), *Ecological developmental biology: Integrating epigenetics, medicine, and evolution*, Sunderland: Sinauer Associates; Denis Walsh (2008), “A commentary on Blute’s ‘updated definition’”, *Spontaneous Generations: A Journal for the History and Philosophy of Science*, 2(1): 6-10.; and Matteo Mameli (2005), “The inheritance of features”, *Biology and Philosophy* 20: 365–399.

⁶⁴ Jessica E. Sutherland & Max Costa (2003), “Epigenetics and the environment”, *Annals of the New York Academy of Sciences* 983: 151–160.

human disease. Andrew Feinberg contends it is becoming increasingly clear that environmentally triggered epigenetic change—information heritable during cell division without involving changes to the DNA code—largely regulates developmental processes.⁶⁵ He suggests that the link between the environment and disease extends beyond cancer:

Thus, common diseases may involve phenotypic variants with both genetic variation and environmentally triggered epigenetic change that modulates the effects of DNA sequence variation.⁶⁶

Insofar as this is correct, this implies that the environment will have a profound affect on *what is to be counted at time t as a disease*. To put the point slightly differently, a disease-state is a product of several variables including (a) function and (b) environment.

All this stands to seriously undermine the notion that disease admits of a natural kind account that is harmonious with traditional essentialism. To see this consider (i*) which implies, among other things, that the possession of E is sufficient for membership of kind D . Yet surely a common set of natural intrinsic properties can, in and of itself, be sufficient for a state S to be diseased only if the *external* environment does not have a profound affect on whether or not an S is properly counted to be of kind D (more on this point below).

The problem cuts deeper when we remember (iii*), which we saw claims: p is a defining property of kind D iff p is caused by E . Accordingly, countenancing traditional essentialism's most fundamental claim that E is an intrinsic set of natural properties clearly

⁶⁵ Andrew P. Feinberg (2007), "Phenotypic plasticity and the epigenetics of human disease", *Nature* 447: 433-440.

⁶⁶ *Ibid.*, at p. 438. This view that a wide range of diseases have been linked to an upset in DNA methylation is echoed more recently in, Martin Hirst & Marco A. Marra (2009), "Epigenetics and human disease", *The International Journal of Biochemistry & Cell Biology* 41: 136-146, at p. 136-137.

forces the essentialist to deny that the external environment is a defining property of any *S* of kind *D*. That is, it forces the essentialist to deny a fundamental mechanism of modern evolutionary theory, that an organism's phenotype is determined by both its genotype and the external environment.

To go against what is surely leading claim of modern science is simply far too high a price to pay for the naturalist seeking to advance a value-free *scientific* account of health and disease. Thus if we are correct to insist that states are not diseases independent of environment, then it appears we have a conceptual argument showing that traditional essentialism concerning natural kinds is incompatible with naturalism concerning health and disease. This section will conclude by advancing two examples and a thought experiment showing that some uncontroversial disease-states are in fact context-dependant and, as such, that the above antecedent is in fact true.

(II) States are not diseases independent of environmental factors: Two examples and a thought experiment

Finally, and perhaps most importantly, I would like to suggest a more direct effect the environment has on whether a condition counts as a human disease. The upshot is that disease is context-dependent because many current diseases would not be diseases given a different external environment. The problem this poses for the essentialist is as follows: If whether a state is a human disease is not independent of the individual's environment, then the essential properties of the state will not be in and of itself a disease. That is to say, at least two main essentialist tenets (i* and ii*) are mistaken because it is *not* the case that diseases are categorically distinct from non-diseases in virtue of a unique set of natural

properties *E*. To see this, consider the following two examples and a thought experiment.

Example #1: Consider that it has recently been reported that in Canada, the rate of type II diabetes mellitus in Aboriginal Peoples—First Nations & Inuit—is 3 to 5 times higher than that of non-Aboriginal Canadians and rates are significantly increasing among the Inuit.⁶⁷ The rapid rise of the prevalence of type II diabetes mellitus in Aboriginal populations has been attributed to the adoption of a “Westernized”—i.e., high calorie intake and low exercise—lifestyle. James Neel advances the most popular evolutionary theory for the genetic susceptibility of Aboriginals for diabetes with his “thrifty genotype” hypothesis.⁶⁸ Neel states that “thrifty” is used “in the sense of being exceptionally efficient in the intake and/or utilization of food”.⁶⁹

Neel postulates that the exceptionally efficient phenotype was adaptive in the “feast or famine” environments of hunters and gatherers. Because our current “westernized” environment has become one of “feast” but not famine these thrifty genotypes have become detrimental, leading to the phenotype of diabetes. Neel thus contends that “[t]he

⁶⁷ Health Council of Canada (2005), *The health status of Canada’s First Nations, Metis and Inuit Peoples*. Interestingly, and relatedly, according to Janet Smylie & Paul Adomako (Eds.), (2009), *Indigenous children’s health report: Health assessment in action*. Toronto: The Centre for Research on Inner City Health, the obesity rate for First Nations children living on reservations is 36%, compared to 8% for Canadian children overall.

⁶⁸ For authors stating this view, see, for instance, Catherine H.Y. Yu & Bernard Zinman (2007), “Type 2 diabetes and impaired glucose tolerance in aboriginal populations: A global perspective”, *Diabetes Research and Clinical Practice* 78: 159–170; Arya M. Sharma (1998), “The thrifty-genotype hypothesis and its implications for the study of complex genetic disorders in man”, *Journal of Molecular Medicine* 76(8): 568–71; and Jared Diamond (2007), “The double puzzle of diabetes”, *Nature* 423: 599–602.

⁶⁹ James V. Neel (1962), “Diabetes Mellitus: A “thrifty” genotype rendered detrimental by “progress”?”, *The American Journal of Human Genetics* 14: 353–362, at p. 354.

changing dietary patterns of Western civilization had compromised a complex homeostatic mechanism”.⁷⁰ According to the “thrifty genotype” hypothesis proposed by Neel, the prevalent and significant disease of type II diabetes mellitus is crucially the result of *both* genetic and environmental factors. Insofar as this is correct, disease is context-dependent.

Neel is surely correct to think that a disease-state can be the result of a mismatch between “normal” functioning and a novel environment.⁷¹ If we accept this conclusion concerning disease-states, we should notice that such a view is incompatible with essentialism. This is because we are explaining the change in disease-status without the appeal to any intrinsic change in the essential properties of the relevant state. But if this is so, then we seem to be denying that there exists a common set of natural properties (*E*) that is intrinsic to all and only diseases—and, as such, we are rejecting the main tenets of essentialism.

Example #2: Indeed evolutionary theory strongly advances the notion that diseases are the product of the complex interplay of genetic and environmental factors. To further stress this important point, consider the autosomal recessive blood disease α -Thalassemia. For our purposes, what is striking about this disease is not that it has been identified as one of the most common single gene disorders in humans but that its prevalence is almost

⁷⁰ James V. Neel (1999), “The “thrifty genotype” in 1998”, *Nutrition Reviews* 75: S2-S9, at S2.

⁷¹ Insofar as this correct, then it would seem reasonable to think that a health-state could also be the result from abnormal functioning and a novel environment. To illustrate, Edmond A. Murphy proposes that a polycythemia (ICD classifies as D45) which would be abnormal and can be dangerous at sea level may be live-saving for those who inhabit very high altitudes. See Edmond A. Murphy (1973), “The normal”, *American Journal of Epidemiology* 98(6): 403-411, at p.409.

exclusively limited to tropical and subtropical regions of the world.⁷² Further still, there is extensive microepidemiological evidence that strongly supports the notion that malaria is the selective agent for the strikingly high prevalence of α -Thalassemia in tropical environments.⁷³

And what is more, there is another very strong reason to think that α -Thalassemia comes about because of the environment. This is based on extensive surveys have shown that the prevalence of the disease is less than 0.01 in Britain, Iceland, and Japan (environments historically free of malaria).⁷⁴ Thus, it is claimed that in environments where malaria is endemic (as is the case in the tropic and subtropical regions of the world) the malaria resistant genetic disease α -Thalassemia confers a selective advantage. Insofar as diseases are dependent upon environmental factors, disease is context-dependent.

A Thought Experiment: Type II diabetes mellitus and α -Thalassemia are two diseases that persuasively demonstrate that states are not to be counted as diseases independently of consideration of environmental factors. Because environmental factors are context-dependent variables, such a state will not be in and of itself a disease. To see how a notable disease would surely not be a disease-state given a different environment let us consider one final theoretical argument.

⁷² DR Higgs, MA Vickers, AO Wilkie, IM Pretorius, AP Jarman *et al.* (1989), "A review of the molecular genetics of the human alpha-globin gene cluster", *Blood: The Journal of the American Society of Hematology* 73(5): 1081-1104.

⁷³ Adrian V.S. Hill (1992), "Malaria resistance genes: a natural selection", *Transactions of the Royal Society of Tropical Medicine and Hygiene* 86: 225-226, 232. J. Flint, A. V. S. Hill, D. K. Bowden, S. J. Oppenheimer, *et al.*, (1986), "High frequencies of α -thalassaemia are the result of natural selection by malaria", *Nature* 321: 744-750.

⁷⁴ DR Higgs, *et al.*, (1989), *op cit.*, at p. 1094.

To illustrate, take the uncontroversial infectious disease malaria. To borrow a thought experiment from Hilary Putnam,⁷⁵ imagine Twin Earth that is exactly like Earth except for on Twin Earth malaria is a pandemic, mosquitoes are in abundance, and there is a complete lack of low-oxygen environments. Given these stipulations, it seems likely that on Twin-Earth the *absence* of the Sickle cell trait would be pathological. Indeed, instances appear all over where, *mutatis mutandis*, exactly the same point can be made:

Environmental factors show a condition not to be in and of itself a disease. Rene Dubos proposes a similar conclusion when he claims:

Overwhelming evidence indicates [that] many forms of disease have emerged or have been disseminated in the modern world because our ways of life have created new and complex constellations of circumstances favorable for their spread.⁷⁶

Dubos, we saw above, is echoed more recently by scientists, such as Feinberg and, Hirst and Marra, who firmly insist that epigenetics and human disease are linked. Indeed, as we shall come to see in the next chapter many reasonable people, including Boorse, insists that *homeostasis*—a concept that reflects that the internal environment of an organism is often affected by changing external environments—is a necessary feature of a proper understanding of health and disease.

What all this illustrates is that the essentialist appears, in principle, mistaken that

⁷⁵ Hilary Putnam (1973), “Meaning and reference”, *The Journal of Philosophy* 70: 699-711. It is perhaps worth mentioning here that Colin McGinn identifies 9 characteristics of natural-kind terms from the literature, of which (v) is: we can construct plausible “Twin earth” cases for natural kind terms. See Colin McGinn (1991), *The problem of consciousness*. Oxford: Blackwell, p. 156.

⁷⁶ Rene Dubos quoted in William Bechtel (1985), “In defense of a naturalistic concept of health”, In J. Humber & R. Almeder (Eds.), *Biomedical ethics reviews*, Clifton, NJ: Humana, 131-170, at p. 145.

there is some underlying natural property⁷⁷ peculiar to all diseases that is *ipso facto* lacked by all non-diseases.

5. Chapter Conclusion

In sum, I have acknowledged that a great deal hangs on whether disease is a natural kind. I also have acknowledged that one should hold little hope for a convincing knockdown objection against the essentialist view that diseases are a natural kind. What does this mean for the naturalist seeking to advance a more promising account of health and disease? I believe a proper response is guided by Elliot Sober's apposite words:

It is pie-in-the-sky metaphysics and science to hold on to some guiding principle simply because *it is possible* that there might be some substantive formulation and development of it.⁷⁸

Indeed, we should not fail to recognize that whether or not a naturalist concerning health and disease can employ an essentialist view of disease as a natural kind must surely live or die on empirical merit.

As matters stand now, however, since (i) it is true that the consequences of a state often determine its disease-status; (ii) there is overwhelming empirical evidence that

⁷⁷ To be sure, just what sort of "essential" natural properties are involved here is controversial. The standard way to explicate the requisite "essential" properties has been in the terminology of intrinsic vs. extrinsic properties. This terminology, however, has led to further distinctions. Some identify an essential/accidental distinction, others what has been called an Ellis-intrinsic/Ellis-extrinsic distinction, while others a non-relational/relational distinction, and still others distinguish between properties that are purely qualitative or not. I will not pursue this matter here. For my purposes it suffices to show—regardless of the most deserving distinction—that all genuine members of the natural kind disease do not share common essential properties of the sort traditional essentialism avers. For further discussion of these distinctions and more see, I. L. Humberstone (1996), "Intrinsic/Extrinsic", *Synthese* 108: 205-267.

⁷⁸ Elliott Sober (1980), "Evolution, population thinking and essentialism", *Philosophy of Science*, 47: 350-383, at p. 353.

human disease is context-dependent because two ineliminable contributing features of human pathology are surely current human functioning and the external environment; and (iii) our best scientific evidence has failed to identify a distinctive natural property that would confirm disease *qua* disease is a natural kind, I conclude that there are several good reasons to reject the essentialist view that *all and only* diseases possess a common set of “essential” underlying natural properties.

That such a view runs against current medical practice’s understanding of disease in terms of degree and risk provides a further, and I think, a decisive reason for abandoning the notion that a promising value- and norm-independent account of health and disease lies in treating disease as a *natural kind* and characterizing disease-states solely in terms of their distinct *essential* natural properties. And if this is correct, then “natural kinds” will not mark the requisite value- and norm-independent distinction between health and disease.

For the naturalist, the proper conclusion therefore seems clear: a more promising value-free scientific account of health and disease cannot be justified by the dubious essentialist view that disease *qua* disease is a natural kind fixed by a set of common underlying so-called “essential” natural properties. The Cambridge-change objection therefore still stands.

Chapter Five

“Normality”

In medicine, particularly, beliefs about what is normal are central in making judgments about disease.

— Phillip V. Davis and John G. Bradley¹

1. Introduction

The rejection of the view that diseases constitute a natural kind and are identifiable in virtue of a shared distinct essence may reasonably motivate some defenders of the BST to find a different way of avoiding Cambridge-change objections. Hence, it might be suggested that this could be done by taking up a different account of what should be counted as normal human functioning and, as such, by working with a different account of what is to count as *normal*. The task of this chapter will be to investigate whether the naturalist can bring to bear a more appropriate conception of what is to be counted as *normal* human functioning and more specifically whether such a conception could adequately serve to underpin a value- and norm-independent account of health and disease.

To that end, the chapter will proceed as follows. To situate the discussion, I begin with a quick review of three different conceptions of normality. A biological conception of normality quickly emerges as the most attractive candidate for the naturalist aiming to employ an account of *normal* human functioning that stands a chance of avoiding Cambridge-change objections. Given the naturalist’s need for a value- and norm-

¹ Phillip V. Davis & John G. Bradley (1996), “The meaning of normal”, *Perspectives in Biology and Medicine* 40(1): 68-77, at p. 68.

independent account of health and disease, two noteworthy ways of formulating a biological concept of normality are examined, but both are rejected. Along the way, I will bring to light some significant obstacles that stand in the way of advancing a suitable statistics-free, naturalist conception of what is to be counted as *normal* human functioning. As we shall come to see, these are problems that will undermine the naturalist's claim, in general, and the BST's claim, in particular, that it is possible to provide medicine with a principled value- and norm-independent account of health and disease.

2. Three conceptions of normality

Robert Wachbroit provides a solid starting point to enter into our discussion, for he provides useful characterizations of three distinctly different conceptions of normality:

- (i) a statistical conception
- (ii) a biological conception
- (iii) an evaluative conception².

What is notable about the BST is that it intertwines *both* statistical *and* biological concepts of normality to underpin its normal-pathological contrast, a contrast fundamental to its distinction between health and disease.³ I have argued that the upshot of my discussions of the current framework of the BST's attempt to avoid appeals to values and

² Robert Wachbroit (1994), "Normality as a biological concept", *Philosophy of Science* 61: 579-591.

³ In order to capture the robust extension of disease, Boorse insists that a normal versus pathological distinction be employed to underpin any account of health and disease. Furthermore, this distinction, Boorse claims, is the basic theoretical concept of Western medicine. See Boorse (1987), pp. 364-65 and (1997), p. 7. In Boorse (1977), p. 546 at fn 3, he says: "The pathology of a disease is its morbid anatomy, i.e. the structural changes in body tissues that underlie its signs and symptoms. 'Pathological', however, can be a synonym for 'diseased' and 'abnormal'. The two usages are related by the medical assumption that every disease has some pathology, known or unknown".

norms by defining “normal” human function and functioning (biological fitness) entirely in terms of statistical data (in other words, by adopting a statistical conception of normality), is that this approach seems to leave the BST open to Cambridge-change objections.

To avoid facing Cambridge-change objections, the defender of the BST may seek to remove the fundamental role assigned to the use of statistical data of reference classes in its specification of what is to count as a *normal* function. After all, I have argued that the Cambridge-change objection gets bite because of the ever-present possibility of change in what *are* the statistical facts about reference classes. I concede that it might be possible to do this, especially if a biological conception of normality were adopted. However, I will argue such attempts to abandon a statistical conception of normality create their own more serious problems.

2.1 *Evaluative normality*

I shall begin with the third of Wachbroit’s conceptions of normality—the evaluative conception—because I think it can be rather easily and forcefully dismissed if it is remembered what those trying to develop a naturalistic account of health and disease are trying to do. To see this, consider what an evaluative conception of normality might look like when we interpret it in a way that incorporates values or norms into the account.

Presumably the idea of an evaluative conception of normality would be to say something like the following: That the behavior of individual *I* is *normal* would be to say that pattern of *I*’s behaviour is consistent with or fulfills what is to be properly counted as “normal” because it is fixed as such by religious norms, or by cultural norms, or by

institutional norms, or by other kinds of conventional norms, or perhaps by ethical norms.⁴

But now it should be completely obvious that, if this *is* what is intended in an “evaluative” conception of normality, then it will simply not do for the purposes of someone hoping to use the notion of normality in a naturalistic account of health or disease. A naturalistic account of health and disease, I argued in Chapters 1 and 2, is an account which makes health and disease directly and only dependent on facts that count as being objective on the criteria I spelled out there. That is, facts that obtain independently of people’s beliefs about them and attitudes to them and, indeed, to use the Anscombe/Searle terminology I described in Chapter 1, on facts that are to be counted as “brute” facts rather than facts which obtain only “by human agreement” as Searle put it in his 2010 *Making the social world*.⁵ But clearly if the notion of normality used in the naturalistic account of health and disease was given the kind of analysis set out in the evaluative conception of normality as described in my last paragraph, then normality would *not* be amenable to analysis as an objective state of the requisite sort.

For our purposes, it will suffice to note that evaluative conception of normality is held to be crucially different from the statistical and biological conceptions of normality in that the norms which define the normality are as we might put it, created norms rather than discovered norms as they seem to be on the other two conceptions.⁶ The point to press is

⁴ See Wachbroit (1994), *op cit.*, at p. 580.

⁵ Searle (2010), *op cit.*, p.10.

⁶ I realize that some may object on religious grounds that we don’t create religious norms: they are at most created by god or they reflected the natural order ordained by god. I will only say here that I find such objections thoroughly unconvincing for the reason that any legitimate appeal to an objective norm or value

that the adoption of the norms cited to confer legitimacy upon the claim “the behaviour of *I* is normal” is supposed to involve the adoption of created norms that are conceived as being “created” in the sense that their adoption is not entirely (i.e., without remainder) justified by appeal to claims which are exclusively claims about purely value-independent physiology states.

Now what all of this means is that what is to count as a “normal” function and what is to count as a “normal” functional ability (see clauses (D2) and (D3) of Boorse’s official definitional schema⁷) becomes, on this account of normality, in a very straightforward and obvious sense determined by those values or goals guiding the choice of norms which the people adopting the norms might have.

In light of this, it becomes clear that the defender of the BST cannot retreat to invoke an evaluative conception of normality of this kind. After all, the whole point of the development of the BST was exactly to avoid the need to invoke such norms and to legitimately produce a value- and norm-independent conception of health and disease.

that is logically independent of human beings would seem to square off against Mackie’s (1977), *op cit.*, well-argued and forceful challenge to moral realism known as “the argument from queerness”. I construe Mackie’s attack to be against the thesis, roughly put, that there exist moral facts (or principles) and these moral facts (or principles) are logically independent of human beings. Here I merely mention Mackie’s argument as something I think is persuasive, but I don’t have the time or space to argue here. At any rate, given the scope of this chapter, I see no reason to even hang one’s hat on Mackie’s much discussed and debated argument against moral realism unless and until a philosophically strong case could be made that such “objective” norms would be compatible with naturalist scruples (i.e., make the classification of states as healthy or diseased be a value- and norm independent objective matter); See Boorse (1997), p. 4.

⁷ For ease of reference they are: (D2) A *normal function* of a part or process within members of the reference class is a statistically typical contribution by it to their *individual* survival and reproduction; and (D3) A *disease* is a type of internal state which is either an impairment of normal functional ability, i.e. a reduction of one or more functional abilities below typical efficiency, or a limitation on functional ability caused by environmental agents.

2.2 Statistical normality

The concept of statistical normality is not new. Over the last hundred or more years it has been both familiar and influential. In 1835, the statistician Adolphe Quetelet first employed statistics (and notably a reference to mean values) to arrive at what he claimed was a real, objective “average man” (l’homme moyen).⁸ Moreover the idea that we might be able to develop an account of what is normal in terms of what is “natural” and explicating that statistically is also not new. Since Quetelet’s “average man”, the classification of “normal” physiological characteristics has almost universally been in terms of the so-called “natural” distributions of these physiological characteristics.⁹

Boorse, of course, continues this tradition. He has attempted to refine the idea that the normal is the natural by defining it in terms of a statistical conception of normality, which he then links to the modern notion of “uniform functional design” (in places he uses the phrase “species design”). More specifically, as I outlined in Section 1 of my Chapter 3, Boorse’s suggestion about the way in which the notion of uniform functional design is to be taken as playing a rôle in his theory can be taken as in effect coming to the following:

- (i) that we can define *functional design* of some species S as that “the internal functional organization” of members of the species S which is *statistically typical* of the internal functional organization of the members of the species S;
- (ii) that we then say that the internal functional organization of some member

⁸ Adolphe Quetelet (1842), *A treatise on man and the development of his faculties*. New York: Burt Franklin, Reprinted in 1968. See also, Brian P. Cooper and Margueritte S. Murphy (2000), “The death of the author at the birth of social science: The cases of Harriet Martineau and Adolphe Quetelet”, *Studies in the History and Philosophy of Science* 31: 1-36.

⁹ Jirí Vácha (1978), “Biology and the problem of normality”, *Scientia* 72: 823-846.

of species *S* is *normal* if and only if the contributions that the internal functional organization in question makes to the survival and reproduction success of the *individual* members of the reference class counts as being *statistically typical*;

- (iii) that we then count as a *uniform* configuration of the internal functional organization of some member of species *S* a configuration which is *normal* by clause (ii).¹⁰

Set out this way, it becomes very clear, as I argued in Section 1 of Chapter 3, that a statistical conception of normality is playing a *fundamental* rôle in Boorse's account of what is to count as the uniform functional design of an organism, of what internal functional design is *normal* in a given species, of what is to count as *normal* functioning of that internal functional design, and of what is to be counted as *natural*. Hence, where others assign a central rôle to the evolutionary history of some functional design in their accounts of how to pick out what counts as normal functional design, Boorse relies at least very heavily on what is statistically typical and minimally, despite some hesitations (see below), he claims that references to what is statistically typical cannot be eliminated from an account of normal functional design (i.e., species design).¹¹

It is worth noting that Boorse is not as optimistic as Quetelet was about the role statistics can play in constructing a value- and norm-independent conception of the normal, at least when it comes to health. For Boorse explicitly concedes at one place that

¹⁰ See my (D*) in Section 1 of Chapter 3.

¹¹ Boorse, in a nutshell, understands species design to be a naturalist conception which he states is “the internal functional organization typical of species members, which...forms the subject matter of physiology...” See, Boorse (1997), p. 7 and footnote #72 below where this quote is given in its entirety. Here Boorse can be seen as building upon C. Daley King's famous proposal of “normal” as “that which functions in accordance with its inherent design, i.e., a pattern norm”. C. Daley King (1945), “The meaning of normal”, *Yale Journal of Biology and Medicine*, 17: 493- 501, at 500.

the view that statistical normality is either a necessary or a sufficient condition of health needs qualification. He says:

It cannot be necessary because unusual conditions, e.g., type O blood or red hair, may be perfectly healthy. It cannot be sufficient because unhealthy conditions may be typical. No doubt the average person or organ is healthy in a practical sense of displaying no indications for treatment, but that is not the same as complete freedom from disease. Some of what medical texts consider disease processes are at work in virtually everyone below the level of clinical detection. There are also particular diseases-atherosclerosis, minor lung inflammation, perhaps tooth decay-that are nearly universal.¹²

Nevertheless, Boorse then goes on to say quite strikingly:

In spite of these difficulties we will give statistical normality an important role in our view, which shows that necessary and sufficient conditions are not the only possible components of an analysis.¹³

Like Boorse, as I will explain later, I believe that the most promising naturalist account of health will not abandon the view that normality, at least concerning health and disease, is at some *essential* level a statistical notion.¹⁴ Hence, in my view attempts to spell out a theory of normality and especially a theory of normal function and normal functioning, which either gives overriding pride of place to the evolutionary history of the

¹² Boorse (1977), pp. 546-47. This same point is both forcefully and colourfully made by C. Daly King: "A million cripples do not, by their mere number, acquire soundness nor does the presence of a majority of lunatics in a given population make insanity normal, as any director of a lunatic asylum will testify." See King (1945), *op cit.*, at 494.

¹³ Boorse (1977), p. 547.

¹⁴ There is the strong intuition that "normal aging" is not a disease, which nicely dovetails with the view that disease is a deviation from (some sense of) normal functioning. And Scadding, for example, argues persuasively that disease is phenomena that differs from the norm of the species such that it places the individual at a "biological disadvantage". See J. G. Scadding (1967), "Diagnosis: The clinician and the computer", *The Lancet*, 2: 877-882. A compelling case is made for Scadding's view in R. E. Kendell (1975), "The concept of disease and its implications for psychiatry", *British Journal of Psychiatry*, 127: 305-315. At any rate, Boorse is correct to point out that "[t]extbook normals for clinical variables like height, weight, pulse and respiration, blood pressure, vital capacity, basal metabolism, sedimentation rate, and so on are certainly statistical means surrounded by some range of "normal variation". See, Boorse (1977), p. 546.

organism's functional ability to enhance its past, present or future, biological fitness;¹⁵ or which gives overriding pride of place to its possible physical or chemical causal contributions to the capacities under consideration of that organism¹⁶ will of necessity be in some important sense incomplete.

2.3 *Biological normality*

Wachbroit suggests, negatively, that a *biological* account of normality will be something different from a statistical or an evaluative account, but how *positively* he would explicate that notion is perhaps rather less clear. In Section 3 of his paper he examines and rejects the suggestion that a generically biological account of normality would be one in which the concept of biological normality might be defined or explicated in terms of claims about the biological functions, presumably functions of structures or organs or organisms or perhaps of the processes which take place in such structures, organs, or organisms. He cites Boorse (1977) and King (1945) as exponents of this view. But he argues against this suggestion, claiming, "that accounts of biological functions cannot *explain* the concept of

¹⁵ See, for instance, Wright (1973), *op cit.*; Millikan (1984), *op cit.*; Neander (1991), *op cit.*; Peter Godfrey-Smith (1994), "A modern history theory of functions" *Noûs*, 28(3): 344–362.; and John Bigelow & Robert Pargetter (1987), "Functions" *The Journal of Philosophy*, 84(4): 181–196.

¹⁶ See perhaps most strikingly, of course, Robert Cummins (1975) "Functional analysis" *The Journal of Philosophy*, 72, 741–76, especially p. 762; Ulrich Krohs, in his (2009), "Functions as based on a concept of general design" *Synthese* 166: 69–89, rightly points out that even those who move in significant ways away from Cummins' views nevertheless view Cummins' core ideas as fundamental to their different approaches. Krohs cites Paul E. Griffiths (1993), "Functional analysis and proper functions", *British Journal for the Philosophy of Science*, 44: 409–422, Ron Amundson & George V. Lauder (1994), "Function without purpose: The use of causal role function in evolutionary biology", *Biology and Philosophy*, 9(4): 443–469; Denis M. Walsh & André Ariew (1996), "A taxonomy of functions" *Canadian Journal of Philosophy*, 26(4): 493–514; Peter McLaughlin (2001) *What functions explain: Functional explanation and self-reproducing systems*. Cambridge: Cambridge University Press, pp. 113–114; Tim Lewens (2004), *Organisms and artifacts: Design in nature and elsewhere*. Cambridge: The MIT Press, pp. 136–138.

biological normality because they *presuppose* it”¹⁷ and suggesting that consequently some account of biological normality is needed which proceeds in some rather different way. His suggestion is that the biological notion of normality is “...allied with the contrast between function and malfunction”.¹⁸ His specific suggestion is this:

The biological sciences use the [following] explanatory strategy. A biological system is decomposed into an unperturbed state (in this case the normal state as characterized by physiological theory) and perturbations (deviations from the normal state or abnormalities). For example, let us consider physiological explanations of cardiac contractions. One important feature of a normal heart is that it undergoes rhythmic contractions. An actual heart, however, may exhibit a more complicated series of contractions due to stimulation from caffeine or nicotine, lack of sleep, anxiety, or damage to the cardiac muscles. The physiologist characterizes complicated contractions by, in effect, decomposing the movements into a normal rhythmic motion plus an arrhythmic motion. This second term may itself be decomposed into a series of motions, including low-frequency (flutter) and high-frequency (fibrillation) contractions. If this second term is sufficiently small, the cardiac contractions are normal to a first approximation; if greater accuracy is needed (e.g., we want to understand how this heart is malfunctioning), then the other terms are brought into account. Thus, the decomposition not only allows us to characterize a complex cardiac motion but also enables us to explain in what its abnormality consists.¹⁹

Now this explication is all very interesting and perhaps very plausible, but as an explication of what marks a biological conception of normality it is perhaps less than ideal at least for our purposes. For what we would want is a characterization of what demarcates the *generic* category of *biological* conceptions of normality from other kinds of conception of normality. For example, in the paragraph immediately following the above paragraph, Wachbroit continues:

¹⁷ Wachbroit (1994), *op cit.*, p. 581. Italics his.

¹⁸ Ibid., p. 587. See also, pp. 580-581.

¹⁹ Ibid., pp. 589-590.

The explanatory strategy employed in physiological explanations differs from the one that Sober (1980) argues is employed in evolutionary theory. According to Sober, the Darwinian rejection of typological thinking for a population perspective can be understood as a rejection of the above strategy where the aim is to explain deviations or variations from some "unperturbed" state. With variation the rule, the aim of evolutionary explanations is to explain the *constraints on* variation rather than the *presence of* variation.²⁰

In my view, a characterization of the *family* of biological accounts of normality would best be one that did not presuppose any *particular* biological explanatory strategy. Obviously providing such a characterization transcends the scope of this dissertation, but I do think that a very rough sketch may bring some useful clarity into the discussion. Given my present purposes, I suspect that the following crude characterization will suffice:

A biological account of the notion of normality will be one in which (i) reference is essentially made to states, properties (including capacities), or actual or possible activities of entities which are, were or will be in some sense *living* entities; (ii) reference is made to such entities' *internal functional organization* (presumably a physiological structure) and its actual or possible activities and capacities; (iii) what is to be counted as (a) normal states, properties and actual or possible activities of such entities, or (b) normal functional structures of such entities, or (c) normal functioning of such entities, is to be identified by methodologies that are suited to answering questions about entities or groups of entities that are, were, or will be in some sense alive.²¹

To be sure, the question of what the next step, if any, for this characterization ought to be is a very, very difficult question. And, moreover, the characterization is obviously crucially in danger of circularity. Nevertheless, it has the virtue of generality for it seems not, for example, to assume that the methodology will or will not be a function of facts about the evolutionary history of the entities, nor does it assume that the methodology will

²⁰ Ibid., p. 590. Italics his.

²¹ I owe this characterization to John A. Baker.

or will not necessarily reach into molecular or genetic structures of the components of such entities.

And yet the characterization is not empty since, of course, the questions of what is to count as a living entity's internal functional organization and what is to count as a methodology that is "suited" to answering questions about such entities are presumably not empty questions. After all, these are the sorts of questions that can and have received various different answers over the years, some of which have been shown to be wrongheaded answers. To see this, surely one has only to notice the radically different accounts of how best to characterize what is to count a function of some internal structure of some organism, some, as I said in a footnote earlier, giving overriding pride of place to the evolutionary history of the structure's ability to enhance biological fitness, some giving overriding pride of place to its possible causal contributions to the capacities under consideration of that structure (and not on its evolutionary history).

With this account of what is to count as a biological conception of normality the question we now face is whether it would be possible to find an account of normality that would fit this characterization but would avoid the need to invoke claims about what is statistically typical. To that question I now turn.

3. Does an adequate account of normality need to be at some level statistical?

Consider the biologist's claim that the function of the human heart is to circulate the blood. This claim refers to the normal heart. Yet, it is not about any specific heart of a given human; nor need the claim reflect a statistical norm or mean average. Indeed, the truth of the claim does not entail that there currently are human hearts circulating blood.

Suppose that a highly contagious virus renders all hearts unable to properly function, with the consequence that all surviving humans have artificial pumps circulating their blood. If this most unfortunate situation were to happen, then the function of the human heart would still, on the biological account, be to circulate the blood. What is to be stressed is that when the biologist speaks of the normal *qua* biological normal heart, he does so with the realization that some, most, or all hearts may in fact not be functioning “normally”.²² That is to say, the biological conception of normality is importantly distinct from actual or current functioning.

This suggests the possibility of avoiding a Cambridge-change objection by defining “normal” functions in terms of biological normality. Now suppose we could do away with the BST’s bio-statistical view of normality and replace it with a purely biological conception of normality—a conception, in other words, which counts as biological on the characterization I gave in the last section. Then there would be significant implications.

First, without recourse to statistics, objections against the viability of acquiring a suitable naturalistic account of normal human functioning from the biological facts of nature would seem to strike with newfound force.²³ Since I intend to show that one cannot expunge statistics and retain a persuasive naturalist account of health, I will not pursue this line here.

Second, if we abandon a statistical conception of normality we effectively strip the

²² Ibid., pp. 579-581.

²³ For criticisms directed against Boorse’s account of “normal function” see Amundson, (2000), *op cit.*, and Ereshefsky (2009a), *op cit.* For other more general objections against biology being endowed with functional categories of scientific importance see, for example, Amundson & Lauder (1994), *op cit.*, and Griffiths (1993), *op cit.*

BST of its specific account of normal functioning (i.e., what is identified as solutions to the quantitative issues about functions). After all, the BST still insists that health is the absence of disease and that disease is “a reduction of one or more functional abilities below typical efficiency” (D3). But as we saw in Chapter Three, the BST’s formulation of “typical efficiency” is crucially a statistical conception. It is the statistical norm of the relevant functions of the same species, sex and age at time *t* on species-typical occasions. Here “typical efficiency” is being used as a synonym for normal functioning. Thus, if “typical efficiency” is no longer a statistical norm, then the BST must employ another naturalist account of typical efficiency/normal functioning.

However, there is no decisive reason for thinking that a less problematic non-statistical naturalist account of *normal* function and, *a fortiori* normal functioning, is waiting in the wings. If normality is understood as biological normality as described above and suggested by Wachbroit, then the most it may tell us is what is a normal function (i.e., what is identified as solutions to the qualitative issues about functions). That is to say, such a conception of biological normality identifies the normal function of various biological organs and processes. For example, it would identify that the function of the human heart is to circulate the blood.

Such an understanding will correctly have the BST count any heart that does not circulate blood not to be normal and, as such, not to be healthy.²⁴ While I concede that the naturalist can plausibly claim that biological normality constitutes a necessary condition of

²⁴ It is worth mentioning here that insofar as biological normality may not speak to how human hearts, for example, *actually* are functioning this conception of normality would seem to run against Boorse’s goal-directed view of biological function. This point is discussed in detail below.

“normal” or “healthy” functioning, this is hardly sufficient to underpin a persuasive naturalist account of health and disease. Significantly lacking are principled grounds for not counting *every* level of functioning to be an instance of “normal” functioning. To put this point slightly differently, the naturalist is lacking a principled account of *quantitative* normal function and thus, is unable to specify which level of functioning is requisite for normal functioning to be successfully realized.

This is a serious shortcoming because medical practice does not, and surely given its clinical goals, *should not* count every heart that circulates blood to be healthy. We should therefore not expect the defender of the BST to say that an individual’s heart is normal so long as it *merely* circulates blood. As Boorse notes:

Now the most obvious logical feature of medical normality is that most functions have a normal range of values...there is a normal range of values around a mean, with either one or two pathological tails.²⁵

The defender of the BST must tackle this problem immediately. To that end, the challenge faced by naturalists is to specify the level of functioning (quantitative) that is requisite to count an individual to be functioning “normally” (or abnormally) and as such healthy (or diseased). And, *a fortiori*, the naturalist must do so without essential recourse to talk—explicit or otherwise—of what is statistically typical for as soon as that takes place the BST faces possible Cambridge-change objections. However, here an important problem arises: a biological conception of normality does not itself seem to specify the requisite range of actual functioning.

Let us therefore briefly examine two accounts of normality which clearly fit the

²⁵ Boorse (2002), at p. 101.

characterization of what is to count as a biological account of normality that I gave above, but which at least *prima facie* might seem not to involve essential reference to claims about what is statistically typical.

4. Normality and homeostasis

There is a long tradition in medicine of characterizing normal functioning accounts of health and disease in terms of notions like “balance” and “imbalance”, “equilibrium” and “lack of equilibrium”. The connection between health and equilibrium states can be traced at least as far back as Hippocrates who held that all human diseases arise because of one or more imbalances in the four humors.²⁶ Although we no longer link health with the balance in blood, phlegm, black and yellow bile, modern medicine upholds this tradition by identifying accounts of normal functioning and *a fortiori* health with the coordinated balance of “homeostatic” mechanisms. More than being consistent with the tradition, however, current medical practice actively promotes a conceptual link between normal biological function and homeostasis.²⁷ Indeed, one would be hard-pressed to find a recent medical textbook where accounts of vital biological functioning are not explained in terms of homeostatic mechanisms.

That naturalist conceptions of health and disease can do away with statistics by dovetailing their accounts of normal biological functioning with homeostasis is thus not

²⁶ Mark J. Schiefsky (2005), *Hippocrates on ancient medicine, translated with introduction and commentary by Mark J Schiefsky*, Leiden: Brill Academic Publishers.

²⁷ To illustrate, a search on the PubMed database for the terms “homeostasis” and “normal” brought up 18,184 matches. The terms “homeostasis” and “disease”, and “homeostasis” and “health” brought up 19,144 and 9,080 matches respectively. Search done April 8, 2010.

without some traction. Though, any temptation to think that these accounts make homeostasis sufficient for health would be seriously mistaken. Crucially, as will emerge, reference to claims about what is statistically typical is in the end essential.

Correspondingly, the aim for this section is to advance and critically defend the view that homeostasis *is not* sufficient for health. If sound, it follows that the model of homeostasis cannot serve as the sole underpinning for an adequate account of normal functioning. This provides further reason to think that the naturalist remains unable to explicate the requisite “normal state of the organism” without appealing to statistics.

4.1 *Cannon and Boorse on homeostasis*

The physiologist Walter B. Cannon coined the term “homeostasis” in 1926 and defined it as follows:

The highly developed living being is an open system having many relations to its surroundings— in the respiratory and alimentary tracts and through surface receptors, neuromuscular organs and bony levers. Changes in the surroundings excite reactions in this system, or affect it directly, so that internal disturbances of the system are produced. Such disturbances are normally kept within narrow limits, because automatic adjustments within the system are brought into action, and thereby wide oscillations are prevented and the internal conditions are held fairly constant...The coordinated physiological reactions which maintain most of the steady states in the body are so complex, and are so peculiar to the living organism, that it has been suggested (Cannon, 1926) that a specific designation for these states be employed—*homeostasis*.²⁸

Cannon’s choice of the Greek-derived prefix “homeo” (meaning “like” or

²⁸ Walter B. Cannon (1929), “Organization for physiological homeostasis”, *Physiological Reviews* 9(3): 399-431, at. p. 400. Also see, Cannon (1926), “Physiological regulation of normal states: some tentative postulates concerning biological homeostatics”, In: *Jubilee volume to Charles Richet*. Paris: Editions Medicales: 91-93.

“similar”) rather than “homo” (meaning “same” or “fixed”) was to emphasize the point that the stabilization of bodily states was a relative rather than an absolute attribute “and should be understood in its physiological sense rather than in a strictly physico-chemical way”.²⁹ Though acknowledging the emphasis placed on the stabilization of bodily states has a long-standing history, Cannon credits the work of the 19th Century physiologist Claude Bernard for first giving precision to the general underlying concept of homeostasis with the insight:

...that in animals with complex organization the living parts exist in the fluids which bathe them, i.e., in the blood and lymph, which constitute the "milieu interne" or "intérieur"—the internal environment, or what we may call the *fluid matrix* of the body...And as organisms become more independent, more free from changes in the outer world, they do so by preserving uniform their own inner world in spite of shifts of outer circumstances.³⁰

Cannon’s conception of “homeostasis”, and its emphasis on normal conditions “within narrow limits” sharpened Bernard’s view that “all the vital mechanisms, however varied they may be, have only one object, that of preserving constant the conditions of life in the internal environment”.³¹ The crucial upshot was that Bernard, like Hippocrates before and Cannon after, claimed that disease results when the normal state of the organism is upset.

For our purposes, the crucial idea is that the demarcation of normal functioning as bodily homeostasis may provide a way to account for normal functioning without relying

²⁹ Steven J. Cooper (2008), “From Claude Bernard to Walter Cannon: Emergence of the concept of homeostasis”, *Appetite* 51: 419–427, at p. 424.

³⁰ Cannon (1929), *op cit.*, pp. 399–400. Earlier in this work, Cannon also credits Hippocrates, Eduard F. W. Pflüger, Léon Fredericq and Charles Richet for each recognizing key aspects of the concept.

³¹ Claude Bernard translated in *Ibid.*, p. 400.

on statistics. This notion has some plausibility especially since Boorse explicitly states, “the homeostatic breakdown is pathological”³² and that:

Certainly many aspects of normal and abnormal physiology fit this model. Countless biological variables like blood temperature, acidity, speed of flow, and composition with respect to innumerable substances and organisms must be kept within narrow limits in a state of health.

It is worth quoting at length a passage which makes clear that Boorse in fact intends homeostasis to be a necessary part of his understanding of pathology and hence of disease:

Obviously, no fact is more pervasive than what is often called the “dynamic equilibrium” of normal physiology: the normal functional variation within organisms acting and reacting to their environment. The normal level of almost all part-functions varies with what an organism is doing, what other part-functions are being performed, and the environment. Heart rate, blood pressure, respiration, and countless other variables vary from exertion to rest. The secretion of digestive enzymes is coordinated with meals, sweating with body temperature, and so on. Many functions are performed only intermittently; when performed, they raise or depress other functions. Indeed, a biomedical functional pattern is anatomically enshrined in the division between sympathetic and parasympathetic nerves, a contrast in turn endocrinologically duplicated, complicated, and regulated. *Though I did not stress the dynamism of normal physiology in presenting the BST, I always assumed it...* A common pattern is that environmental stress evokes short-term compensatory functions that maintain homeostasis up to a point, but beyond that point the coping mechanisms break down and a discontinuity, a discrete state of illness, results.³³

Equally clear, however, is the fact that Boorse does not view the state of homeostasis to be a sufficient condition for normal functioning:

Homeostasis cannot, however, profitably be viewed as a general model of biological function. Many life functions are not homeostatic unless one stretches the concept to cover every goal-directed process. Perception,

³² Boorse (1997), p. 79.

³³ Boorse (1997), pp. 78-79. Italics mine. Here it is worth in passing drawing attention to Boorse’s use of the term ‘normal’ in this passage, for it is very tempting to read him as using this term here in a way which it is hard to make sense of unless it is cashed in terms of what is statistically typical – however, that is not my point here.

locomotion, growth, and reproduction upset an equilibrium rather than maintain one. To say that their ultimate aim is internal equilibrium is unfounded; it is equally true, or truer, that the ultimate aim of internal equilibrium is perception, locomotion, growth and reproduction. Thus there is no point in trying to view corresponding diseases such as deafness, limb paralysis, dwarfism, or sterility as homeostatic failures. One can see why various equilibria are crucial to life without confusing homeostasis with the broader idea of normal functioning.³⁴

This passage shows that Boorse contends that the state of homeostasis is not a sufficient condition for health because many life functions are not homeostatic. There are two salient points that merit comment. First, it is not surprising that Boorse contends that, if homeostasis is to be a sufficient condition for health, then it has to capture every goal-directed process. Echoing Sommerhoff's (1950) analysis, Boorse views goal-directedness as the key feature that distinguishes living organisms from dead or non-living matter.³⁵ To illustrate this point, Boorse draws upon a passage from Sommerhoff: "...*the beast is not distinguishable from its dung* save by the end-serving and integrating activities of apparent purposiveness and organic order in material systems".³⁶ In fact, a failure to appreciate Sommerhoff's beast-dung point, Boorse suspects, has led the normativists to ignore the plausibility that the life/non-life contrast may automatically generate a health/non-health scientific concept.³⁷

A second, and related point, is whether Boorse is correct to insist that life functions such as perception, locomotion, growth and reproduction *upset* rather than maintain the

³⁴ Boorse (1977), p. 550. The view is echoed in Boorse (1987), p. 369.

³⁵ Boorse (1997), p. 9. Margolis also argues that the mechanism of homeostasis requires a prior account of goal-directed activities. Joseph Margolis (1976), "The concept of disease", *Journal of Medicine and Philosophy* 1(3): 238-255.

³⁶ Sommerhoff (1950), *op cit.*, p. 6; Boorse (1997), p. 9.

³⁷ Boorse (1997), p. 10.

“normal” state of the organism. In itself, it is difficult to know what to make of Boorse’s criticism, since it is not at all clear why the above-mentioned life functions must run against Cannon’s conception of homeostasis. Moreover, given that Boorse says both that the homeostatic breakdown is pathological and that aging and pregnancy are not diseases (i.e., are not pathological), he seems committed to the view that there need be no homeostatic breakdown during perception, locomotion, growth and reproduction. The difficulty one runs into here is that it is not at all clear from these conceptions (and perhaps any conception) of homeostasis what is supposed to count as the “normal state of the organism”.

A useful perspective is revealed when Georges Canguilhem points out when we understand normal functioning as bodily homeostasis, “it means, in short, that organic life is an order of precarious and threatened functions which are constantly re-established by a system of regulations”.³⁸ Certainly, Boorse does not persuasively show that any of the identified life functions *upsets* “the coordinated physiological reactions which maintain most of the steady states in the body” or that the above life functions are not “constantly re-established by a system of regulations”. Boorse merely asserts what must be shown. Thus, Boorse’s claim that homeostasis is not a sufficient condition for health because many life functions either upset homeostatic equilibrium and/or are not homeostatic is unpersuasive.

³⁸ Georges Canguilhem (1978), *On the normal and the pathological*, Dordrecht, Holland: D. Reidel Publishing Co., p. 161.

4.2 *Why Homeostasis is not sufficient for health*

There is, however, a more persuasive reason for insisting that homeostasis is not a sufficient condition for health: We do not want to say that an individual is healthy if she suffers from two generally recognized diseases, X and Y, where Y is such that its symptoms restores the homeostasis which would otherwise be upset by X. One way to see this is to recognize that there are states, which current medical practice is surely correct to count to be diseases, that are such that an individual may have one or more diseases with no upset in homeostasis.

To illustrate, consider the fact that an individual suffering from diabetes mellitus may have her blood sugar levels restored by an insulinoma—an islet cell tumour of the pancreas.³⁹ Here the secretions of an insulinoma abnormally raise insulin levels. In the case of a diabetic, these may serve to “normalize” the blood glucose levels and, as such, instead of causing hypoglycemia, the tumour effectively ameliorates diabetes.

The signs and symptoms of a disease may also be unintentionally masked by the prescribed treatment for another disease. For example, acid-suppressing proton pump inhibitors for benign ulcers have been shown to mask symptoms of early gastric cancer.⁴⁰ Even harmful behaviors may unwittingly serve to restore homeostasis given that the smoking of menthol cigarettes has been shown to mask symptoms of respiratory disease.⁴¹

³⁹ See, for example, Muhammad Fuad Hamed, Graeme E. Hole, & Zoe Muir (2006), “A mysterious case of normalising blood sugar: insulinoma in a long-standing diabetic patient”, *Age and Ageing* 35: 317–318; Elif Arioglu, Nicole A. Gottlieb, Chrostan A. Koch, John. L. Doppman, *et al.*, (2000), “Natural history of a proinsulin-secreting insulinoma: From symptomatic hypoglycemia to clinical diabetes”, *The Journal of Clinical Endocrinology & Metabolism* 85(10): 3628-3630.

⁴⁰ S. M. Griffin & S. A. Raimes (1998), “Proton pump inhibitors may mask early gastric cancer”, *British Medical Journal* 317:1606–1607.

⁴¹ Samuel Garten & R. Victor Falkner (2003), “Continual smoking of mentholated cigarettes may mask the

It is worth noting here that a diseased organ may be asymptomatic and, moreover, not upset homeostasis in virtue of its ability to compensate for itself. Consider the kidneys, which are two organs. The upshot is that one may be born with one kidney and never experience renal failure. Correspondingly, one may also have unilateral kidney disease,⁴² which would show neither signs nor symptoms of renal failure. An individual, for example, with uncomplicated unilateral medullary sponge kidney (Cacchi-Ricci disease) would generally show no disruption in homeostasis.⁴³

The point to press with these examples is that an individual can be both diseased and yet, on the definitions of homeostasis provided, seemingly in homeostatic equilibrium. And, what is more, insofar as it is surely very tempting to talk in terms of the signs and symptoms of a disease being “masked”, one could expect there to be no upset in homeostasis.

Let me now stress the fact that since an individual may have one or more diseases with no upset in homeostatic equilibrium, homeostasis cannot be a sufficient condition for health. To this effect, I will motivate and reject what might otherwise appear to be a promising account of normality that would fit my characterization of what it is to be a biological account of normality but which would be seemingly not one with essential reference to what is to count as being statistically typical.

early warning symptoms of respiratory disease”, *Preventive Medicine* 37:291–296.

⁴² This particular example of unilateral kidney disease is drawn from Boorse (1997), p. 49.

⁴³ In their 2008 publication “Medullary sponge kidney”, The National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC) reports that problems caused by MSK (e.g., hematuria, kidney stones, and urinary tract infections) do not usually appear until ages 30 to 40. <<http://kidney.niddk.nih.gov/kudiseases/pubs/medullaryspongekidney>> Accessed July 4, 2011.

4.3 Homeostasis: A rejoinder?

It may seem to some that the above argument reaches a hasty conclusion. Perhaps one might object that the force of the argument turns on a limited view of homeostasis, which, simply, is not deep enough, literally. The objection being that what the above demonstrates is merely that general *bodily* homeostasis is not a sufficient condition for health. But all this shows is that we need to delve deeper and construe the model of homeostasis such that when we insist that homeostasis is a sufficient condition for health what we contend is, to put it very crudely, that an organism is healthy when each and every vital homeostatic mechanism is not upsetting equilibrium.

To illustrate, one might run the objection as follows. Despite having no upset in general bodily homeostasis, an individual with diabetes mellitus (which reduces insulin secretion) and an insulinoma (which increases insulin secretion) would have no less than two processes upsetting, among other things, the ability of a particular vital homeostatic mechanism—the pancreas—to maintain blood glucose levels. The argument being, one might contend, that the individual is not properly counted to be healthy on this deeper understanding of homeostasis because there are two processes upsetting the pancreas' insulin-regulating ability—which is a vital lower-level homeostatic mechanism and thus, there fails to be the requisite lower level homeostatic equilibrium sufficient for health.

So granted, it is fortunate for the individual that, at the general level of the organism, the two diseases are compensating for each other to effectively maintain bodily homeostasis. But at the lower-level of the organ, a vital homeostatic mechanism *is nevertheless* being upset. *Mutatis mutandis*, the same conclusion could be drawn for the individual whose prescribed treatment masks symptoms of early gastric cancer and for

the individual whose menthol cigarettes mask symptoms of respiratory disease.

The upshot of a rejoinder of this kind is that it offers a model of homeostasis that is not, at least *prima facie*, committed to the absurd view that an individual is healthy merely because and insofar as the general state of the organism is in homeostatic equilibrium. For this reason alone, it may seem that when the focus shifts to the homeostatic equilibrium of particular mechanisms, the claim that homeostasis is sufficient for health becomes a lot more plausible. But looming here is a serious two-prong objection, to which we shall now turn.

4.4 The argument revisited: A two prong objection

4.4.1 The need to extend the model of homeostasis to the cellular level

The first prong picks up the keen point that there is always the possibility that, at any level, the homeostatic mechanism itself may be diseased. The corollary of this possibility, to quote the astute phrasing of Joseph Margolis, “obliges us to construe bodily functions in terms of higher-order norms”.⁴⁴ And correspondingly, given the task at hand, this firmly places an onus on the naturalist to specify the healthy state of the human organism without abandoning the conceptual link between homeostasis and health. Georges Canguilhem nicely frames the difficulty of construing the requisite norms:

If the individual organism is the one which, of its own accord, proposes the norms for its restoration, in the case of malformation or accident, what sets up as norms the specific structure and functions which cannot be grasped by the individuals other than as they are manifested? Thermoregulation varies from the rabbit to the stork, from the horse to the camel. But how do we understand the norms peculiar to each species, rabbits, for example, without

⁴⁴ Margolis (1976), *op cit.*, at p. 246.

erasing the slight, fragmentary dissimilarities which give individuals their singularity?⁴⁵

In other words, the challenge is for the naturalist to specify the “healthy” homeostatic states of the organism with norms that are *only* grounded in empirical observations of how the relevant species functions. This is a daunting task. Indeed, a serious difficulty quickly appears when we recognize, as we must, that individuals attain health with an immense variability of functioning.⁴⁶ As Ron Amundson, following Jirí Vàcha, remarks:

A high degree of variability exists among individuals on any physiological measurement, with even the most extreme values found within healthy individuals. Extreme values of physiological parameters, associated with disease in some individuals, are compensated for in others.⁴⁷

So, in the face of both the possibility that the system of regulations may be diseased and the clear variability of successful functioning, how is the naturalist to construe the “normal” or, perhaps more accurately, the range of the homeostatic mechanisms sufficient for health? Are some functioning ranges to be considered normal so long as it is compensated for by another function? Surely not. There is thus the need to formulate the model of homeostasis to ensure that two diseases cannot suffice for health. But how?

At least this much seems clear: A satisfactory homeostatic model must have the wherewithal to count some functioning ranges as diseased processes when it is

⁴⁵ Canguilhem (1978), *op cit.*, at pp. 161-62.

⁴⁶ For an interesting discussion of some the difficulties such variability poses for conceptions of “normal” and “health”, see Jirí Vàcha (1985), “German Constitutional Doctrine in the 1920s and 1930s and pitfalls of the contemporary conception of normality in biology and medicine”, *Journal of Medicine and Philosophy* 10(4): 339–367.

⁴⁷ Amundson (2000), *op cit.*, at p. 43.

compensated for by another function(s) such that there is no upset in higher-level homeostasis. And so it seems that we are going to have to broaden the conception of homeostasis to include every contributing process towards homeostatic equilibrium, down to, at least, the cellular level.⁴⁸ Otherwise, we lack the resources to renounce the absurd view that an individual with, say, early-stage pancreatic cancer is healthy merely because there is no upset of any higher-level homeostatic mechanisms vital for health (I return to this point below).

Or to put the point more generally, the force of argument raised earlier against the claim that homeostasis is sufficient for health simply strikes deeper when the aim is shifted from general bodily homeostasis towards particular homeostatic mechanisms. There is thus the need to construe homeostasis in terms of yet more basic levels of bodily functioning. It should not go unnoticed that this obliges the homeostatic model to further account for the variability of successful functioning. It is at this point the second prong of the objection emerges.

4.4.2. Homeostasis is not a model that captures how many cells successfully function

But if the need to extend the model of homeostasis to include more basic levels of functioning is granted, then there still remains the significant question: Is the model of homeostasis equipped to facilitate the requisite extension? I do not think that it is. This is

⁴⁸ Boorse explicitly recognizes this: “Up to a certain point, many organs, like heart or kidney, can functionally compensate for tissue disease by hypertrophy. So it is not true that clinically evident pathological states entail dysfunction at the organ level, as opposed to that of the cells and tissues.” See Boorse (2010), *op cit.*, at p. 61; and also Boorse (1977), *op cit.*, pp. 550, 556 & 565. To his credit, Boorse recognizes that “cells are goal-directed toward metabolism, elimination, and mitosis” and clearly intends for the BST to extend dysfunction and normality all the way down to the intracellular level. See Boorse (1975), *op cit.*, p. 57 and (1997), *op cit.*, pp. 7, 85, & 91.

because homeostasis is not a model that adequately captures how many vital cells successfully function. That is not to deny that many cells seem irresistibly directed to preserve their *own* internal environment, such as when the cell membrane regulates the chemical composition of its body fluids. But it is no less true that many cells appear internally directed to die and, as such, not to maintain their own internal environment. An example is the orderly death of the endometrium lining cells during menstruation.

In fact, so regular and predicable is cell suicide, in all multicellular organisms, that countless scientists employ the term programmed cell death (PCD), or apoptosis. *A fortiori*, the genetically controlled death of many human body cells (e.g., skin, intestinal, *T* lymphocytes, and the endometrium lining cells) by apoptosis is necessary for both normal development and health. In a recent article from *The New England Journal of Medicine*, the authors graphically stress the importance of apoptosis when suggesting that a human lacking apoptotic cell death would likely accumulate 2 tons of bone marrow and lymph nodes, and a 16-km intestine by the age of 80!⁴⁹ Further still, without apoptosis, cells would divide uncontrollably and result in cancers (I return to this below).

Thus it would be a great mistake to insist that homeostasis at the cellular level is either necessary or sufficient for health—we require cells that are programmed to upset their internal environment and die! Clearly, homeostasis is not a model that adequately captures how many cells successfully function.

⁴⁹ Richard S. Hotchkiss, Andreas Strasser, Jonathan E. McDunn, & Paul E. Swanson (2009), “Cell death, *The New England Journal of Medicine* 361:1570-1583, at 1570.

4.5 *The emergence of three serious problems*

So I think we have to admit to the following conditional: if we insist that homeostasis is sufficient for health, then the model of homeostasis cannot extend to the cellular level. For internal cellular homeostasis is neither necessary nor sufficient for health. But notice that with this concession we can only link disease to the internal malfunctioning of cells if it is upsetting an external homeostatic mechanism. This limitation is highly problematic, for three reasons.

First, it runs against ordinary medical practice. Current medical practice counts individuals with asymptomatic early gastric, prostate and/or breast cancer to be diseased. And it is surely correct to do so. Yet, given the absence of any signs of homeostatic disruption, it is difficult to envision when homeostasis is viewed sufficient for health how such individuals would be properly counted as diseased. This is a serious difficulty for the model of homeostasis given that there are many uncontroversial diseases,⁵⁰ which have asymptomatic periods (particularly in the early-stages) and, *a fortiori*, would not show signs of an upset in homeostasis beyond the cellular level. Indeed, that there are such diseases strongly suggests that medical practice's operational account of health and disease extends to the cellular level.

To further motivate this view, consider the extent to which traditional cancer treatment options aims to eliminate cancer cells either by killing them (e.g., chemotherapy, radiation therapy) or by removing them (e.g., surgery). The modest success of these

⁵⁰ For example, most forms of cancer, hypertension, syphilis, AIDS, Huntington's disease, sarcoidosis, Cacchi Ricci disease (medullary sponge kidney), Celiac disease, sickle cell disease, and Wilson's disease have well-documented asymptomatic periods.

treatments provides good reason to insist that cells can be diseased.

Second, it runs against important medical research. To illustrate, researchers have implicated aberrant apoptotic regulation—resulting in too much or too little cell suicide—in a variety of diseases, including cancer, ischemic stroke, ALS, AIDS, Alzheimer’s disease and rheumatoid arthritis.⁵¹ It is neither insignificant nor irrelevant that recent research has identified that an overexpression of Bcl-2, an essential regulator of PCD, is both common in pancreatic cancer and correlates with resistance to PCD.⁵² In fact, it has recently been claimed that over 50% of neoplasms have defects in the apoptotic machinery.⁵³ If this striking claim is at all accurate, then it is fundamentally wrongheaded not to directly connect health and disease to human functioning at the cellular level. Yet the model of homeostasis seems unable to make this connection.

This is significant because it runs against an emerging model of medical research: The cancer stem cell model. The fundamental tenet of this approach, roughly put, is that there is a pathological counterpart to the stem cell—“the cancer stem cell” (CSC)—that drives the growth and metastasis of tumors. The crucial idea being that:

Tumours [sic] are driven by a cellular component that retains stem cell properties, such as stability, undifferentiation, long-term self-renewal, and capacity to replicate and undergo differentiation.⁵⁴

⁵¹ See, for instance, Sodhi K. Rupinder, Aulakh K. Gurpreet, & Singh Manjeet (2007), “Cell suicide and caspases”, *Vascular Pharmacology* 46: 383–393; Robert M. Friedlander (2003), “Apoptosis and caspases in neurodegenerative diseases”, *The New England Journal of Medicine* 348:1365-1375; and Richard C. Duke, David M. Ojcius, & John Ding-E Young (1996), “Cell suicide in health and disease”, *Scientific American* 275(6): 80-87.

⁵² Diego J. Muilenburg *et al.*, (2010), “Targeting Bcl-2-mediated cell death as a novel therapy in pancreatic cancer”, *Journal of Surgical Research* 163(2): 276-281.

⁵³ Hotchkiss, *et al.*, (2009), *op cit.*, at 1572.

⁵⁴ A. D. Purushotham & R. Sullivan (2010), “Darwin, medicine and cancer”, *Annals of Oncology* 21: 199–203, at pp. 201-202.

A remarkable insight of the CSC model is that only a minority population of cancer stem cells is endowed with the ability of self-renewal, which then exclusively initiates tumors. The majority of cancer cells have limited or no ability for proliferation.⁵⁵

For our purposes, it is important that recent evidence suggests that one of the ways that cancer stem cells arise with their functional properties is that “oncogenic mutations may inactivate the constraints on normal stem cell expansion, resulting in cancer stem cells that originated from normal stem cells”.⁵⁶ The observation that the origins of at least some forms of cancer is due to mutations inactivating *normal* stem cell functioning provides good reason to think that cancer stem cells are *diseased* stem cells. Perhaps not surprisingly, given the nature of science, the connection between stem cells and cancer is hotly disputed.⁵⁷ However, a real strength of the CSC model is that it sheds light on what molecular biologists have known for some time: that all types of cancer, despite their vast complexities and idiosyncrasies, have one special biological property in common—the territorial expansion of a mutant clone.⁵⁸

It is therefore important to take seriously the CSC model’s hypothesis that a minority population of self-renewing “diseased” cancer stem cells develops and sustains cancer. Many researchers are doing just that as they seek to develop therapeutic cancer

⁵⁵ Piero Dalerba, Robert W. Cho, & Michael F. Clarke (2007), “Cancer stem cells: Models and concepts”, *Annual Review of Medicine* 58: 267-384, at p. 269. Michael F. Clarke & Margaret Fuller (2006), “Stem cells and cancer: two faces of Eve”, *Cell* 124:1111–1115.

⁵⁶ Clarke & Fuller (2006), *op cit.*, at p. 1114. Recent evidence also strongly suggests there is another way for cancer stem cells to arise: when progenitor cells acquire the ability to self-renew. See, for example, Lobo *et al.*, (2007), “The biology of cancer stem cells”, *Annual Review of Cell and Developmental Biology* 23: 675-699; Mel Greaves (2010), “Cancer stem cells: Back to Darwin?”, *Seminars in Cancer Biology* 20(2): 65-70.

⁵⁷ Craig T. Jordon (2009), “Cancer stem cells: controversial or just misunderstood?”, *Cell Stem Cell* 4:203-205.

⁵⁸ Mel Greaves (2001), *Cancer: The evolutionary legacy*. Oxford: Oxford University Press.

treatments specifically designed to eliminate the cancer stem cell and ignore nontumorigenic cancer cells.⁵⁹

Whatever the biological mechanism that gives rise to cancer stem cells, the tacit underlying assumption of the CSC model that cells can be diseased seems well-grounded. For it is well-appreciated that cancer develops as a chromosomal gene changes in single cells.⁶⁰ Indeed it should not escape our notice that J. D. Watson, one of the three co-discoverers of the structure of DNA, is among those who contend that the etiology of most cancers arise through the overexpression of key cellular regulatory genes.⁶¹

However, if we want to say that the cell is *per se* diseased, then it seems we cannot appeal solely to a model of homeostasis. This is because the model of homeostasis lacks the requisite framework to directly connect health and disease to human functioning at the cellular level. This is a serious problem. Not the least of which is that the model of homeostasis thus seems committed to upholding a lacuna between the connections that medical research, in general, and, molecular biology, in particular, is advancing between cellular mechanisms and health and disease.

Third, and finally, this disconnection with medical practice and research renders the model of homeostasis vulnerable to a recasting of our earlier objection. An individual may have one or more cellular diseases with no upset in any higher-level homeostatic equilibrium. To amplify the force of this objection, consider pancreatic cancer. In the

⁵⁹ See, for instance, Mark Shackleton *et al.*, (2009), “Heterogeneity in cancer: Cancer stem cells versus clonal evolution”, *Cell* 138(5): 822-829.

⁶⁰ Greaves (2001), *op cit.*

⁶¹ James D. Watson, *et al.*, (2008), *Molecular biology of the gene*, 6th Edition, San Francisco: Pearson/Benjamin Cummings.

United States, by the time pancreatic cancer is detected the prognosis is grim; 26% of all patients will have advanced to the regional stage and 52% to the distant stage of the disease.⁶² In Canada, the mortality rate is estimated to be 99% for those diagnosed with pancreatic cancer in 2009.⁶³ What makes pancreatic cancer so notoriously difficult to diagnose is that it is asymptomatic in the early stages of the disease.

We know, however, that pancreatic cancer begins at the cellular level. But, as we saw above, the model of homeostasis seems only able to link disease to the internal (mal)functioning of cells if it is upsetting an external homeostatic mechanism. To this extent, the model of homeostasis thereby cannot account for many diseases that modern medicine counts among the most ubiquitous and serious.

We also know that despite being clinically silent—and not upsetting homeostasis—it is obvious that individuals with early-stage pancreatic cancer are not healthy. In fact, given its very poor prognosis—a 5-year survival rate less than 5% after diagnosis⁶⁴—it is a gross understatement to say that early-stage pancreatic cancer is anything but a serious disease. Homeostasis, therefore, cannot be, properly considered, “sufficient for health”.

4.6 Section Conclusion

One might assume that homeostasis is sufficient for health. To be sure, current medical practice employs a model of homeostasis to explicate accounts of normal functioning and

⁶² Ahmedin Jemal *et al.* (2009) “Cancer statistics, 2009”, *CA: A Cancer Journal for Clinicians* 59: 225-249.

⁶³ Canadian Cancer Society’s Steering Committee (2009), *Canadian cancer statistics 2009*. Toronto: Canadian Cancer Society.

⁶⁴ World Health Organization. “Cancer” <<http://www.who.int/tobacco/research/cancer/en/>> Accessed July 4, 2011.

a fortiori health. In this section, however, I have argued that homeostasis is not, properly speaking, “sufficient for health”. The main reason is that an individual may have one or more uncontroversial diseases with no upset in general bodily homeostatic equilibrium.

A promising rejoinder, I proposed, was to shift the focus to the homeostatic equilibrium of particular mechanisms (Section 3.3). In this way it might be thought that the claim that homeostasis is sufficient for health is much more plausible. In response to this I presented two objections showing why this rejoinder is not satisfactory: (1) It ultimately requires extending the model of homeostasis to the cellular level. Otherwise, we lack the resources to renounce the absurd view that an individual with early-stage cancer is healthy merely because there is no upset of any higher-level homeostatic mechanisms vital for health. (2) Homeostasis is not a model that captures how many cells successfully function because, simply put, we require cells that are programmed to upset their internal environment and die (Section 3.4). It therefore follows that, for some cells, internal cellular homeostasis is neither necessary nor sufficient for health.

Accordingly, I argued that if one’s aim is to plausibly insist that homeostasis is sufficient for health, then the model of homeostasis cannot extend to the cellular level (Section 3.5). The cost of doing so, however, appears far too high. I argued that it runs against current medical practice and research. Perhaps even more troubling, it seems to lack the wherewithal to avoid mistakenly counting an individual with clinically silent pancreatic cancer to be healthy.

In sum, I take the above to provide a decisive reason for rejecting the view that homeostasis is sufficient for health. If sound, this shows that the naturalist cannot employ

a model of homeostasis to solely underpin an adequate account of normal functioning.

It therefore remains an open question whether or not the naturalist will be able to explicate the requisite “normal state of the organism” without appealing to statistics.

This discussion of homeostasis provides us with a deeper understanding of the magnitude of the difficulties the naturalist faces bringing to bear a more appropriate conception of normal human functioning. As discussed in Section 3.4, if we understand health as normal functioning and disease as malfunctioning, then what is supposed to count as the “normal functioning of the organism”?

Once again, the naturalist, in virtue of its *modus operandi*, seems committed to employing norms that are *only* grounded in empirical observations of how the relevant species functions. And again, the challenge is for the naturalist to specify the “normal functioning of the organism” in the face of both the possibility that the system of regulations may be diseased and the clear variability of successful functioning. But to what higher-order norms can the naturalist appeal?

Boorse maintains that one can turn to physiology and comments:

One possible thesis is that, empirically, variability is so extreme in our species and others that no significant “species design” can be described, or none detailed enough to be a theoretical foundation for medicine. This seems to me to amount to saying that human-physiology (or insect-physiology) textbooks are actually false, i.e., that the endless array of facts about human physiology that medical students learn in their first and second year is untrue.⁶⁵

The next section examines Boorse’s call to turn to physiology to find the real content of “species design”. While I concede that physiology has an important, perhaps even

⁶⁵ Boorse (1997), p. 33.

necessary, role to play here, it would be a mistake to insist that, in the absence of statistics, the requisite conception of normal functioning of the organism solely resides in physiology. Let us now see why.

5. Physiology

Suppose, following Boorse, one holds that the appropriate conception of what is to count as a function resides in physiology, a subfield of biology.⁶⁶ Then, presumably, if one is concerned to explicate what it is for, e.g., a human heart to function normally, then one needs to examine what is known of the internal structure of the heart and of what is taking place when a “normal” heart is functioning normally. Thus, if one turns to the extensive details of the structure and functioning of the heart readily found in the textbooks of human physiology, then one will find something like this:

...the heart is a single organ, the right and left sides of the heart function as two separate pumps. The heart is divided into right and left halves and has four chambers, an upper and a lower chamber within each half. The upper chambers, the atria (singular, atrium), receive blood returning to the heart and transfer it to the lower chambers, the ventricles, which pump blood from the heart...Blood returning from the systemic circulation enters the right atrium via two large veins, the venae cavae, one returning blood from above and the other returning blood from below heart level...blood entering the right atrium, has returned from the body tissues, where O₂ has been taken from it and CO₂ has been added to it. This partially deoxygenated blood flows from the right atrium into the right ventricle, which pumps it out through the pulmonary artery, which immediately forms two branches, one going to each of the two lungs...[where] blood loses its extra CO₂ and picks up a fresh supply of O₂ before being returned to the left atrium via the pulmonary veins...This O₂-rich blood returning to the left atrium subsequently flows into the left ventricle, the pumping chamber that propels the blood to all body systems except the lungs...Both sides of the heart simultaneously pump equal amounts of blood. The volume of O₂-poor blood being pumped to the lungs by the right side of

⁶⁶ Boorse (1987), at p. 370. See also, Boorse (2002), at p. 90.

the heart soon becomes the same volume of O₂-rich blood being delivered to the tissues by the left side of the heart.⁶⁷

One can hardly deny the impressive details and diagrams that physiology textbooks provide about how the human body “functions”. That being said, I am not convinced that the way in which physiology accounts for functions and normal functioning (i.e., what I earlier characterized as solutions to the “qualitative” and “quantitative” issues about functions) does in the end offer an adequate account which avoids reference to what is statistically typical in some important sense. Further still, if we understand “normal functioning” to be successful functioning, then, it is difficult to envision how physiology enables the naturalist to capture the requisite value- and norm-independent range of “normal” functioning without drawing upon statistics (cardiology textbooks, note, define normal cardiac function in terms of an ejection fraction of 55-75%).

An important pressure point here seems to be that physiology must be sensitive to a basic fact about biology. For all human functions, a successful level of functioning will vary between individuals given their age, sex, present activity, and will also vary depending on environmental factors.⁶⁸ From a general perspective of human functioning, newborn infants neither walk nor talk and cannot reproduce yet the typical young adult can. At a more specific level, the heart’s pulse pressure of 80 mmHg may allow for a 35 year-old male to successfully function when exercising, but when resting this same level

⁶⁷ Lauralee Sherwood (2010), *Human physiology: From cells to systems*, 7th Edition. Belmont, CA: Brooks/Cole, at pp. 304-306.

⁶⁸ Boorse is, of course, aware of the need to reflect the normal physiological differences that exist between the young and the old, males and females and stipulates age and sex restrictions for the BST’s reference classes to do just that.

of functioning is harmful.

This is not to deny that there may be some specific level of the human heart's capacity to pump blood (e.g., a level of pumping that would simply fail to circulate blood to any individual's vital organs, regardless of activity) that may properly be counted falling short of "normal" functioning for all human hearts. However, what this shows is that there is a *minimal* level of functioning requisite for successful functioning, not that physiology can stipulate human functions are performing successfully if they perform at a particular level of functioning. This is because, roughly put, often the same level of functioning may prove to be successful in one individual and pathological in another. In addition, there are a variety of reasons⁶⁹ that would render a human heart unable to adequately circulate blood where, it seems to me, it would be a mistake to count the heart not to be normal or healthy.

Consider, for example, when the body (again for a variety of reasons) simply does not have enough blood for the heart to pump. Given a lack of blood, one surely expects there to be very low blood pressure. But notice here that the very low blood pressure may not have anything to do with, strictly speaking, a problem with the heart. After all, the human heart cannot pump what is not there and it would be a mistake to think otherwise. Here, a very low blood pressure reading thus reflects that there is a problem with the heart's *ability* to effectively pump blood, not that there is a problem with *the heart*.⁷⁰

⁶⁹ For example, toxins, hormonal abnormalities, blood volume, blood viscosity, and the resistance of the blood vessels are but five factors would affect the heart's ability to circulate blood.

⁷⁰ That this may be the case provides reason to advance a dispositional view of functions which specifies normal functioning subjunctively (e.g., the heart *would* perform its normal function if there was the requisite amount of blood). In Chapter 6, we shall see in some detail the way in which Boorse and others advance this

All human part-functions perform successfully only when in concert with one or more of the individual's other functions. Successful human functioning also requires favorable external environments. There is after all a point at which no human organism can survive exposure to extreme heat, the lack of oxygen or water. For these reasons, I find myself agreeing with Ereshefsky, following Wachbroit, that physiology describes human functions in “unperturbed states”.⁷¹ Speaking to this Ereshefsky claims:

[P]hysiology texts provide idealized and simplified descriptions of organs, not descriptions of their inherent natures...To assert that physiology texts provide the natural states of organs or systems goes well beyond the intended purposes of such descriptions.⁷²

This proves to be a significant blow to the defender of the BST who is aiming to avoid Cambridge-change objections by employing a statistics-free and thus a different conception of normal human functioning. This is because of the point which Boorse (after rejecting several notable ideas in health definitions—i.e., treatment by physicians, statistical normality, pain and suffering, disability, adaption, and homeostasis—as simple necessary or sufficient conditions of disease) rightly makes:

To capture the modern extension of “disease,” what seemed required was a modern explication of the ancient idea that the normal is the natural—that health is conformity to “species design”. In modern terms, species design is the internal functional organization typical of species members, which (as regards somatic medicine) forms the subject matter of physiology: the interlocking hierarchy of functional processes, at every level from organelle to cell to tissue to organ to gross behaviour, by which organisms of a given species maintain and renew their life.⁷³

sort of a view.

⁷¹ Ereshefsky (2009a), *op cit.*, p. 223. Wachbroit (1994), *op cit.*, p. 589.

⁷² Ereshefsky (2009a), *op cit.*, p. 223.

⁷³ Boorse (1997), p. 7.

As I read Boorse, his point (when augmented with Ereshefsky's phrasing) is that because physiology does not describe the natural states of organs or systems (describing instead idealized or simplified descriptions) it does not *by itself* offer an adequate explication of the idea that "the normal is the natural". And as such, physiology *by itself* does not have the explanatory resources to ground the requisite value- and norm-independent account of normal functioning. Hence, we need to go beyond physiology's standard account of normal functioning and look to the rôle that physiological structures play in the wider life of the organism.

This is precisely what Boorse attempts to do by making essential reference to what physiological structures typically can and do do—again I note my explication of Boorse's theory in (D*) in Section 1 of my Chapter 3. There I point out that Boorse equips the BST with a goal-directed view of biological function, which requires a function "to be an *actual* contribution to a goal"⁷⁴ and which is statistically typical for such structures in the relevant species. More precisely Boorse states:

"A function of X is Z" means that in some contextually definite goal-directed system S, during some contextually definite time interval *t*, the Z-ing of X falls within some contextually circumscribed class of functions being performed by X during *t*—that is, causal contributions to a goal G of S.⁷⁵

On this view, function is emphatically wedded to a particular context. And because of this, the BST's account of normal function must draw upon the *actual* range of functioning of the relevant physiological parts. Thus, given the BST's current framework, the defender of the BST should not be satisfied to underpin its understanding of normal

⁷⁴ Ibid., p. 66.

⁷⁵ Boorse (1976b), p. 82.

function with either “ideal” states to which no token conforms; or the above concept of biological normality which does not require the “normal function” to be realizable in the present; or physiological descriptions in unperturbed states.

Equally clear is the point that the requisite value-free range of *normal* functioning needs to be more contextually defined than it is in the state-descriptions found in physiology textbooks. Unless such further considerations are factored into the subject matter of physiology, the requisite account of normal functioning does not and cannot reside entirely in the discipline of physiology.

6. Chapter Conclusion

As we saw above, the defender of the BST does not find the requisite statistics-free conception of normal functioning in physiology textbooks. So where does that leave us?

If the above is correct, then we are left with legitimate doubt about the BST’s prospects to bring to bear a persuasive alternative naturalistic account of typical efficiency/normal functioning. Thus far, we have seen that the following considerations favour such skepticism. First, the abandonment of statistical conception of normality requires the naturalist to have a more compelling non-normative conception of “normal” human functioning ready-at-hand. Second, the essentialist view that disease *qua* disease is a natural kind does not stand up to scientific scrutiny. Third, the ever-looming requirement that no value-free naturalist project worth its salt can appeal to an evaluative concept of normality. Fourth, there is the realization that homeostasis is not a sufficient condition for health. Fifth, there is the problem of capturing the needed naturalist account of normal functioning (i.e., what is identified as solutions to the quantitative issues about

functions), when the biological conception of normality does not itself specify the needed, statistics-free, range of *actual* functioning. Sixth, the corresponding problem of specifying a value-free range of normal functioning needs to be more contextual than physiology texts seemingly is able to give. These six points show the difficulty of specifying the requisite naturalist account of normal functioning when statistical normality is abandoned.

Finally, and perhaps most importantly, abandoning a statistical conception of normality risks making the BST's goal-directed view of biological function untenable. The crux of the problem is that the framework of the BST requires a value- and norm-independent account of typical efficiency/normal functioning that the naturalist nowhere supplies.

The problem becomes acute because when a statistical conception of normality is abandoned, so too is recourse to packing "typical efficiency" as "efficiency above some...minimum in its species distribution" as Boorse does.⁷⁶ In effect, then, it is not at all clear how to advance Boorse's account of "normal functional ability: the readiness of each internal part to perform all its normal functions on typical occasions with at least typical efficiency".⁷⁷ Without principled grounds for specifying when a function is performing with *typical efficiency*, ultimately the BST cannot winnow functioning that is "diseased" from "healthy". Clearly this stands as a pressing, significant, and perhaps insurmountable obstacle to the defender of the BST's attempt to avoid the Cambridge-

⁷⁶ Boorse (1997), p. 8.

⁷⁷ Boorse (1977), p. 555 and (1997), p. 8.

change objections by employing a statistics-free notion of normal human functioning *qua* normal. The recognition of this reveals the naturalist's apparent need to employ a statistical conception of normality of function.

Considerations such as these oblige one to concede that the naturalist does not have a more compelling non-normative conception of normal human functioning ready-at-hand. In sum, not only is there no reason to think that the naturalist can save the BST from Cambridge-change objections by abandoning a statistical conception of normality; it is difficult to envision how a satisfactory statistics-free, naturalist conception of normality could be brought to bear. A different approach is therefore needed.

Chapter Six

“Normal Functioning”

1. Introduction

If the preceding chapter is correct, then a satisfactory naturalist conception of health and disease will require a statistical conception of normality. This provides strong motivation for the naturalist to insist that the problem with Boorse’s biostatistical theory of health (BST) is not that it invokes a statistical conception of normality *per se*; but rather that it dovetails a statistical conception of normality with a particular conception of function.¹ That is, the problem is with the particular analysis of the performance of a function, which Boorse uses in spelling out his account of normal functioning.

Admittedly, this rejoinder must be taken seriously, for, if successful, it would surely make my Cambridge-change objection against the BST ineffective as an objection and that *would* be important, since the BST is the most influential naturalist theory of health. Thus the aim of this chapter is to examine the possibility that Boorse’s suggestion on how to develop a value- and norm-independent account of health and disease might be saved if it is spelled out with a different account of *function*.

1.1 The rôle of statistical considerations in addressing qualitative and quantitative issues in relation to functions

In this chapter I will be examining the notions of a function, of functioning, and of “normal” functioning. In these discussions it will, for reasons which will emerge, be

¹ It would be premature to think that a fatal blow is delivered to the BST *per se* if Boorse’s analysis of function is shown to be wrong. I expand upon this below.

essential to ensure that it is completely and unambiguously clear at which level of the account of what it is to be a function and to perform a function the notion of “normality” plays a role. For even if a non-statistical conception of what it is to be a function the account of health and disease will still need to include an account of what it is to perform a function normally. To ensure this, it will help to return to the important distinction we made in Chapter 3 (Section 3.3) between qualitative and quantitative issues in discussion of functions. Recall that a qualitative/quantitative distinction was made to distinguish between two different classes of issue. One class of issues are the issues about which biological processes are to count as functions and the other are issues about which level of functioning is requisite for the function to count as being successfully, adequately, etc., performed.² The former issues I called the qualitative issues about functions and the second the quantitative issues.

So as to avoid any confusion, I have decided³—at the risk of verbosity—to further explicate the rôle of the notion of normality in these two kinds of issues about functions (qualitative and quantitative) by describing a thought experiment.

(I) “Waligators”

Imagine that a group of naturalists, deep in the Amazon forests, were to discover a single carcass of what seems to be a new species of organism. What is immediately noted about

² Note that talking about successful, adequate, etc., performance for function reflects the fact that in ascribing a function to something we are implying that it serves some goal, purpose, etc. It is worth noticing that Boorse very definitely would be comfortable with this comment – see footnote #47 below. But it must also be acknowledged that other theoreticians would be very uncomfortable with this suggestion – for example what have been called ‘selected effect’ theories of function. As will emerge I think I can leave such accounts on one side.

this particular creature is that it has what looks like two walrus tusks emerging from each side of what slightly resembles an alligator's jaw. Suppose they set out to ask, "what is the function of the tusk-like protuberances of this 'waligator'⁴?" It is important to be clear how very tricky their task will be. First and foremost it is important to acknowledge that naturalists face epistemological constraints that are of considerable importance. Function ascriptions must accord with a naturalistic methodology that will be rooted in an empirical epistemology. Moreover, the naturalist must advance well-warranted function ascriptions which meet rigorous scientific standards.⁵ Given (as we shall see in Section 2.1, below) the fundamental rôle the account of function must play in the BST, a failure to meet such standards would fatally undermine the claim to provide medicine with a satisfactory value- and norm-independent account of health and disease.

Secondly, notice that at this point it is not at all clear that the naturalists *should* ascribe a "function" to the waligator's "tusks" at all, regardless of which naturalist candidate account of function is preferred.⁶ For it is not clear that our naturalists are not observing vestigial tusks, i.e., structures which at an earlier stage of evolution *used to have* tusk-like functions but now no longer have those functions. Nor is it clear that the genetic

³ I am grateful to John A. Baker for pointing out the merits of describing a thought experiment here.

⁴ My thanks to David Waller for helping me to come up with the name 'Waligator'.

⁵ Susan Haack nicely emphasizes what I take to be the kind of commitments a naturalist about health and disease must accord with when she says: "Scientific inquiry relies on experience and reasoning: the sciences have developed many ways to extend the senses and enhance our powers of reasoning, but they require no additional kinds evidential resource beyond these, which are also the resources on which everyday empirical inquiry depends". Susan Haack (2004), "Point of honor: On science and religion", *Skeptical Inquirer* 28(2): 56–62, at p. 57.

⁶ Rival conceptions of function require different criteria for function ascription like, for instance, that the tusks are not vestigial (Boorse; Bigelow & Pargetter); or that the trait for the waligator's tusks are *selected for* (Godfrey-Smith; Neander); or *selected by* natural selection (Millikan; Neander); or that the tusks actually contribute to the goals of individual survival or reproduction (Boorse); or that the tusks would give a

trait for the waligator's tusks (if there is one) had been selected for; perhaps the trait is maintained because of pleiotropy or a link that has it "hitchhike genetically on useful traits".⁷ Nor is it even clear that the "tusks" of this particular waligator are not present because of disease and, moreover, having these tusk-like protuberances was what actually caused its death. At this point, in other words, our naturalists do not know the *functional organization* of the waligator-type. That is, as some put it, the uniform mode⁸ of functions of the waligator are unknown. All this is to say that the solutions to the qualitative issues about function(s) of the waligator are presently unknown.

And, as it stands, the solutions to the quantitative issues about functions are *also* thus far unknown. With only one dead waligator-token, the naturalists have no evidence as to what would be a "statistically typical" level of performance for this "tusk" if it is a tusk, let alone what would be the normal level of performance for animals of this type, whatever this type is.

Suppose, however, that our naturalists were subsequently to come across two different living waligators each day for the next year of their Amazon expedition, so that on the second week of their expedition, there would be a class of fourteen specimens each of which looked very much like the first specimen, i.e., the one they have named a

survival-enhancing propensity to the waligator in its natural habitat (Bigelow & Pargetter).

⁷ For examples, see for instance, Peter Godfrey-Smith (1994), *op cit.*, at pp. 347-348. Briefly, "pleiotropy" is a term used to describe the situation whereby a gene gives rise to two (or more) phenotypic effects; and "genetic hitchhiking" refers to when two genes that are close enough together on a chromosome such that selection for one of them causes the other gene to evolve as well, even if the latter neutral or deleterious. See Elliott Sober (2010), "Natural selection, causality, and laws: What Fodor and Piatelli-Palmarini got wrong", *Philosophy of Science* 77: 594-607, at p. 597.

⁸ In drawing what I take to be a similar distinction to my qualitative/quantitative, Amundson distinguishes between *level of performance* and the *mode* of performance. He writes, "Functional mode is the manner in which a functional outcome or performance is achieved". See Amundson (2000), *op cit.*, at p. 36.

waligator. *Now* they can do some comparing and contrasting. Importantly, (though it will depend on the conception of function they are working with how soon this could happen), they will begin to want to ascribe functions to these “useful” traits. The time will maybe come (once they have seen enough specimens) when the naturalists are in a position to observe and examine an increasing number of living waligators and they would presumably begin to identify what they can class as “useful” traits of what they conclude they are justified in taking to be “tusks”.

Imagine, for example, that the naturalists have observed that all but the very young and two much older “waligators” seemingly “use” their tusks to penetrate an insect’s nest and “inject” a toxic secretion that paralyzes the insects, which it then eats. The observations suggest to our naturalists the hypothesis that the “function” of the “tusk” is to serve as an “insect-paralyzing-poison-injector”. This, I want to say, could be counted as the kind of suggestion that could be classified as a solution to a qualitative issue about what is to be counted as the function of the tusks. Now what is important here is the fact the naturalists seem to have no real choice but to use evidence of a statistical kind to solve the issue of what is the function or what are the functions of the tusk-like protuberances in this species.

Here, of course, the naturalists may not be correct in their hypothesis about what the solutions to the qualitative issues about the function(s) of the tusk-like protuberances. For perhaps their sample size is, for some reason, unrepresentative of the species. Yet, as each day passes with similar observations, there will be *ex hypothesi* more waligators from which to establish statistically typical species-type qualitative functions, which—in the words of one famous account—brings increasingly stronger inductive support or

confirmation.⁹

Now once they have a statistically well-warranted hypothesis about what are the solutions to the qualitative issues about function(s) of the “tusks” they might ask of a particular waligator, say *W*, whether what they now for good reason count as its tusks are *functioning* normally, i.e., are performing *normally* what are now known to be their functions. A first reasonable step in addressing this issue seems to demand that one first confirm whether *W*’s tusks are at all in fact able to penetrate an insect’s nest at all and in fact inject a toxin that paralyzes the insects. After some examination, suppose *W*’s tusks are confirmed to be capable of performing the functions which, it is hypothesized, solve the qualitative issues about the structure’s function.

But even with this established, the naturalists might yet wonder if *W*’s tusks are functioning *normally*: after all, even if *W*’s tusks may be able to penetrate an insect’s nest and inject a toxin (the solution to the qualitative questions about the functions of the structures), nevertheless the toxin injected might be, for one reason or another, too weak or too small in volume, to paralyze the intended prey, as it once did. There is thus an important difference between one’s biological mechanisms performing its function, i.e., “functioning”, and “functioning successfully” or “adequately” and these questions, I will suggest, lead one to look at the question of whether they are performing their functions, i.e., functioning *normally*.

So, how could the naturalist reasonably ascertain whether or not *W*’s tusks are performing their hypothesized function *normally*? At this point, it surely seems that the

⁹ Carl G. Hempel (1966), *Philosophy of natural science*. Englewood Cliffs, N.J.: Prentice-Hall, Inc.

naturalist is forced to concede that the “normal functioning” of the waligator’s tusks must be established from reading off the “level” of actual performance of the waligator’s tusks (i.e., the level of performance in doing the task the tusks are inferred to be *for*) from the waligators thus far observed (How else could the naturalists possibly know how a new species normally functions?) This concession is crucial because it means that the *actual* level at which the species-*tokens* are performing the relevant function thus establishes what it is for the species-*type* to perform its function *normally*. The upshot is that it allows the naturalist to employ empirical science to measure the actual level of performance of the waligator’s functions and, as such, determine what level of performance of a function is, statistically speaking, “normal functioning”.

So one might then address the task of determining whether *W*’s tusks are functioning *normally* by comparing, say, the density of *W*’s tusks, the toxicity, and amount of toxin produced (or whatever biological mechanisms enable the waligator’s tusks to perform the function or functions which in solution to the qualitative issues are postulated as this structure’s function or functions) against the statistical norm of other relevantly similar (i.e., same age, sex, etc.) waligators. This statistical norm, according to the BST, determines which level of performance of a function (i.e., functioning) is requisite for *normally* and indeed *successfully* performing the function. Accordingly, only when the level of performance of *W*’s tusks are at or above the level of species-typical performance of the function (i.e., do not count as “subnormal” functioning) will the BST count *W*’s tusks to be “functioning normally”.

Here again, it is important to stress that at this point our group of naturalists may not be correct about their suggested solution to the quantitative issues about waligator-*type*

functioning—for the possibility remains that the actual measured levels of performance of the waligator’s functions are in fact unrepresentative of the species (e.g., what was measured to be statistically normal functioning is neither statistically normal nor successful functioning for the species). I return to this latter point in the next chapter.

But for now I hope it is clear that one may draw an important difference between “function”, “functioning” and “normal functioning”. Thus, a biological structure and/or mechanism (e.g., waligator’s tusks) may have a species-type function (the solution to the qualitative questions about its functions) that in a species-token (e.g., *W*) may (or may not) as a matter of fact have the capacity to perform its function at all, let alone at what is a statistically typical, i.e., normal level and, *a fortiori*, it may (or may not) be functioning at a level that is properly counted to be normal functioning (the solution to the quantitative questions about its functions).

1.2 *Narrowing the focus*

Let us press on by noting that a significant strength of the BST is its capacity to incorporate various different accounts of function.¹⁰ One might therefore say that the BST requires a *concept* of normal functioning, though it need not require any particular *conception* of normal functioning.¹¹ Of course, the naturalist will not willingly embrace

¹⁰ Boorse explicitly recognizes this: “...it is worth noting that my analyses of health and function are separable, in that one could ground the BST on a different analysis of function”. See Boorse (1997), pp. 10–11.

¹¹ Here I am drawing on the well-known concept/conception distinction made by Ronald Dworkin. See Ronald Dworkin (1977), *Taking rights seriously*. Cambridge, Mass.: Harvard University Press, pp. 134–36 & 226. Very briefly, Dworkin distinguishes between the general term or idea (the concept) and the criteria held to track what the concept means (a particular conception). Accordingly, people can have different, competing and, sometimes, incompatible conceptions of the concept of, say, “function” or “health” or...?

just any conception of function; only those accounts of function toeing the naturalistic line will be eligible for serious consideration.¹² In response to a multitude of problems, a significant number of competing accounts of function have emerged in the literature,¹³ which stand, initially at least, to be legitimate candidates to underpin the BST. What is to be stressed is that insofar as these problems run against attempts to advance a satisfactory value- and norm-independent account of function, these will be problems that the defender of the BST also must overcome. At any rate, for reasons that will become clear, if other objections strike forcefully against naturalist accounts of function, then my argument will only be stronger.

Nevertheless a persuasive claim to ground the BST on some particular account of function would in the end have to demonstrate its capacity and/or the incapacity of other candidate accounts to overcome legitimate objections. Moreover, those of us concerned to underpin health claims with the best conception of health and disease available have a powerful motive for insisting that the BST be grounded upon the best possible account of function: such an account would ultimately require a full analysis of the various competing accounts of function. While such an analysis would far exceed the scope of this chapter (and, indeed, this dissertation), we need not abandon the task at hand: to examine the

¹² I hope it is clear that the naturalist requires the BST to employ a value-free account of function. For this reason, the candidacy of those accounts of functions that, for instance, Perlman categorizes as ‘Non-Naturalistic’ and ‘Quasi-Naturalistic’ and Boorse labels ‘Value-Centered (VC)’ should be dismissed out of hand pending persuasive argument to the contrary. See Mark Perlman (2004), “The modern philosophical resurrection of teleology”, *The Monist* 87(1): 3-51, at pp. 6-10; and Boorse (2002), *op cit.*, pp. 67-68.

¹³ Good recent surveys on the different accounts of function proposed in the literature are to be found in André Ariew, Robert Cummins, and Mark Perlman (Eds.), (2002), *Functions: New essays in the philosophy of psychology and biology*. Oxford: Oxford University Press; Mark Perlman (2004), *op cit.*; and Arno Wouters (2005), “The function debate in philosophy”, *Acta Biotheoretica* 53(2): 123-151.

prospects for advancing a different naturalist account of function that would preclude the BST from essentially leaving itself open to a Cambridge-change objection. It is acceptable to press on because I argue that when it comes to health and disease the familiar accounts of function become impaled on the horns of a dilemma: either for one reason or another they provide an inadequate biomedical account of normal functioning or they leave the BST open to what I have called the Cambridge-change objection—either of which alternative fatally undermines the BST’s goal to provide medicine with a value- and norm-independent account of health and disease.

Another advantage of our limited focus is that we escape the need for a detailed survey of the numerous competing accounts of function. This is because, for reasons that will become clear, the structure of the Cambridge-change objection allows us to draw upon a division that has become commonplace in the function literature and to group function accounts according to the time period required for function ascription. This makes it possible for us to examine those accounts of function that might reasonably be considered candidates to ground the BST from the perspective of three general groups, which I call, respectively, *Backward-looking*, *Present-looking*, and *Forward-looking*.¹⁴

¹⁴ With these names I am following Perlman (2004), *op cit.*, who in turn credits Bigelow & Pargetter; but these are terms that are often found in the literature. To name but three notable instances, see: John Bigelow & Robert Pargetter (1987), *op cit.*; Godfrey-Smith (1994), *op cit.*; and C. Allen, M. Bekoff, & G. Lauder (Eds.), (1998), *Nature's purposes: Analysis of function and design in biology*. Cambridge, Mass: The MIT Press. It is not unusual, however, to see different names employed to reflect these three major strands in the philosophical function literature. For instance, Hardcastle distinguishes between backward-looking, forward-looking and causal-role approaches; and Boorse, citing Kitcher, distinguishes between: “distant past, recent past, present, and logical combinations thereof”. See Valerie Gray Hardcastle (1999), “Understanding functions: A pragmatic approach”, In V. G. Hardcastle (Ed.), *Where biology meets psychology: Philosophical essays* (pp. 27- 43). Cambridge, Mass: The MIT Press; Boorse (2002), pp. 65-66; and Philip Kitcher (1993), “Function and design”, *Midwest Studies in Philosophy* 18: 379–397. Drawing on Perlman’s taxonomy, I go on (in Section 3.1 below) to subdivide the ‘backward-looking’ category into

Despite differences in the time period required for a trait's effect to be properly ascribed a function, I argue that none of these three general groups succeed in avoiding the horns of the above-mentioned dilemma.

First, however, we need to identify, with as much clarity as possible, what it is about Boorse's underlying account of *normal functioning* (i.e., what it is to be a function and correspondingly of what it is to perform that function and of what normal functioning involves) that ultimately leaves the BST open to a Cambridge-change objection. This will position us to examine the prospects for advancing an alternative naturalist account of function and of normal functioning that would enable the BST to avoid the Cambridge-change objection. A further advantage of this approach is that it directs us toward the final account of health and disease that in the end I will be defending. With that in mind, we turn to the BST's account of "normal functioning".

2. The BST's account of "function" and thence of "normal functioning"

Because what I have to say will be largely critical, I want to make clear from the outset that in his most recent defence of the BST's account of function, Boorse responds persuasively, in my view, to what he recognizes to be ten leading objections to his account.¹⁵ To appreciate this, consider that Wouters proposes that the intuitions of most philosophers converge on fifteen requirements that an adequate account of function should

'distant/ancient past' and 'recent past'.

¹⁵ Boorse (2002), *op cit.*, pp. 75-105. Here, at p. 107, Boorse claims to answer all major objections except two, which he hopes to answer elsewhere: "attacks on the cybernetic analysis of goal-directedness and Amundson's assault (2000) [, *op cit.*,] on normal function". As far as I can see, none of his responses, as formulated, serve to avoid my Cambridge-change objection.

satisfy save for persuasive argument(s) to the contrary.¹⁶ Whether or not this assessment is correct is of no present concern;¹⁷ I mention Wouters, not to take issue with his claim, but rather, to acknowledge the difficulty of motivating which, if any, naturalist account of function would in fact fulfill the BST's goal to underpin medicine with a value- and norm-independent science of health and disease. That being said, the principle aim for the remainder of this chapter is to demonstrate that the criticisms advanced here are not limited to Boorse's particular account of function. The main point will be that any naturalist claim to underpin medical practice with a value- and norm-independent conception of health and disease defined in terms of a naturalist account of function will become impaled on the horns of the above-mentioned dilemma. To motivate this, it is important to get clear about the tremendous amount of weight the current framework of the BST must place on its account of function.

2.1 The fundamental role of the BST's account of function

To begin to appreciate the immense weight the BST places on its account of function, it will be helpful to explicitly recall what I suggested in Chapter 3 would be a useful expansion and clarification of Boorse's official definition schema:

¹⁶ Wouters (2005), *op cit.*, pp. 133-134. Here Wouters contends that a theory of function should distinguish functions from: side-effects of functions, accidentally useful effects, & vestiges; allow for maladapted functions; enable us to attribute functions to: so-called "instant organisms", items that do not actually perform it, malformed items that are incapable of performing their function, to traits that are selected against, & to parts and processes of sterile organisms, to name but 9 of the requirements.

¹⁷ Though it is perhaps worth mentioning that in my view Wouters' requirements capture all 8 of those objections that Boorse responds to, which are aimed directly at his account of function (the remaining 2 objections take issue with his account of biomedical normality). This is not to say, however, that Boorse agrees with all of Wouters' requirements, because he does not. Boorse, for instance, does not agree that a theory of function should allow for maladapted functions, which he claims are counterintuitive. See Boorse (2002), pp. 84-86.

(D*): The amplified version of Boorse's official definition schema:

- (1*) For most species the *reference classes* for that species will be those subsets of the members of the species that have *uniform* "functional design".
- (2*)(i) The *functional design* of the members of some reference class for a species S is that "internal functional organization" (of a physiological structure) of those members of that reference class which is (a) *statistically typical* of the members of that reference class *and* (b) *statistically typical* of the contributions that the internal functional organization in question makes to the survival and reproduction success of the *individual* members of the reference class, and
- (ii) Some function is the *normal function* of some internal physiological structure if and only if performance of that function is *statistically typical* in the ways (a) and (b).
- (3*) The *internal functional organization* (the functional design) of the members of some species S is *normal* if and only if it counts as being *statistically typical* in the ways mentioned (2*); and (b) an organism is *functioning normally* in respect to some aspect of its internal functional organization (its functional design) if and only if that organisms functioning is *statistically typical* in the ways mentioned in (2*).
- (4*) The *functional ability* of the internal functional organization (functional design) of some particular member of a species S is the ability that member's internal functional organization (functional design) to contribute to the survival and reproduction success of that individual.
- (5*) The *level* of the functional ability of the internal functional organization (functional design) of some particular member of a species S is a measure of the efficiency of that member's internal functional organization's (functional design's) contribution to the survival and reproduction success of that *individual* member *as compared to efficiency of contribution of the functional design of statistically typical members of the relevant reference class* as defined in (1*).
- (6*) A *disease* state of an individual member of a species is a state of the internal functional organization of that individual in which the functional ability of that internal functional organization to make a contribution to the survival and reproductive success of the individual is at a level below what is normal (i.e., statistically typical) for the relevant members of that species.

(7*) *Health* is the absence of disease.¹⁸

Obviously, given the notions “uniform functional design”, “normal function”, and “functional ability” Boorse intends the BST to be taken as leaning heavily on a notion of function.

Placing further demands upon the BST’s account of function are two less obvious, yet no less important, corollaries of (D*). The first corollary is that an organism’s biological fitness is relative to the fitness of other organisms—there is no notion of “intrinsic fitness” at work.¹⁹ There is a real sense in which this forces Boorse to champion the idea that “the normal is the natural—that health is conformity to a ‘species design’”.²⁰ And the “species design”, Boorse states, “is, in fact, simply those functions statistically typical in species members”.²¹ The upshot of the species design, so stated, is the emergence of an empirical ideal;²² it is a statistical abstraction²³, empirically derived²⁴ from the functional design of individuals²⁵ and, as such, natural science is taken to provide the BST with the requisite conception of species-typical design on which its value-free health and disease ascriptions may depend. Boorse clearly assigns this fundamental role to the concept of species design when he says,

¹⁸ See also Boorse’s official definition schema, which I explicitly outlined in Chapter 3, Section 1 as (D).

¹⁹ Boorse surely would agree: “Our guiding principle has been the species relativity of health. We have supposed that the basic notion is ‘X is a healthy Y’ — that it is by comparing X with its reference class Y that one distinguishes the way X does function from the way it ought to.” See Boorse (1977), *op cit.*, p. 562

²⁰ Boorse (1997), *op cit.*, p. 7. Boorse has subscribed to the view that “the normal is the natural” since his earliest writings on health and disease. See Boorse (1975), *op cit.*, p. 57 and (1977), *op cit.*, p. 554.

²¹ Boorse (1997), p. 18. Elsewhere Boorse writes: “Our species and others are in fact highly uniform in structure and function; otherwise there would be no point to the extreme detail in textbooks of human physiology. This uniformity of functional organization I call the species design”. Boorse (1977), p. 557.

²² Boorse (1977), p. 557.

²³ *Ibid.*, p. 558.

...health is conformity to a “species design.” In modern terms, species design is the internal functional organization typical of species members...²⁶

...since the functional organization typical of a species [i.e., the “species design”] is a biological fact, the concept of disease is value-free. Whether or not an organism is diseased can be settled in principle by the methods of natural science.²⁷

It is difficult to see, however, how natural science provides the BST with the requisite value- and norm-independent conception of species design (taking species design to be “those functions statistically typical in species members”) unless it does so by using some account of how human organisms actually do, in fact, *function*. Accordingly, it seems the very plausibility of Boorse’s fundamental claim that the species design is fixed by nature depends on a satisfactory account of function.

All of this is to say that the BST’s account of function clearly underpins a fundamental component of the BST—a conception of species design. But the present schema and the stated goal of the BST place still further demands on its account of function. This is because the BST’s framework is such that species design underpins its conception of normal functioning,²⁸ which Boorse asserts *is* medical normality²⁹ and, according to which, *is* health.³⁰ This effectively makes the BST’s account of function the

²⁴ Boorse (1997), p. 32.

²⁵ Ibid., p. 89.

²⁶ Ibid., p. 7.

²⁷ Boorse (1976a), *op cit.*, p. 63.

²⁸ This is clear from (D*) and also Boorse’s definition of ‘normal functioning’ cited in footnote 30 below.

²⁹ When underpinned by an appropriate function concept, Boorse states that, “medical normality and statistical (nonsub)normality are the same thing”. See Boorse (1997), p. 18. This is consistent with Boorse’s more recent, and more detailed, definition of ‘medical normality’ as: “the readiness of each internal part to perform all its normal functions on typical occasions with at least typical efficiency”. See Boorse (2002), p. 90, and also pp. 72, 93 & 108.

³⁰ The direct link between normal functioning and health is explicitly stated: “*Health* in a member of a reference class is *normal functional ability*”, which Boorse defines as, “the readiness of each internal part to

driving force behind several of Boorse's most important claims, such as, a *value-free*

(i) conception of species design; (ii) conception of normal functioning; (iii) conception of medical normality; and (iv) account of health and disease.

The second, and related, corollary I wish to draw attention to is this: To count as being healthy, it must be the case that all of the function *normal* for the species (i.e., what is identified as solutions to the *qualitative* issues about functions) are found to perform at a non-subnormal level (i.e., what is identified as solutions to the *quantitative* issues about functions), given a normal (i.e., statistically typical) environment;³¹ when this is not the case, the individual will count as being diseased.

On this framework, what essentially matters for health (and disease) is that an individual's "normal functioning" (i.e., what is identified as solutions to the quantitative issues about functions) conform to (diverge from) the statistical norms of the appropriate reference class. What is to be stressed is that not every biological part and process will properly merit function ascription. For (D*)—and, to be sure, Boorse's (D)—clearly demands that the only biological mechanisms the BST counts to be *relevant* candidates for function ascription are those that contribute to individual survival and reproduction.³²

perform all its normal functions on typical occasions with at least typical efficiency" — the exact definition we just saw that Boorse, 25 years later, gives for "medical normality"! See Boorse (1977), p. 562, italics his. Boorse considers this conception of health to be in perfect concert with scientific medicine: "The thesis that health is normal functioning is essentially a medical truism." Boorse (1977), p. 563.

³¹ After proposing what he calls his "final definition", i.e., (D), Boorse later concedes the need to "supplement, not replace the definitions quoted earlier", with a concept of a normal environment. See Boorse (1997), pp. 83-84. This concession is necessary for Boorse to claim that a statistically normal condition can only be a disease if it is caused by the environment. See (D3), Boorse (1975), p. 65 & (1976a), p. 79. It also provides the wherewithal to remove the force of those objections that invoke "special environments" to claim that uncontroversial diseases need not always impair individual survival and reproduction. See Boorse (2010), *op cit.*, p. 77.

³² Boorse has recently written of his goal-directed account of function that it, "...need not claim...that every

The above points might be viewed as lessons from the thought experiment described earlier in this chapter. Importantly, and this is crucially important, depending on the account of function grounding the BST, not every biological mechanism that might be plausibly said to contribute to individual survival and reproduction need be properly considered a “function”, and thus a “normal function” relevant to the determination of health and disease. Quite simply, different accounts might stipulate different time periods required for a trait’s effect to be its function. I will have much more to say on this below. For now, however, it will suffice to point out that here is another reason to insist that the account of function wedded to the BST is of great importance: ultimately what the BST’s counts to be a relevant “normal function” (a solution to the qualitative issue) depends on the particular account of function that grounds it. That is to say, what the BST counts to essentially matter for health and depends upon the account of function grounding it. A vital role indeed.

The aim of this section has been to make two crucial points clear. First, Boorse assigns a fundamental role to the BST’s account of function. Second, since not every account may view the same biological mechanisms to be “functions”, which particular account of function underpins the BST is of great importance in various ways, not the least of which is whether or not it will be such that the BST essentially is open to Cambridge-change objections.

biological mechanism serves a goal, only that every biological mechanism with a function does so.” Boorse (2002), pp. 77-78.

2.2 *Why the BST's current account of function is open to Cambridge-change objections*

As we have seen, the fact of the matter, simply put, is that the BST is forced to maintain that the health of an organism *is* relative to the fitness of other organisms of the same species. This forces the BST to insist that what essentially matters for health (and disease) is that an individual's relevant functions conform to (diverge from) the statistical norms of the appropriate reference class—it is, of course, *this* point that makes it the case that the BST is open to Cambridge Change objections. What follows, I argued in Chapter 3, is that the current framework of the BST essentially involves what I have called a Cambridge-change criterion, which allows a “change” in *relational* properties to warrant a change in the disease-status of an individual. Close examination revealed that this implicit criterion commits the BST to the troubling view that an individual may go from being diseased to healthy, or vice versa, without any physiological change in that individual—a sufficient change in the statistical norms of the reference class will suffice. Emerging from this view, I then went on to argue, are two intractable problems that the BST must come to grips with. At the heart of these problems is the fact that the statistical norms of the BST's reference classes *will* undergo changes.³³

I have tried to show (in Chapter 3, Section 3) that the BST's account of function is framed in a way which leaves it possible for statistical norms to undergo changes, and

³³ In Chapter Three (Section 3.1), I argued the statistical norms the BST employs *will* be “dynamic” for at least two reasons: Briefly, they are (i) the BST must draw upon age-related reference classes with crucially different statistical norms; and (ii) the BST is committed to the view that the relevant statistical norms may change as the various individuals who presently compose the various reference classes change individually or as the membership of the reference class changes.

sometimes even rapid changes. However, I have in fact said very little about why it seems that a satisfactory account of *function*, on which to ground the BST upon, must insist that the statistical norms of the BST's reference classes *will* undergo changes. Motivating this view will take some elaboration, which is the primary aim for the rest of this chapter.

To that end, I want to demonstrate that there are at least two important reasons to insist that the naturalist is crucially limited to just how far back in the history of our species the BST's reference classes may draw its members from. I argue that an important consequence of this limitation is that a satisfactory account of function is committed to the view that the statistical norms of the BST's reference classes will undergo changes. This argument, however, requires some further examination of the crucial link the BST makes between its conception of species design and normal functioning, to which we now turn.

2.2.1 Species design, functions, and normal functioning

We saw (in Section 2.1) that the BST connects a conception of function with a conception of species design, which results in a conception of normal function that is relative to the level of functioning of other organisms that are members of the same species. Thus the statistical norms that the BST draws upon must in principle be responsive to changes in the level of functioning of the present individuals who necessarily make up the reference classes. I take this to be a straightforward implication of the fact that the current framework of the BST is grounded upon a naturalist account of function. Otherwise it is difficult to see how the BST upholds what is arguably Boorse's most fundamental claim:

the classification of human states as healthy or diseased is an objective matter, to be read off the biological facts of nature without the need of value

judgements”.³⁴

Much less clear, however, is the extent to which changes in the relevant states of those individuals who presently compose the reference classes will actually impact the statistical norms. To be clear, there are at least two distinct issues here. There is the matter of tracking the relevant change(s) in the level of performance of a function (i.e., which is grounded in solution to the quantitative issues) that occurs in individual members presently composing the reference classes. But I take this to be an empirical matter that modern science has the capacity to handle.

Perhaps a more serious problem then is the matter of just how far back in the history of our species must the BST’s reference classes draw its members from. The time-slice one settles on carries important implications. For example, the smaller the time-slice of a species’ history the naturalist includes, then the fewer number of past individuals’ functioning will be relevant when it comes to determining the statistical norms of the BST’s reference classes. And thus it seems, the greater the extent to which changes in the relevant functions of present individuals will alter the statistical norms of the reference classes.

The fundamental issue is this: What is the time period that the BST’s account of function counts to be relevant in determining when a trait’s effect becomes a function? That is to say, what we therefore want to know is how far back the species’ history one must go to acquire the “species-typical” (i.e., normal) functioning. Unfortunately, Boorse seems content to shed little light on the matter and, worse still, it is not at all clear what he

³⁴ Boorse (1997), p. 4.

does say at various points is entirely consistent. Nonetheless, it is worthwhile to have the following quotations called to our attention:

- (a) ...I do see a species as extending over time as well as space, so for me some of the past affects what is species-typical.³⁵
- (b) But in calling a function species-typical, one generalizes over a reference class of species members, including past ones.³⁶
- (c) ...the concept of species-typical is not overly historical...if one brings in a chunk of species history, as I do to make the species design more stable...³⁷
- (d) ...to use an extended time-slice of the species. Obviously some of the species' history must be included in what is species-typical...Actually, any time-slice shorter than a lifetime or two seems too short for the very idea of a species-typical functional design...³⁸
- (e) Diseases are, so to speak, failures to get as far as the rest of the species has been for millennia.³⁹
- (f) ...I can say, universal non-function becomes normal, or the organ vestigial, if the non-function persists for a significant period—for long enough.⁴⁰
- (g) On all but evolutionary time scales, biological designs have a massive constancy vigorously maintained by normalizing selection. It is this short-term constancy on which the theory and practice of medicine rely.⁴¹
- (h) While my views on time are vague, my concept of a reasonable time-slice of a species seems analogous to the species concept in paleontology, where... vague boundaries are the rule.⁴²

All the above statements seem to state in one way or another that the time-slice used to determine what is counted to be “species-typical” (i.e., normal) functioning extends into

³⁵ Ibid., p. 66.

³⁶ Ibid., p. 118.

³⁷ Boorse (2002), p. 86, at fn #26.

³⁸ Ibid., p. 99.

³⁹ Boorse (1977), p. 563.

⁴⁰ Boorse (2002), p. 98.

⁴¹ Boorse (1977), p. 557.

⁴² Boorse (1997), p. 66.

the past. But importantly, (a), (b), (c) and (d) explicitly state that the species-design will appeal to only *some* of the past, not *all* of it. This general requirement to include the past, but only some of it, as we shall see in due course, distinguishes Boorse's account from other notable accounts of function. Presumably (d) gives a reasonable idea of the minimum amount of the past we must include: an adequate time-slice of species' history must be no less "than a lifetime or two". But how far back do we have to go to ensure a suitably broad time-slice of our species? Is the BST to appeal to a "millennia" of human history as (e) suggests; or would this run against Boorse's claim in (c) for the "chunk of species history" not to be "overly historical" and/or the "short-term constancy on which the theory and practice of medicine rely", claimed in (g)?

To be told, as we are in (f), that Boorse is going to appeal to "a significant period" that is "long enough" is of little help. And, moreover, a reasonable person may legitimately wonder why Boorse in (h) apparently thinks it is either informative or appropriate to compare his "vague... concept of a reasonable time-slice of a species" with "the species concept in paleontology". As formulated, it is not informative because it does not in any way help us to identify the requisite time-slice of the life of the species. Now I don't know how Boorse understands the species concept in paleontology (he doesn't say), but it seems peculiar to suppose, as he claims in (g), that paleontology (rather than, say, physiology) provides the requisite "short-term constancy" of biological design "on which the theory and practice of medicine rely".⁴³ At any rate, if paleontology does establish the

⁴³ The peculiarity of Boorse's appeal to the paleontological concept of species design may be thought to extend further given that he states: "Even today physiological function statements are not usually supported by, or regarded as refutable by, evolutionary evidence." Boorse (1976b), *op cit.*, p. 74.

“species-typical functional design” (as its solution to the qualitative issues about functions) of (d), then Boorse should demonstrate as much.

So what are we to take from my presentation of Boorse’s position? Well, in light of the above, I think it is important to focus on the heavy lifting the BST demands for a particular time-slice of our species’ history to accomplish. First and foremost, Boorse is quite clear that if it is to be possible to find a conception of species design in which species design is *uniform* and *stable*, *some* time-slice of our species’ history must be appealed to. So it seems we have some sense of how far back in the history of our species the BST’s reference classes must draw its members from. And the time-slice invoked must include enough of our species’ history to establish a uniform and stable species design. There are, however, at least two important reasons to insist that the BST is crucially limited to just how much of the past it may appeal to establish the requisite species design.

Reason #1: An appropriate conception of “species design”

The first reason (a reason that can be extracted from the Boorse’s statements quoted above) is that he needs to restrict the scope of the past in order to ensure that his account has the resources to make it possible to identify the non-functioning vestigial organs he speaks of in (f). Motivating Boorse here is the undeniable fact that the typical human body does not currently function as it always has functioned. The stock example the functions literature takes to demonstrate this is the appendix; but as noted in Chapter 4, Jacobson’s Organ, “junk” DNA, *cutis anserina* (goose bumps), *plica semilunaris* (third eyelid), ear muscles, wisdom teeth, coccyx, and pharyngeal arches in human embryos are counted to be vestigial. Most importantly, however, is the fact that standard medical practice is not

concerned with the function a physiological mechanism may have performed in our distant ancestors but no longer performs. Quite simply, if the BST is thus to provide an adequate value-free foundation for medicine (as Boorse says it does), then it must possess a principled way⁴⁴ to differentiate between, for instance, the normal functioning of the appendix in our distant ancestors and the universal non-functioning of the appendix⁴⁵ modern medicine describes today. This will require the BST to possess a conception of species design that accurately reflects how human beings uniformly function at this point in our species' history.

What the preceding shows is that an appropriate conception of species design will be one that has the BST including a particular time-slice of the species' history that is both suitably broad and narrow enough. For an account of the species design to be adequate it must specify a time-slice of the species' history that, on the one hand, is broad enough to sufficiently establish a uniform and stable species-*type* design; and, on the other hand, is narrow enough to accurately reflect the typical functioning of present species-*tokens*.⁴⁶ The latter requires, of course, that the BST 's account of *normal function* must be limited to the amount of the past an adequate time-slice of the species' history will include. Boorse is thus quite right to insist that species-typical functioning will not be overly historical and, moreover, in doing so he grounds the BST on an account of function that

⁴⁴ I elaborate on this below.

⁴⁵ Some do claim, however, that the appendix performs a current function. I will not pursue this line because my point is that the current human body does not function as it always has — a point that clearly does not depend on any particular view of the appendix.

⁴⁶ Boorse emphasizes a type/token distinction as he responds, persuasively in my view, to several notable criticisms raised against his account of function such as, maladaptive functions, functions vs. accidental effects, and unperformed functions. See, for instance, Boorse (2002), pp. 84-90 and 92-93.

would appear from this vantage to support an adequate conception of species design.

The second reason to insist that the BST must limit the amount of the past it may include is, however, by far the most persuasive. For it stems from what I take to be Boorse's most fundamental claim about the BST: "the classification of human states as healthy or diseased is an objective matter, to be read off the biological facts of nature without the need of value judgements".⁴⁷ For this reason, I will argue that we have a knockdown reason to insist not only that the BST is crucially limited to just how much of the past it includes but also that this is a matter that current empirical science must settle. To argue for this stronger claim, however, will require a brief examination of Boorse's account of function. To this we now turn.

2.2.2 Boorse's "general goal-contribution" account of function

Boorse defends what he calls a *general goal-contribution* (GGC) account of what it is to be a function of some organ or structure. What distinguishes Boorse's GGC account from many other notable accounts of function is the way in which his conception of function connects with goal-directedness: "Functions are, purely and simply, contributions to goals".⁴⁸ With this in mind, Boorse, not surprisingly, views the GGC account of function to be the ideal candidate account of function to underpin the BST.

In the literature there have, of course, been various accounts of what it is to be a function that have been goal directed in their structure – the work of Carl Hempel⁴⁹ of

⁴⁷ Boorse (1997), p. 4.

⁴⁸ Boorse (1976b), p. 77.

⁴⁹ Carl G. Hempel (1965), "The logic of functional analysis", in *Aspects of scientific explanation and other essays in the philosophy of science*. New York: The Free Press, 297-330.

course springs to mind and does that of Ernst Nagel.⁵⁰ The key contention of Boorse's particular version of a GGC account of function is the way in which, as we will see, he spells out his view that his account "takes goal-directedness to be an objective, non-mental property of all living organisms" and, as such, it "lets function statements be literally true throughout the whole biological domain".⁵¹ More formally, Boorse defines his goal-directed theory of biological functions as follows:

(B1) "A function of *X* is *Z*" means that in some contextually definite goal-directed system *S*, during some contextually definite time interval *t*, the *Z*-ing of *X* falls within some contextually circumscribed class of functions being performed by *X* during *t*—that is, causal contributions to a goal *G* of *S*.⁵²

This formulation, Boorse says, is meant to provide a general account of strong function statements. However, in a more recent publication, Boorse states that what he calls a *weak function statement* reveal what a function is. Stipulating that *G* is a goal of system *S* at time *t*, he writes:

(B2) *X performs the function Z* in the *G*-ing of *S* at *t* if and only if at *t*, the *Z*-ing of *X* is a causal contribution to *G*.⁵³

Boorse maintains that there is no substantive difference between (B1) and (B2).⁵⁴

This claim need not detain us here, however. What is important to stress for our present

⁵⁰ Ernst Nagel (1977), "Teleology revisited", *The Journal of Philosophy* 74: 261-301. See further Wachbroit (1994), *op cit.*, pp. 582-584.

⁵¹ Boorse (2002), pp. 63-64.

⁵² Boorse (1976b), p. 82.

⁵³ Boorse (2002), p. 70. Italics his. Here Boorse also mentions in a footnote that "Equivalent variants are '*X serves the function Z*' and '*X functions as a Z-er*'".

⁵⁴ "I suggest that the distinction between weak and strong function statements is illusory to this extent: there is no important conceptual constituent of the idea of "the function" or "a function" which is missing in "performing the function." Boorse (1976), p. 80.

purposes is that Boorse is championing what he refers to as a contextual view⁵⁵ of function, according to which all function statements require particular interests to impose contextual limitations upon three variables—the system *S*, the time interval *t*, and the goals *G*. The key idea of Boorse’s GGC account is that what converts *a* function into *the* function will be particular “background interests in the context in which the function statement is made”.⁵⁶

Importantly, Boorse concludes that the fact that function statements work in a context-sensitive way explains the variety of legitimate responses that may be given to the question, “What is the function of X?” Indeed, the payoff for the contextual view, Boorse argues, is that it makes clear what distinguishes *the* function of X from *a* function of X will be the contextual limitations imposed upon the three variables. Thus, for example, the marine biologist seems correct to assign different functions to whale bones when it limits the system *S* to either the individual whale, the species or from the perspective of an ecosystem as one would do when interested in a whale fall. Boorse insists, however, if one is interested in the standard medical conception of normal function then the following two contextual limitations are in order:

- (1) The system *S* is the species *Homo sapiens*.
- (2) The goals *G* are individual survival and reproduction.⁵⁷

⁵⁵ Boorse (1976b), p. 83. This view is also referred to in the literature as a *pragmatic* view of function. See, for instance, Valerie Gray Hardcastle (2002), *op cit*.

⁵⁶ Boorse (1976b), p. 81. As I read Boorse, this is because biological mechanisms of organisms are directed towards achieving multiple goals and no fixed property serves to identify *the* true goal from *a* goal. A very similar reading of Boorse is given by Mahesh Ananth (2008), *In defense of an evolutionary concept of health*. Aldershot: Ashgate Publishing Co., at pp. 85-86.

⁵⁷ See, for instance, Boorse (2002) at p. 72.

But what about the contextual limitation imposed upon the third variable, ‘the time interval t ’? Boorse, recall, defines *normal functional ability* and medical normality (both are said to share the same *definiens*⁵⁸) in terms of functional readiness. Why in terms of functional readiness? Well, the key reason seems to be two-fold: (i) to account for the fact that most biological functions are performed on specific occasions, which may be rare; and (ii) to echo modern medicine’s view that one may be diseased even if the occasion should never arise for which one would thus be unable to perform the relevant function.⁵⁹ Thus, for example, the function of an individual’s sperm is to fertilize an ovum and, as such, the inability to perform this function would count as a disease even if the occasion-specific opportunity to fertilize an ovum should never arise.

Additionally, however, Boorse seems to be motivated by what he takes to be a fact of the discipline of physiology, the fact that “[e]specially in human physiology, the focus of inquiry is...[on] how the mechanism currently operates and how to keep it in shape”.⁶⁰ More persuasive perhaps is the undeniable fact that standard medical practice is concerned with *current* functional ability. Modern medical practice, for instance, does not count a person to be unhealthy simply because of the fact that that person’s appendix no longer functions as appendixes presumably once did.

To its credit, the current framework of the BST easily accounts for medical practice’s ruling on the appendix. In part this is because of the BST’s conception of species design, which includes only some of the past, not all of it. Thus the issue of how

⁵⁸ See footnotes 29 & 30, in Section 2.1 of this chapter.

⁵⁹ See Boorse (2002), p. 93 and (1977), p. 562.

⁶⁰ Boorse (1976b), p. 85; see also p. 76.

the appendix functioned in the distant past need not occupy the naturalist's attention. But it is also because Boorse grounds the BST on an account of function that is explicitly concerned with "how the mechanism currently operates"⁶¹ or, more accurately, "the actual contribution to a goal".⁶² And, moreover, when grounded on a conception of normal function (i.e., medical normality) that limits the time-slice t to the present and near future, the BST appears well-positioned to echo modern medicine's concern with current functioning.

Thus Boorse would seem to show an acute awareness of a need to insist that the BST's concern with the standard medical conception of normal function imposes a contextual limitation upon the time-slice t to the present and near future. In light of this, it is difficult to overstate the weight Boorse ultimately has the framework of the BST place upon the GGC account of function. First, Boorse views the GGC account of function to impose three legitimate contextual limitations upon the BST. One of these, on the one hand, limits the time-slice t to the present and near future, a point which he takes to legitimately ground his account of *normal functional ability* and, as such, his account of medical normality in terms of functional readiness.

On the other hand, however, the BST's interest in providing an account of species design that can plausibly be counted stable, uniform, and accurate and that reflects the way in which our present biological mechanisms contribute to individual survival and reproduction legitimately requires that the account of how the time-slice is specified

⁶¹ Ibid., p. 85.

⁶² Boorse (1997), p. 66.

include reference to some of the past, but not to all of it. Boorse, then, clearly takes these contextual limitations to impose a principled way to distinguish between functions, past and present. Importantly, this distinction enables the BST to echo both physiology's and modern medicine's concern with current functioning and, as such, it provides the BST a powerful resource to track the standard medical concept of normal function.

We now have a sufficiently detailed explication of Boorse's account of function against which to explicitly motivate what I want to suggest is a second, more persuasive, reason for insisting that not only is the BST crucially limited to just how much of the past history of our species it includes, but also that there is a knockdown reason for acknowledging that the matter is a matter which current empirical science must settle. I would like to add that my discussion here will anticipate and convincingly set aside a tempting naturalist rejoinder to my Cambridge-change objection.

Reason #2: An epistemological price

What is to be stressed is that the BST's conception of normal functioning is grounded upon a particular view about how best to address what I earlier called the qualitative and quantitative issues about normal function.⁶³ In short, it is so grounded because and insofar as the BST requires that a "normal function" (i.e., medical normality) be a function that performs not at just any level, but at or above some level, a level which is determined statistically. Thus the framework of the BST demands that for a biological mechanism (an individual's species-token part) to be "functioning normally" (i) it must contribute to that

⁶³ See also Kingma (2010), *op cit.*, p. 245.

individual's survival and reproduction in accordance with its species-typical design (the solution to qualitative issues about normal function) and (ii) it must perform at or above the level of the relevant statistical norm (the solution to quantitative issues about normal function).

With the above in mind we can see that the species design, which Boorse insists includes a particular time-slice, i.e., some of the past but not all, of the species' history clearly helps to determine what counts as normal function (i.e., what is the solution to the qualitative issue about function). After all, as formulated, it is the conjunction of the GGC account of function and the BST's conception of species-typical design that grounds claims about which biological mechanisms have species-typical functions relevant for health and disease ascriptions, which in turn establishes species-*token* functioning, normal or otherwise.

So, to the extent that our present biological processes may be said to be different from those of our ancestors different time-slices of our species' history would, it would seem, establish different species-typical designs and, as such, establish different functions as species-typical. This is one way in which the time-slice the BST's conception of species design includes carries important implications for what is offered as the solution to the qualitative issue about normal function.

The time-slice the BST includes also carries important implications for what is offered as the solution to the quantitative issues concerning normal function, i.e., issues about the level of the contribution made by biological mechanism to (on Boorse's theory) the survival and reproduction success of that *individual* species member. To illustrate, consider the following surely uncontroversial statement: The less the BST's requisite time-

slice extends into the past history of our species, the fewer the number of past individuals its reference classes will include as members. Given the current formulation of the BST, however, one may say that this properly implies another consequent, namely, that the level of contribution of a lesser number of past individuals will serve to determine which level of performance of a function (i.e., functioning) is requisite for a goal of a function to be successfully realized.

This is an important implication because changes in the levels of performance of a function of *present* individuals will *ceteris paribus* alter the relevant statistical norms of the reference classes to a greater extent than would be the case were the reference classes to include more past members' (obviously unchanging) levels of performance of a function. And conversely, insofar as the greater the time slice of the species' history the BST appeals to includes more members, the less we would expect the statistical norms of the reference classes to be altered from *present* changes in levels of performance of a function.

Perhaps it therefore might be thought that stabilizing the statistical norms by including a greater time-slice of the species' history—but not so much of the past as to undermine the BST's claim to employ an accurate conception of species design—would effectively count as a complete reply to my objection that the BST essentially involves a criterion open to Cambridge-change objections. This line of thinking, however, faces an insurmountable problem: If the BST includes enough of the distant past to plausibly avoid the Cambridge change objection, then the BST becomes an inadequate candidate for the fulfillment of Boorse's aim of underpinning medicine with a theoretical, value-free account of health and disease. (It is perhaps worth mentioning here as an aside that this

problem gives rise to other serious difficulties, a number of which will be discussed in the subsequent sections).

To see this one needs to remember that Boorse's account of function is rightly concerned with the *actual* contribution to a goal which some biological mechanism makes. Accordingly Boorse states, "my reference class includes past members, but I only count how their parts functioned while they were alive".⁶⁴ Now I have suggested that the key contention of Boorse's GGC account of function is the claim that it "lets function statements be literally true throughout the whole biological domain". For this, then, to be the case presumably establishing the level of species-type normal functioning (i.e., the solutions to the quantitative issues about functions) must be simply a matter of empirical science objectively measuring the level of functioning of the relevant species-token's parts *while they were alive*.

Otherwise it is difficult to see how it can be said that the BST upholds what we saw above are surely two of Boorse's most important claims: (i) that function statements are literally true; and (ii) that the distinction between healthy and diseased states is an objective matter, to be read off the biological facts of nature. Thus any naturalist that attempts to stabilize the statistical norms by including a greater time-slice of the species' history must pay a significant epistemological price. That is, the naturalist must have an objective, value- and norm-independent claim to know (at least in principle) the "literally true" levels of functioning of the individuals, past and present, it includes in the BST's reference classes.

⁶⁴ Boorse, (1997), p. 91.

Presumably one obtains the requisite objectivity when the level of performance of a function is, as just noted, read off the biological facts of nature. Naturally I concede modern science's capacity to establish the level of actual functioning by reading off the biological facts of *present* individuals. But surely matters are different when it comes to *past* individuals.

No one, after all, disputes that current empirical scientists cannot go back in time to when our deceased ancestors were alive in order to read off the biological facts of nature the level at which they actually performed when they were alive. As far as I can see, if the levels of performance of a function of past human beings are presently unknowable (at least in principle) to modern empirical science, they will remain unknown. At any rate, the BST's statistical norms cannot advance health and disease ascriptions derived from levels of performance of a function for which the requisite biological facts are unknown and continue to consistently uphold all of Boorse's most important claims. So it surely seems that the current framework of the BST must demand that the naturalist pay an epistemological price: its reference classes must include only those past human beings for which the requisite levels of functioning when they were alive is currently known (or at least currently knowable) to modern empirical science.

And if one is to pay this epistemological price, then we have a knockdown reason to insist that there is limits to how far back in the species' history the naturalist can legitimately draw its reference-class members from. For the naturalist requires that the quantitative issue of which level of functioning is requisite for a goal of a function to be successfully realized be an objective matter, read off the biological facts of nature. Once this is conceded, however, then just how far back in time the BST may extend reference

class membership is a matter for which modern empirical science will have the last word.

Now granted it is a bit unclear how far back in time the BST's reference classes may then draw its members from, since it is unclear the extent to which empirical science has an objective, value- and norm-independent claim to "literally true" levels of the functioning of past individuals while they were alive. But interestingly, in raising a very powerful objection against etiological accounts of function, Boorse effectively precludes the naturalist from extending the BST's reference class membership to past human beings whose levels of functioning is not already currently known by modern science,

How can a purely evolutionary account determine the mean and endpoints of normal [statistically typical] function? One obvious difficulty, of course, is that heart rate, blood pressure, blood urea nitrogen, and so on of past human beings are unknowable to contemporary medicine.⁶⁵

To be clear, this is part of Boorse's specific objection against the notion that Neander's etiological interpretation of "normal functioning" captures the standard biomedical view of normality.⁶⁶ The conclusion Boorse draws from this attack is that no etiological account can match the basic logical features of biomedical normality without invoking a conception of statistical normality; however, we needn't get bogged down in the details. It will suffice to note that Boorse correctly deems his attack to strike forcefully against all etiological accounts, which link function statements with the distant past.

Most important, for our purposes, is the need to recognize that the foregoing part of Boorse's objection also targets the naturalist attempt to stabilize the statistical norms of the

⁶⁵ Boorse (2002), p. 101. The bracketed phrase is added.

⁶⁶ See Ibid., pp. 99-102.

BST's reference classes by including a greater number of past human beings. That is, the naturalist also runs against Boorse's "obvious difficulty" of trying to account for requisite levels of functioning that he takes to be unknowable to contemporary medicine. And *a fortiori* Boorse's objection strikes with increasing force the deeper one looks into the past to stabilize the statistical norms; since the further into the past the BST extends to its reference class membership, the further it strains modern science's capacity to establish an objective, value- and norm-independent claim to the requisite levels of functioning.

Thus, the naturalist has further reason to limit the scope of the particular time-slice from which the BST draws its members. To avoid succumbing to Boorse's powerful objection against grounding the biomedical concept of normal functioning solely upon an etiological account of function. Moreover, we have further reason to insist that modern science settles the matter.

2.2.3 Section Conclusion

Let me sum up. In this section, I have attempted to show the tremendous amount of weight the current framework of the BST places on its account of function. To that end I have tried to show, sometimes in great detail, the way in which Boorse's account of function underpins vital components of the BST—specifically, species design, normal functioning, and medical normality. It follows from this, I have suggested, that the BST's account of function crucially informs two distinct and different issues: First and foremost, it must speak to which biological processes are functions relevant to health and disease (i.e., the solutions to the qualitative issues about functions); and, second, it must speak to which level of performance of a function is requisite for a goal of a function to be

successfully realized (i.e., the solutions to the quantitative issues about functions).

This distinction between qualitative and quantitative issues concerning functions and normal functions is important because it brings to light the distinct yet interdependent roles the BST's account of function must perform: it must underpin the BST's requisite conception of species design, from which the BST's conception of qualitative normal function (i.e., *species-type* normal function) emerges; and it must underpin the BST's requisite conception of medical normality in terms of functional readiness, from which its conception of quantitative normal performance of a function (i.e., *species-token* normal functioning) emerges. The result is that what the BST counts to essentially matter for health and disease may vary depending upon the account of function grounding it.

The result, however, yields a significant caveat, to wit, that the naturalist is limited to the amount of the past history of the species its account of function can reasonably include. In a nutshell, this is because of the way in which the BST's account of function underpins its conception of *normal functioning*, which Boorse asserts to be "health" and "medical normality". Boorse certainly intends his conception of "normal functional ability" to be tantamount to "health" and "medical normality"; thus, his conceptions of "health" and "medical normality" are grounded on what seem to be a particular conception of (normal) function and (normal) functioning. A closely related demand of the BST's account of function—indeed, an integral part of Boorse's assertion—is that it should yield health and disease ascriptions that do not too much run against our basic commonsense intuitions or, perhaps more importantly, the many surely noncontroversial health and

disease claims of modern medicine.⁶⁷ It should not, for instance, have the BST presently count a 21st century individual to be diseased simply because of her vestigial appendix.

I have argued that the BST's interest in finding an account of species design on which species design will be reasonably stable, uniform and accurate species design must reflect the way in which our 21st century biological mechanisms presently contribute to our individual survival and reproductive success; thus the account of species design legitimately requires reference to a time-slice which includes some of the past, but not all of it. I have also argued that the BST's claim to advance a value- and norm-independent empirically derived statistical analysis of normal functioning (i.e., the solutions to the quantitative issues about functions) forces the naturalist to pay a significant epistemological price. That is, the BST must include only those past human beings for which the requisite level of performance of a function when they were alive is currently known (or at least currently knowable) to modern empirical science. Together these two reasons—an appropriate conception of species design and an epistemological price that must be paid—along with Boorse's own powerful objection against grounding the biomedical concept of normal functioning solely upon an etiological account of function forces the naturalist to ground the BST on an account of function that requires only a

⁶⁷ Here what I mean by *noncontroversial* health and disease claims are simply those claims made by modern medicine that, crudely put, would be highly counterintuitive to have an account of health explain away or overrule. Disease claims, for example, such as pancreatic cancer, tuberculosis, cystic fibrosis, and emphysema, which are surely genuine diseases; and, conversely, pregnancy and drapetomania, which are surely not. I believe Boorse would agree with this demand; for he says that “[t]he real threat to the BST is, of course, cases where it and medical usage clearly divide”. Boorse (1997), p. 19.

limited appeal to the past history of our species.

3. Important implications

In this section, I use Boorse's GGC account of function and the above insights to argue that important problems arise for those naturalist accounts of function that might reasonably be considered candidates to ground the BST. I specify the problems that arise for the familiar accounts of function from the perspective of three general groups, which, as stated earlier, I will call Backward-looking, Present-looking, and Forward-looking, concluding that all three groups fail to offer the BST a viable naturalist candidate account of function. In particular, I argue that none of these three general groups succeed in avoiding the horns of a dilemma: Either (i) they, for one reason or another, provide an inadequate biomedical account of normal functioning and/or (ii) they fail to adequately avoid the Cambridge-change objection.

Before proceeding, however, one significant point needs to be noted. The naturalist should not expect an alternate account of function to explicitly refute my central claim that the BST essentially involves a criterion that allows disease status to turn solely on a change with the level of functioning of *other* people's relevant functions and thus is open to a Cambridge-change objection. This is because, as I hope was made clear from the "Waligator" thought experiment, it is difficult to see how the naturalist can refuse to concede that the BST's conception of normal functioning must be defined in terms of the statistical normality of the species-type. In other words, the BST's conception of normal functioning (i.e., what is offered as the solution to the qualitative and quantitative issues about functions) is established by observing and measuring how actual species-*tokens* do

in fact “function”. And so it seems the BST must insist that what essentially matters for health (and disease) is that an individual’s “normal functioning” conform to (diverge from) the statistical norms of the appropriate reference class.

An immediate problem with this is that many notable statistical norms of functioning *will* change throughout any given individual lifespan as he or she ages and different reference classes become relevant, regardless of the BST’s account of function. That reference classes have different statistical norms (recall from Chapter 3, Section 3.1) is necessary to reflect the fact that many of our functional abilities universally increase as we age (e.g., a typical newborn is unable to walk, talk and procreate) but also universally decline as we age. The very fact that the BST must draw upon age-related reference classes with crucially different statistical norms, however, is itself problematic because it clearly implies a Cambridge-change criterion which Boorse does not explicitly acknowledge.

This is troubling because it commits the BST to the view that an individual could go from being diseased to healthy, or vice versa, without any physiological change in that individual. As would be the case, for instance, when an increase in an individual’s age makes a new age-related reference class—with different statistical norms—become relevant such that the individual’s level of functioning *now* sufficiently conforms to (or diverges from) the crucially different statistical norms of the individual’s new reference class.⁶⁸

⁶⁸ A word should be said here that perhaps it might be objected that I am merely hitting upon a general problem with lots of distinctions. For example, bald versus not bald. It surely seems ridiculous to say that one hair marks the difference between being bald or not being bald; nevertheless, we still think there is a

This is a significant point because—aside from discovering the “blueprints” for human functioning—it is difficult to see how the naturalist either persuasively brings to bear an alternative to the BST’s statistical conception of normal functional ability (read: health and medical normality) or circumvents properly counting an individual to go from diseased to healthy, or vice versa, without any physiological change in that individual solely because of changing *relational* properties (i.e., dynamic statistical norms). This position, I submit, forces the naturalist into conceding that the framework of the BST is unavoidably open to Cambridge-change objections. If so, the defender of the BST must stake oneself to the claim that a different account of function sufficiently tempers the Cambridge-change objection. In other words, the central issue becomes whether or not the BST’s essential vulnerability to a Cambridge-change objection renders the BST to be an unsuitable candidate for the rôle that Boorse has cast it to play, i.e., the rôle of underpinning medicine with a theoretical, value-free science of health and disease. I propose to now examine this issue from the perspective of the above-mentioned three general groupings of accounts of function.

distinction between being bald and not being bald. Vagueness at the boundaries happens with many scientific distinctions. On this I am in complete agreement. However, what is to be stressed is that unlike the bald/not bald distinction where vagueness arises because we do not know precisely where to draw the line (i.e., how many hairs one must have not to be bald) the BST has clear cut criteria for distinguishing the line between health and disease (i.e., with the statistical normality of the relevant biological functions). Moreover, however the line between bald/not bald is drawn do we not expect baldness to ultimately turn on amount of hairs on one’s head? My issue here is precisely that by the BST’s lights, continuing with the hair analogy, I could properly be considered to be bald—when I was not before—with no change in the numbers of hairs on my head. The problem is not that there is anything wrong with this *per se* (evaluative norms could be at work; or perhaps ‘bald’ is merely being used as a gradable context dependant adjective); rather it is that the BST’s principled framework leads to such counterintuitive consequences; namely, that I could go from being diseased to healthy and back to diseased without any corresponding physiological change of mine to speak of. And, as such, the BST implies profoundly troubling consequences for an account of health that is said to provide a scientific grounding for medicine—a matter to which we shall return.

3.1 *Backward-looking accounts of function*

Among the class of backward-looking accounts of function, there is a distinction the literature draws between two subcategories, depending on how far back into the past an account looks. Following some notable authors,⁶⁹ I will distinguish between accounts of function that look back to only the recent past from those that look back to the distant/ancient past. That is, I will use historical limitations to mark a rough yet important boundary for the class of backward-looking accounts of function.

3.1.1. *The distant/ancient-past looking accounts*

The core idea of distant/ancient-past looking accounts is that the “function” of a trait is that the “function” of a trait is defined in terms of the etiology or causal history that explains its current presence by reference to the distant/ancient past.⁷⁰ The literature typically names Larry Wright’s “etiological analysis” as the pioneer of this approach.⁷¹ Boorse notes that, in response to criticisms of Wright’s account,⁷² later writers have attempted to add to their accounts key restrictions “requiring both a specific type of etiology, selection, and specific selection mechanisms for different domains—for example, designer’s intention for artifacts, Darwinian selection of a trait’s genotype for organisms”.⁷³ As a result, importantly different distant/ancient-past looking accounts have emerged, of which two of the more influential are advanced by Millikan and Neander.⁷⁴

⁶⁹ For some specific authors, see footnote #14, above.

⁷⁰ Accounts emerging from this approach are also commonly referred to as ‘etiological’, ‘historical’, ‘evolutionary’, and ‘selected effects’ theories.

⁷¹ Larry Wright (1973), *op cit.*

⁷² See, for instance, Boorse (1976b).

⁷³ Boorse (2002), p. 65.

⁷⁴ Millikan (1984), *op cit.*; and Neander (1991), *op cit.*

To simplify the exposition, let us turn to Neander's "selected effects analysis" of function. Very briefly, according to Neander, the "proper" function of a trait is what the trait was "selected for".⁷⁵ For example, it is the proper function of the heart to pump blood because pumping blood is what hearts did in the past and this is what caused them to be favored by natural selection.⁷⁶ For our purposes it is enough to note that on this view there is no historical restriction that limits just how far back in the past natural selection must have occurred.

Without such a limitation, I shall now argue that the distant/ancient past accounts have the naturalist defender of the BST stepping backwards squarely into both horns of the dilemma I described earlier—that either for one reason or another, the naturalist defender of the BST provides an inadequate biomedical account of normal functioning or he leaves the BST open to what I have called the Cambridge-change objection—either of which alternative fatally undermines the BST's goal of providing medicine with a value-free account of health and disease. I shall begin with the first horn.

First horn: Provides an inadequate biomedical account of normal functioning

Importance must be attached to the BST's interest in finding a conception of species design on which species design is stable, uniform and accurate. Recall that the conception of species design must reflect the way in which our 21st century biological mechanisms are

⁷⁵ Neander (1991), p. 174-75. She offers the following formal definition: "It is the/a proper function of an item (X) of an organism (O) to do that which items of X's type did to contribute to the inclusive fitness of O's ancestors, and which caused the genotype, of which X is the phenotypic expression, to be selected by natural selection."

⁷⁶ Ibid., p. 168.

construed as *presently* contributing to our individual survival and reproduction. As I said earlier, it is striking that modern medical practice does not appear willing to count one to be diseased simply because of one's vestigial appendix. But here is where an important point of departure emerges between modern medicine and any version of the BST that embraces a strongly historical account of function. Let me illustrate the problem with a return to Neander and the conclusion she draws:

...for a trait to have a proper function is not for it presently to have any actual causal role, statistically typical contribution to fitness, or disposition. Instead, a trait has a proper function if there is something that it is supposed to do. According to my etiological theory, a trait is supposed to do whatever it was selected for by natural selection...It is the function of kidneys (both normal and in abnormal) to filter wastes from blood because that is what kidneys did in ancestral organisms that caused them to be favored by natural selection (and this fact remain true even if renal failure becomes universal).⁷⁷

Notice on this view that vestigial organs have a function, to wit what it was that appendixes did in our ancestors "that caused them to be favored by natural selection". But clearly to ascribe a current function to the vestigial appendix is an obvious and significant departure from modern medicine's conception of normal functioning.

An immediate worry is that an appeal to a strongly historical account of function therefore has the BST operating with an unsatisfactory conception of species design. And, what is more, if, as this view claims, vestigial organs in general and the appendix in particular have current functions, then the BST winds up committed to forcing a notable departure from modern medicine. This is because by the BST's lights our modern appendix will presumably be incapable of performing a species-type function (i.e., what is

⁷⁷ Ibid., p. 183.

offered as the solution to the qualitative and quantitative issues about functions). This is not acceptable because the BST, according to its current framework, must therefore insist that we are *all* diseased simply because of our vestigial appendix, which is nonsense.

Employing a distant/ancient-past looking account of function will further derail the BST's conception of normal functioning from standard medical thought and practice. As Gould and Vrba correctly note, "current utility carries no automatic implication about historical origin".⁷⁸ They propose the term "exaptation" to acknowledge effects that contribute to fitness but were not favored by natural selection for their current contribution. Among the examples they give of "unselected" traits being co-opted from their present function to perform some other function include feathers selected for thermal regulation but co-opted for flight; the black heron's wings selected for flight but co-opted for shadowing its prey; bones selected to store phosphates but co-opted for structurally supporting vertebrates; and lysozyme selected for killing bacteria but co-opted for mammalian lactation.⁷⁹

What is to be stressed is that insofar as exaptations are not favored by natural selection proponents of a distant/ancient account of function that demands as much are unwilling to ascribe a function to these new effects that may nevertheless contribute to fitness.

All of this is especially troubling because the BST winds up not being able to take to heart and invoke the persuasive view (that most biologists recognize) that notable useful

⁷⁸ Stephen Jay Gould & Elisabeth S. Vrba (1982), "Exaptation — A missing term in the science of form", *Paleobiology* 8(1): 4-15, at p. 13.

⁷⁹ *Ibid.*, pp. 7-10.

features of biological mechanisms are not, and need not be, the result of being “selected” for their current role; but instead may exist because of environmental events;⁸⁰ pleiotropy; random genetic drift, exaptation, being co-opted spandrels,⁸¹ or simply because there was no suitable genetic variation for selection to have occurred at all.⁸² This is not to deny that many, perhaps even most, useful “functions” are adaptations⁸³ but to the degree that such “unselected” useful features exist surely undermines the BST’s ability to employ a conception of species design that accurately reflects how human beings in fact uniformly function at this point in our species’ history.

Most importantly, it intractably undermines the BST’s ability to track modern medicine’s concern for biological mechanisms that currently contribute to our fitness, regardless of their historical origin. To insist, explicitly or by implication, that only the contributions to our fitness “selected” by natural selection are relevant functions when it comes to health and disease is not merely misguided, it is patently absurd. This, because

⁸⁰ For example, Rodríguez-Trelles *et al.*, propose that novel environments can induce “hidden” gene products to develop new phenotypic expressions. See, Francisco Rodríguez-Trelles, Rosa Tario, & Francisco J. Ayala (2005), “Is ectopic expression caused by deregulatory mutations or due to gene-regulation leaks with evolutionary potential?”, *BioEssays* 27: 592–601.

⁸¹ Briefly, “spandrels” is a term borrowed from architecture (which refers to the triangular spaces that exist as necessary architectural by-products of mounting a dome on rounded arches) to designate the class of features arising as necessary but incidental by-products and not as adaptations for direct utility in themselves. See, S. J. Gould & R. C. Lewontin (1979), “The spandrels of San Marco and the Panglossian paradigm: A critique of the adaptationist programme”, *Proceedings of the Royal Society of London. Series B, Biological Sciences* 205(1161): 581-598; and Stephen Jay Gould (1997), “The exaptive excellence of spandrels as a term and prototype”, *Proceedings of the National Academy of Sciences* 94: 10750-10755.

⁸² Peter H. Schwartz convincingly argues this point as part of basic problem he claims confronts recent-history accounts of function: the fact that there is the possibility for a trait with an effect that is crucial to survival and reproduction to be maintained in the population for non-selective reasons. He refers to this possibility as “Non-Selective Maintenance”(NSM). See Peter H. Schwartz (1999), “Proper function and recent selection”, *Philosophy of Science* 66: S210-S222, especially at S214-S217.

⁸³ Briefly, ‘adaptation’ is a term used to describe the evolutionary process whereby a feature of the species that promotes fitness was built by selection because of its current role. See, Gould & Vrba (1982), *op cit.*, p. 6.

standard medical practice is surely correct to count a typical adult unable to walk due to osteoporosis to be diseased even if bones were favoured by natural selection to store phosphates, and thus we want a satisfactory account of health to insist as much.

To sum up: the above presents two reasons to think that a strongly (and I have argued overly) historical account of function commits the BST to underpinning its health and disease claims with a conception of normal functioning that does not accurately reflect how human beings currently, in fact, uniformly function. Firstly, it assigns current functions to vestigial organs, and thus commits the BST to saying that we are all diseased.

Secondly, it does not assign functions to “unselected” exaptations and novel effects that may make important fitness contributions, and thus is unable to adequately echo the standard biomedical account of normal functioning—an account concerned with present species-typical functioning; not with the “function” a physiological mechanism may have once performed in our distant ancestors but no longer performs. So long as this is the case, distant/ancient-past looking accounts of function renders the BST an inadequate candidate to fulfill Boorse’s aim to underpin medicine with a theoretical, value-free science of health and disease.

Second horn: Fails to adequately avoid the Cambridge-change objection

At this point it might be thought that natural selection, as a basic explanatory principle in biology, can be invoked to “explain away” the force of the above problems. At present, I am not convinced that a persuasive rejoinder cannot be brought to bear that mitigates the

problem I have suggested exaptations raise against the BST.⁸⁴ However, I will not pursue this tack because I think there are other more serious problems that will nevertheless remain, namely, the problem of ascribing current functions to vestiges still stands and the problem that the naturalist championing the BST cannot pay the epistemological price that the distant/ancient-past looking account of function demands. Together these two problems place the BST, grounded on a distant/ancient-past looking account of function, into an untenable position, as such the BST falls squarely upon both horns of the above-mentioned dilemma, and thus remains unable to adequately avoid the Cambridge-change objection.

We saw above the way in which a strongly (and I argued overly) historical account of function that ascribes current functions to vestiges provides the BST with an inadequate biomedical account of normal function. Not only does this impale the BST on one horn of a dilemma but it also provides further reason to insist that the BST is theoretically dubious, and thus impaling it on the second horn of the dilemma.

And this brings us to the second of the two above-mentioned problems: The

⁸⁴ For instance, a *weak etiological theory* has been advanced in the literature, which denies that etiological accounts must understand functions in terms of selection. This theory defines functions as effects of traits that contributed to the fitness of ancestors and that this, in turn, contributed to the trait's continued existence. See David J. Buller (1998), "Etiological theories of function: A geographical survey", *Biology and Philosophy* 13: 505-527. And Buss *et al* quite convincingly conclude that: "...adding exaptation to the conceptual toolbox of evolutionary psychology does not diminish the importance of natural selection as the primary process responsible for creating complex organic design — a point apparently endorsed by all sides involved in these conceptual debates. Selection is responsible for producing the original adaptations that are then available for co-optation. It is responsible for producing the adaptations, of which spandrels are incidental by-products. It is responsible for producing structural changes in exaptations in order to fulfill their new functions. And it is responsible for maintaining exaptations in the population over evolutionary time, even in the rare cases where no structural changes occurred." See, David M. Buss, Martie G. Haselton, Todd K. Shackelford, April L. Bleske & Jerome C. Wakefield (1998), "Adaptations, exaptations, and spandrels", *American Psychologist* 53(5): 533-548, at p. 543.

naturalist defender of the BST cannot pay the requisite epistemological price,⁸⁵ which I will now argue drives the second horn directly and further into the heart of the BST.

Quite simply, if the BST is going to ascribe current functions to vestiges and, hence, view them to be relevant functions when it comes to health and disease, then the naturalist (as argued above) must have an objective, value-free claim to know (at least in principle) the “literally true” levels of functioning of the individuals, past and present, it counts to establish the statistical norms of the BST’s reference classes. But surely the level of *actual* performance (i.e., quantitative functioning) of our distant ancestor’s appendices cannot be read off the biological facts of nature.

Indeed, as argued above, a similar point holds for the levels of performance of all the functions counted to be relevant for health and disease claims. So, when vestiges are ascribed current functions, notice we have an acute instance of a point I argued earlier, namely, that we have a knockdown reason to insist that there are limits to how far back in the species’ history the naturalist can legitimately draw its reference-class members from. This is because, recall, the naturalist requires that the quantitative issue of which level of functioning is requisite for a goal of a function to be successfully realized be an objective matter, read off the biological facts of nature. For this reason the BST is ill-equipped to formally embrace what the distant/ancient-past looking account of function demands, namely, including distant past members of the species into its reference classes for which

⁸⁵ Here it may be worth noting another sense in which the naturalist defender of the BST aiming to employ an overly historical *selected effect* account of function cannot pay a requisite epistemological price. For Amundson and Lauder amply demonstrate that in actuality there are many instances (e.g., due to pleiotropic effects) where it will be virtually impossible to have the direct evidence needed to single out the particular trait, or particular combination of traits, that was selected for. See Amundson & Lauder (1994), *op cit*.

the requisite levels of functioning when they were alive is surely unknowable to modern empirical science. And, as such, the naturalist cannot pay the epistemological price the BST demands of a distant/ancient account of function.

And, what is more, because the naturalist must limit how far back in time the BST may draw its reference class members from, the BST underpinned by a distant/ancient backward-looking account of function cannot but appeal to statistical norms that *will* change. As we saw earlier, the fewer the number of past individual's unchanging levels of functioning the BST's reference classes may properly include, the greater the extent to which changes in the levels of performance of a function of *present* individuals will *ceteris paribus* alter the relevant statistical norms of the reference classes.

Accordingly, it is not insignificant that between 1959-1999, the world population has doubled, increasing from 3 billion to 6 billion.⁸⁶ But further, the U.S. Census Bureau projects the world population to increase to 9 billion by 2044, another increase of 50 percent that is expected to require 45 years. Clearly a substantially increasing population with significantly increasing (or decreasing) relevant (i.e., when it comes to health and disease) states cannot but have a substantial impact on the relevant statistical norms of the BST's various reference classes, given the naturalist's aim to underpin medicine with the BST. It therefore follows that if the U.S. Census Bureau's projections are to be taken seriously (and why wouldn't they be?), then the naturalist winds up being committed to saying that an overly historical account of function simply cannot preclude

⁸⁶ U.S. Census Bureau. *International Data Base, December 2010 Update*. <<http://www.census.gov/ipc/www/idb/worldpopgraph.php>> Accessed July 4, 2011.

the BST from appealing to changing statistical norms. For ease of reference, I will refer to this as the “increasing population problem”.

It is important to recognize that the “increasing population problem” strikes with considerable force against any attempt to avoid the Cambridge-change objection by underpinning the BST on an overly historical account of function, not just against those accounts that are properly termed “selected-effect” accounts of function. As it stands, it seems that the naturalist must acknowledge that as the world’s population grows, changes in the levels of performance of a function of present individuals will come to have an increasingly greater impact upon the relevant statistical norms. This acknowledgement becomes especially problematic because there is very good reason to think that tomorrow’s present individuals will perform at levels of quantitative functioning that *will* notably alter the BST’s dynamic statistical norms.

To motivate this view it will help to recall an important point noted earlier (in Chapter 3, Section 3.1), namely, that there has also been a significant increase in the average height, weight and life expectancy globally and amongst particular populations. Furthermore still, it is striking that an ever-increasing population lives (and will live) in a world that the World Health Organization (WHO) argues has changed significantly in the past 30 years becoming a “globalized, urbanized, and ageing world” and in doing so has left people more exposed and more vulnerable to health threats.⁸⁷ Now no reasonable

⁸⁷ For instance, according to the World Health Organization: “Ageing and the effects of ill-managed urbanization and globalization accelerate worldwide transmission of communicable diseases, and increase the burden of chronic and noncommunicable disorders”. See, World Health Organization (2008a), *The world health report 2008: Primary care now more than ever*. Geneva: WHO Press, esp., at pp. xiii & 6.

person would deny that we live in a world that is substantially different from the world of only a generation or two ago. If, as the WHO claims, these changes are impacting the health of both individuals and populations, then we have strong reason to insist that the projected population increases will notably alter the BST's statistical norms.

And, what is more, the “increasing population problem” strikes with increasing force to the extent that claims, such as the WHO advances, that our changing world strongly impacts population and individual health is to be accepted. Here it is worth noting that recently, Sir Michael Marmot and others have argued persuasively that economic and social inequities are related to causation of ill-health.⁸⁸ A significant conclusion Marmot draws is that “[h]ealth follows the social gradient”.⁸⁹ That is to say, when it comes to health disparities it is not, strictly speaking, the amount of material resources one has that matters as much as it is how much one has relative to others.

In fact, the Senate of Canada not only explicitly acknowledges that income disparities between the wealthiest and poorest members of the society affects health but recently states that “[f]ully 50% of the health of the population can be explained by socio-

⁸⁸ See, for instance, Michael Marmot (2006), “Health in an unequal world: social circumstances, biology and disease”. *Clinical Medicine* 2006; 6(6): 559-572; World Health Organization (2008b), *Closing the gap in a generation: health equity through action on the social determinants of health. Final report of the commission on social determinants of health*. Geneva, World Health Organization; and Norman Daniels, Bruce Kennedy, & Ichiro Kawachi (2000), *Is inequality bad for our health?* Boston, Mass: Beacon Press.

⁸⁹ Michael Marmot (2006), “*op cit.*”, at p. 561. Daniels, Kennedy and Kawachi echo this point when they state “[w]e now know...that middle-income groups in relatively unequal societies have worse health than comparable, or even poorer, groups in more equal societies”. See Daniels *et al.* (2000), *op cit.*, at p. 3. Additionally, both the Canadian Public Health Association and the Public Health Agency of Canada have long insisted that income and social status are strong determinants of health. See, Health Canada (2001), *The population health template: Key elements and actions that define a population health approach*. Ottawa: Author; and The Public Health Agency of Canada website. <<http://www.phac-aspc.gc.ca/ph-sp/docs/common-commune/chap2-eng.php>> Accessed July 4, 2011; and Canadian Public Health Association website. <<http://www.cpha.ca/en/default.aspx>> Accessed July 4, 2011.

economic factors”.⁹⁰ Save a persuasive reason to think that an increasing population will not further perpetuate the growing socio-economic disparities⁹¹ this striking statistic serves to further motivate the view that the projected population increase *will* change the BST’s statistical norms.

If, as I have suggested, the projected population increase begets meaningful change in the BST’s statistical norms, then the “increasingly population problem” stands forcefully against any attempt to motivate the BST’s candidacy to underpin medicine with a theoretical, value- and norm-independent account of health and disease with a distant/ancient-past account of function.

What the above reveals is that the naturalist must not only abandon the notion that by including a greater time-slice of the species’ history one can persuasively stabilize the BST’s statistical norms to adequately temper Cambridge-change objections; but also must concede that an overly historical account of function fails to prevent *relational* properties (i.e., dynamic statistical norms) from making a healthy individual diseased, or vice versa, without any physiological change in that individual. For these reasons, the BST, when coupled with a distant/ancient-past account of function, remains unable to avoid the Cambridge-change objection, and thus remains inadequate to serve its own purposes.

3.1.2. *The recent-past looking accounts*

The core idea of recent-past looking accounts is that the “function” of a trait is defined by

⁹⁰ Senate Canada (2009), *A healthy, productive Canada: A determinant of health approach. The standing senate committee on social affairs, science and technology: Final report of the subcommittee on population health*. Ottawa: Author, at p. 8.

⁹¹ See Section 3.2 in Chapter 7 below for some disparaging statistics showing that Canada’s socio-economic disparities are in fact growing.

reference to only a slice of its history, not all of it, and that the slice invoked is the past that is in some sense “recent”. The attraction of such a view seems to be that a trait’s “function” is taken to be defined in terms of the contributions that having the trait makes to the reproductive and survival success of *contemporary* organisms.⁹² Notable adherents of the recent-past backward-looking approach include Woodfield, Nagel, Godfrey-Smith, Griffiths and Schwartz.⁹³ Moreover, on a natural interpretation of the textual evidence I gave earlier it would seem that Boorse’s GGC account of function, which defines functions in terms of contributions to *actual* goals follows a similar line.

From the above description of this approach, one might quite reasonably expect that a satisfactory account of function to ground the BST would be one that emerges from the recent-past looking class of accounts. But this is not the case.

Suppose the naturalist seeks to ground the BST upon some recent-past account of function (an account, remember, that is going to be different from Boorse’s own account). Then if this “alternative” account was to succeed in saving the BST from Cambridge-change objections, it will presumably be because and in so far as the new alternative account limits the relevant time slice along the lines described in the first paragraph of this section. The hope will then be that by so limiting the relevant time-slice the account will

⁹² For a brief yet informative overview about some notable theorist’s motivations behind setting their requirement for a historical limitation to the recent-past see, Schwartz (1999), *op cit.*, especially at S212-S213.

⁹³ Perlman’s taxonomy places the “goal-contribution” accounts of Woodfield (1976), Nagel (1961), Nissen (1997), and Boorse (1976, 1977, 2002); and the ‘modern-history’ account of Godfrey-Smith (1994) within the “recent-past” subcategory of the backward-looking approach. See Perlman (2004), *op cit.*, pp. 6, 12 & 18-20. To this list, it seems to me appropriate to add Griffiths’s account of function and Schwartz’s “continuing usefulness” account. See, Griffiths (1993), *op cit.*; and Schwartz (1999), *op cit.*, especially at p. S219.

also limit the extent to which the statistical norms of the BST's reference classes will undergo variation through time, thus preventing the Cambridge-change objection getting off the ground.

The problem is that it is difficult to see how any naturalist recent-past looking account of function could go far enough in so limiting variation as to *adequately* stabilize those statistical norms which the BST needs if it is to be able to spell out a sufficiently precise account of *successful* fulfilment of function, something needed by the BST for its account of health and disease as Boorse's (D)—and I hope my (D*)—clearly demands. That is because any recent-past looking account of function must limit the amount of the past it includes if the naturalist is both to employ an appropriate conception of species design as well as to pay the epistemological price I have argued the BST demands.

We have seen how such a limitation on the past will, among other things, enable changes in the levels of performance of a function of present individuals to alter the relevant statistical norms of the reference classes more than they otherwise might have, if a greater number of past individuals were allowed to be included in the reference class. Thus, as was the case with strongly (nay overly) historical accounts of function, recent-past looking accounts of function end up committed to saying that a substantially increasing population with significantly increasing (or decreasing) *levels* of functioning will *ceteris paribus* alter the relevant statistical norms of the relevant reference classes.

This becomes a significant concession since it renders the BST, if grounded upon a recent-past account of function, vulnerable to the “increasing population problem”. Given that the world's population is expected to increase substantially (to 9 billion people) in the next 33 years, for reasons mentioned above, it is difficult to see how a recent-past looking

naturalist account of function enables the BST to simultaneously uphold what is surely one of Boorse's most important claims—that function statements are literally true—and adequately preclude it from appealing to changing statistical norms. And this carries grave consequences. The failure to achieve the latter leaves the BST wide-open to Cambridge-change objections; the failure to uphold the former fatally undermines the claim that the distinction between healthy and diseased states is an objective matter, to be read off the biological facts of nature. And as such, because the BST will not successfully avoid the horns of the above-mentioned dilemma, we have sufficient grounds to reject the notion that a recent-past looking naturalist account of function enables the BST to fulfill Boorse's aim to provide medicine with a theoretical, value- and norm-independent science of health and disease.

3.2 *Forward-looking accounts of function*

The core idea of the forward-looking accounts is that the “function” of a trait is to be defined, not by its evolutionary history, but in terms of its potential effects on a future outcome of some sort, which in most versions is contribution to the future reproductive success of members of the species.

Arguably the most familiar forward-looking account is Bigelow and Pargetter's “Propensity Theory” of function. The general idea of this theory is that “[s]omething has a (biological) function just when it confers a survival-enhancing propensity [relativized to an environment] on a creature that possesses it”.⁹⁴ “Functions”, according to Bigelow and

⁹⁴ Bigelow & Pargetter (1987), *op cit.*, p. 192.

Pargetter, “are truly dispositional...[and] are specified subjunctively: they *would* give a survival-enhancing propensity to a creature in an appropriate manner, in the creature's natural habitat”.⁹⁵ Importantly, Bigelow and Pargetter go on to extend the term “habitat” to apply to the surroundings of an organ (or cell) within an organism (or organ). The upshot, they claim, is the capacity to attribute functions to components of an organism, no matter how small, so long as they have the propensity to enhance survival in the creature’s natural habitat, and this, they notably suggest, allows for defining “healthy individuals” in terms of their “Propensity Theory”.⁹⁶

It is worth explicitly noting that forward-looking accounts of function that link biological function to the disposition or the propensity of some trait or biological structure to enhance survival possess two features I have in fact already argued are requisite if an account of function is to plausibly track modern medicine’s conception of normal functioning. Firstly, it easily allows for a biological structure to have a function that it is in fact never called on to perform. And secondly, it has no problem immediately ascribing a function to a novel trait of biological structure provided, of course, it is of the right sort. Hence forward-looking accounts of function readily sidestep significant problems—problems that we have seen other candidate accounts of function seem ill-equipped to avoid.

Notably, when it comes to ascribing functions to traits or biological structures,

⁹⁵ Ibid., p. 193.

⁹⁶ Ibid., pp. 192-193. William Bechtel also attempts to define “health” in terms of a propensity account of function. According to Bechtel: “That [the propensity interpretation of fitness] is all that is required for our purposes...A healthy state of the system is one in which it makes the best use of its physiological endowments in responding to selection pressures”. See, Bechtel (1985), *op cit.*, at pp. 151-154.

forward-looking accounts that construe functions as survival-enhancing—as, for instance, Bigelow and Pargetter’s “Propensity Theory”—yield similar results as Boorse’s GGC account of function. This is because their claim that “function” is dispositional and to be specified by the subjunctive (i.e., if the creature should be in its natural habitat, then it will perform its survival-enhancing functions) yields a strikingly similar understanding of normal functional ability to that of Boorse’s (Boorse, recall, defines *normal functional ability* as the readiness of each internal part to perform all its normal functions on typical occasions with at least statistically typical efficiency). In fact, they explicitly identify their theory as a “cousin” to Boorse’s goal-directed theory of function.⁹⁷ Such forward-looking accounts of function, however, *should* be thought to share a much less-welcome similarity with Boorse’s GGC account: their inability to successfully avoid the horns of the dilemma I mentioned earlier—either for one reason or another, they provide an inadequate biomedical account of normal functioning or they leave the BST open to the Cambridge-change objection. Or so I shall now argue.

Suppose the naturalist were to embed a forward-looking propensity account of function in the BST. Now whether or not a biological trait or structure confers a survival-enhancing propensity surely always depends on the environment in which it appears. So Bigelow and Pargetter’s claim that a survival-enhancing propensity must be relativized to the creature’s natural habitat appears correct.

For the defender of the BST to insist as much, however, creates some serious problems. Herein lies the rub: the BST must account for the fact that the same biological

⁹⁷ Bigelow & Pargetter (1987), *op cit.*, p. 182.

structure may confer a survival-enhancing propensity in one sort of environment but may not in a different sort of environment. To illustrate the problem consider the infamous Sickle cell trait. Now the Sickle cell trait surely confers a survival-enhancing propensity on an individual whose “natural habitat” is where the infectious disease malaria is a pandemic, mosquitoes are in abundance, and there is a complete lack of low-oxygen environments (call this environment, A^*). Given that the *absence* of the Sickle cell trait would be (on the versions of the BST stated in (D) and (D*)) pathological in A^* , it seems that here the defender of the BST is thus committed to ascribing a function to the Sickle cell trait and, moreover, demanding that it (i.e., the trait’s relevant biological structure) performs at a minimal level of functioning for an individual to be counted healthy in A^* .⁹⁸

Consider now a different sort of environment that is at high-altitude, an environment where neither malaria nor mosquitoes are endemic (call this B^*). The point to press is that in B^* the *presence* of the Sickle cell trait would not only *not* confer a survival-enhancing propensity—it would be pathological. Accordingly, we should expect a forward-looking propensity account of function to preclude the BST from ascribing a function to the Sickle cell trait in B^* . But if it is correct to ascribe a function to the Sickle cell trait in A^* but not

⁹⁸ Here we have somewhat been hitting upon a familiar objection raised against the “Propensity Theory, namely, that it lacks the wherewithal to distinguish between genuine functions and beneficial yet accidental effects, and thus is “forced to attribute a function to more or less everything”. See, for instance, Peter McLaughlin (2001), *op cit.*, at p. 128. Quoted in, Ananth (2008), *op cit.*, at p. 188. Ananth not only agrees with McLaughlin’s conclusion but he takes this to warrant the rejection of the history-free propensity account of function. Admittedly, if the “Propensity Theory” is truly going to eschew appealing to historical facts, then it seems committed to (i) ascribing a function to any effect of a trait just as soon as it confers a survival-enhancing propensity; and (ii) not ascribing a function to a trait’s effect that, once did, but no longer confers a survival-enhancing propensity. For our purposes, what is to be stressed is that these are commitments that the BST will take on when embedded with a forward-looking propensity account of function.

in B*, then the defender of the BST winds up committed to saying that the species design must be relativized to the environment.⁹⁹ This is because the BST's conception of species design *is* tantamount to the solution to the qualitative issues of function.

This, however, leads to a further less desirable commitment: for the defender of the BST must surely acknowledge the fact that humans occupy vastly different “natural habitats” and thus, it seems, the BST is committed to insisting upon multiple—and crucially different!—human species designs in which to account for the vastly different environments. Such an insistence is an obvious and significant departure from modern medicine's biomedical conception of normal functioning, which counts states to be “diseases” (e.g., malaria resistant genetic disease α -Thalassemia, Sickle cell anemia, Type II diabetes mellitus, and cowpox) despite strong evidence that they confer a survival advantage in some environments.

Moreover, it is a claim we should resist accepting insofar as we surely want to resist prescribing that modern medicine can “cure” a diseased individual, not by removing any pathological condition but merely by relocation to a different environment, in which the relevant biological function would no longer give a survival-enhancing propensity. But further, here the defender of the BST is surely stuck with advancing an inadequate biomedical account of normal functioning since it is difficult to see how one will avoid

⁹⁹ It is worth noting that Bigelow and Pargetter appear very comfortable with such a commitment: “First, like the corresponding account of fitness, this account of functions must be relativized to an environment. A creature may have a high degree of fitness in a specific climate-but a low degree of fitness in another climate. Likewise, a character may confer propensities which are survival-enhancing in the creature's usual habitat, but which would be lethal elsewhere. When we speak of the function of a character, therefore, we mean that the character generates propensities that are survival-enhancing in the creature's natural habitat.” See Bigelow & Pargetter (1987), *op cit.*, p. 192.

false presumptions caused by calling something or someone healthy (or diseased) which lacks the biological function (or dysfunction) on which such presumptions surely depend.

A related and, I think, even more serious problem is that this way of linking functions to the creature's natural habitat effectively blocks the BST's capacity to operate with anything like an adequate conception of a single, uniform, and stable species design and, hence, has the BST operating with an unsatisfactory conception of species design. This is because, when the relevant species design becomes relativized to an individual's natural environment, the defender of the BST is thus forced to concede that it could be the case where an individual in A* is properly counted to be healthy and another individual in B* is properly counted to be diseased, *despite there being no physiological difference between the two individuals*. In turn, this commits the defender of the BST to the troubling view that an individual could go from being diseased to healthy, or vice versa, without any physiological change in that individual. That is to say, the BST is thus left wide-open to Cambridge-change objections.

I conclude that naturalist attempts to avoid the horns of the above dilemma by grounding the BST on a forward-looking account of function will prove unsuccessful. If it is true that function must be relativized to an environment, then I hope to have convincingly shown the way in which the defender of the BST winds up committed to saying that the species design must be relativized to the environment; and thus ends up impaled on both horns. But further, even if I am wrong in thinking that a forward-looking account of function effectively blocks the BST's capacity to operate with an adequate conception of what it is to have a single, uniform and stable species design, the BST still

remains vulnerable to the “increasing population problem”.

In sum, it is not merely the case that there is no reason to think that a forward-looking account of function equips the BST to avoid the Cambridge-change objection. But it is also difficult to envision how a history-free, forward-looking account of function, which presumably must appeal neither to historical facts of functioning nor to past environments,¹⁰⁰ *could* have the resources to insulate the BST from Cambridge-change objections.

3.3 *Present-looking accounts of function*

The core idea of present-looking accounts is that the “function” of a trait is to be defined in entirely ahistorical terms. Arguably the most famous ahistorical account of function is Cummins’ “causal-role” analysis of function, in which he advances what has come to be called “Cummins functions”. Very briefly, Cummins’ view is that functions are to be understood in terms of a “capacity” such that an item’s function is just its causal contribution to the performance of some capacity of a larger system. He writes:

To ascribe a function to something is to ascribe a capacity to it which is singled out by its role in an analysis of some capacity of a containing system [e.g., human organism]. When a capacity of a containing system is appropriately explained by analyzing it into a number of other capacities [e.g., the circulatory system, the digestive system, the nervous system, etc.,] whose programmed exercise yields a manifestation of the analyzed capacity, the

¹⁰⁰ Godfrey-Smith objects that Bigelow and Pargetter understand “natural habitat” historically. See Godfrey-Smith (1995), *op cit.*, pp. 351-353. Against this, Ananth argues that Bigelow and Pargetter are not committed to an evolutionary history and, as such, can avoid Godfrey-Smith’s objection if they only understand “natural habitat” in terms no longer than the recent past habitat. See Ananth (2008), pp. 185-186. I will set this matter aside because, insofar as the defender of the BST is concerned, even if Ananth is correct, then it would just introduce a further problem to overcome, a problem we saw raised against recent-past naturalist accounts of function, *i.e.*, satisfying the epistemological price the BST demands.

analyzing capacities emerge as functions.¹⁰¹

For our purposes we need only further note that, according to Cummins' analysis, for an item to have a function implies that it must *presently* have the capacity/disposition¹⁰² to perform the particular task which contributes to the particular system of which it is a part.

With the above in mind, by now the basic idea of my argument is quickly conveyed. Presumably a principled distinction between present-looking and backward-looking accounts of function demands that an entirely ahistorical present-looking account of function must appeal neither to historical facts of functioning nor to past environments. But then problems arise. I have argued that an adequate conception of the species design must include specification of a time-slice of the species' history that is broad enough to enable adequate specification of a species-*type* design that is both uniform and stable. If it is correct to think that a present-looking account of function is unable to appeal to historical facts, then the naturalist cannot realistically hope for a present-looking account of function to have the resources to needed to stabilize the statistical norms of the BST's reference classes. But without such resources, then the naturalist simply cannot preclude the possibility that a version of the BST that embraces a present-looking account of functions will be confronted with the problem of changing statistical norms.

The point is that, because the statistical norms of the BST's reference classes *will*

¹⁰¹ Cummins (1975), *op cit.*, at p. 756. Brackets mine. See also pp. 760-761.

¹⁰² I understand Cummins to advance a dispositional account of function given that he writes: "Thus, function-ascribing statements imply disposition statements; to attribute a function to something is, in part, to attribute a disposition to it. If the function of *X* in *S* [is] to *Z*, then *X* has a disposition to *Z* in *S*". See Cummins (1975), *op cit.*, at p. 758. Variables have been changed for consistency.

undergo changes, this opens these versions of the BST up to the Cambridge-change objection since the naturalist must assert that present changes in both individuals and the environment directly and significantly impacts what the BST counts to be functions (i.e., what is identified as solutions to the qualitative issues about functions) and normal functioning (i.e., what is identified as solutions to the quantitative issues about functions).

To illustrate, let's return to Boorse's case (Chapter 3, Section 3.3) of the whole earth going pitch black for two days, and imagine a world cast in complete and total darkness. Given the human eye's inability to see in total darkness, this much seems clear: a sudden environmental change has, for all intents and purposes, rendered our eyes useless. That is to say, the human eye universally no longer makes a causal contribution to the particular system of which it is a part. If this is so, one would expect an ahistorical present-looking account of function to view the human eye as no longer serving *any* function. After all, on what grounds could the naturalist resist saying that the eyes had lost its function (i.e., what is identified as solutions to the qualitative issues about functions) in the human species given our present universal blindness? It seems extremely implausible to try to square Cummins' claim that "function" is to be specified by a capacity to make a causal contribution with the notion that the eyes had not lost its function in the human species despite our universal blindness, without invoking historical facts.

And so it seems that at this point the BST would have to say that the human eye has ceased to serve any function and hence to lack a specifiable function (hence the theory lacks the conceptual resources to ascribe a function to the eye and address here what I called qualitative issues about functions) and as such, must now count the many previously blind individuals—individuals that it would have counted to be diseased a day prior (when

the mechanisms of the human eye were still a relevant function for health and disease) —to now be healthy! For the defender of the BST to maintain that an individual could be correctly counted to be diseased yesterday and with no diagnostic change in the individual's functional ability be correctly counted to be disease-free (i.e., healthy) today clearly not only leaves the BST wide open to the Cambridge-change objections; it, implicitly or otherwise, advocates what I have called a Cambridge-change criterion.

Further still, I hope it is clear that the “increasing population problem”, for the reasons presented above, effectively stands to undermine the ability of the BST with this ahistorical account of function to block the Cambridge-change objection. As we have seen, to the degree that a present-looking account of function will have the defender of the BST read off the relevant biological facts (i.e., the item's, mechanism's, structure's, etc., “capacity” to make it's causal contribution to the performance of some capacity of a larger system) of *present* individuals to establish the level of actual functioning requisite for the BST's statistical norms, the BST becomes vulnerable to the “increasing population problem”.

Add to this the recognition that the “increasing population problem” implies that the statistical norms of the BST's reference classes *will* undergo changes, and we have yet further grounds to insist that the Cambridge-change objection effectively and forcefully stands against the BST. Thus we have warrant, I conclude, to reject the notion that a present-looking account of function might save the BST's candidacy to underpin medicine with a theoretical, value- and norm-independent account of health and disease.

4. Chapter conclusion

Let me very quickly summarize what I have attempted to do in this long chapter. I have argued against the notion that the most influential naturalist account of health and disease, Boorse's BST, could be saved if it is spelled out with a different account of *function*, different from the one espoused by Boorse. To this end, I have examined those accounts of function that might reasonably be considered alternative candidates for incorporation into the BST from the perspective of three general groups, which I call, respectively, *Backward-looking*, *Present-looking*, and *Forward-looking*.

While I concede that within each of these general groups the defender of the BST has numerous accounts of function from which to choose—accounts with, to be sure, substantial and important differences—I submit all three groups fail to offer the BST a viable naturalist candidate account of function. More specifically, I have argued that none of these three general groups seemingly have the resources to avoid the horns of a dilemma: either (i) they, for one reason or another, provide an inadequate biomedical account of normal functioning; and/or (ii) they fail to adequately avoid the Cambridge-change objection. Either of these alternatives, because of the tremendous amount of weight the current framework of the BST must place on its account of function, fatally undermines the BST's goal of providing medicine with a value- and norm-independent account of health and disease.

Chapter Seven

Conclusions and Prospectus

1. Introduction

In the preceding chapter, we saw that the Cambridge-change objection provides the resources for an argument against the BST's goal of providing medicine with a value- and norm-independent account of health and disease. More specifically, I argued that, with the BST, the naturalist is without an account of function capable of both setting aside the Cambridge-change objection and providing an adequate biomedical account of normal functioning. Thus I conclude that the BST is an unsuitable candidate for the rôle that Boorse has cast it to play, i.e., to underpin medicine with a theoretical, value- and norm-independent science of health and disease. To the degree that the BST represents the best articulated and defended version of an account of health and disease that is ontologically and semantically objective by (O*) and (S*), the Cambridge-change objection poses a threat to the viability of the naturalist view of health and disease.

Though I am convinced the above considerations provide substantial reason to reject BST versions of the naturalist's claim to be advancing a satisfactory conception of health and disease, I am not dismissing the possibility that a more adequate account of function could toe the naturalist line and that correspondingly naturalism could be saved. For such an account to actually be able to save the BST (or any other such naturalist conception of health and disease for that matter) from the above problems I am, however, doubtful. Let me now try to explain why I have such doubts.

We must first keep in mind that a satisfactory account of function to ground a

naturalist conception of health and disease must have the resources to address what I have called the qualitative and quantitative issues of functions. In addressing these issues, however, the naturalist must place constraints on such an account of function to yield a value- and norm-independent scientific analysis of normal functioning.¹ Given the constraints set out in the previous chapters, this creates the following pressure point: it is hard to see how such an account can provide an adequate biomedical account of normal functioning such that the distinction between healthy and diseased states is an objective matter, presumably to be read off the biological facts of nature, while avoiding the Cambridge-change objection. My difficulty is that, in light of the arguments advanced in Chapter 5, I cannot see a plausible way for the naturalist to advance the requisite account of normal functioning without making use of a statistical conception of normality of some sort—a problematic move that opens a naturalist conception of health and disease up to the Cambridge-change objection.

With that said, there is something immensely beguiling about trying to formulate a scientific account of normal functioning and thus, it will be fruitful to make my concerns explicit. Valerie Gray Hardcastle correctly emphasizes that, “Science, in fact, creates many, if not most, biological and social categories out of structural components” and goes on to claim,

...that scientists use these sorts of structural categories to bootstrap their way into functional descriptions. Certainly, they can pick out the forelimbs of a single species or group or population and go on to figure out what the normal distribution of the trait is, how it might be malformed or diseased or truncated,

¹ Valerie Gray Hardcastle nicely emphasizes a very similar view in Hardcastle (2002), “On the normativity of functions”, in A. Ariew, R. Cummins & M. Perlman (Eds.), *Functions: New essays in the philosophy of psychology and biology*. Oxford: Oxford University Press, 144-156, at p. 153.

and so on.²

But how does the naturalist specify what the *normal* distribution of the trait is but by using a statistical conception of normality of some sort? In the next section I will examine one such suggestion if only as an illustration of the problems which I think face any such attempt. What I have in mind is the sort of suggestion that turns on the idea that we might be able to explicate the notion of function and of normal function by reference to structural/morphological and hence non-statistical considerations: I will say from the outset that I am not at all convinced that the requisite sort of relationship between morphology/structure and function exists.

2. On a “reverse engineering” strategy: Some doubts

Suppose the naturalist was to insist that science can successfully identify functions based solely from morphological considerations, i.e., from examination of the physical structures of organs and organisms. Perhaps, then, one might turn to morphology and employ a strategy that Daniel C. Dennett calls *reverse engineering*, in order to secure the requisite account of normal functioning from accounts of current structures.³ Given that I have argued that an adequate account of function should reflect how our species actually functions, this approach appears, initially at least, promising.

² Hardcastle (1999), *op ct.*, at pp. 37-38.

³ According to Dennett “...you just can’t do biology without doing reverse engineering...biology is not just like engineering; it is engineering. It is the study of functional mechanisms, their design, construction, and operation”. Daniel C. Dennett (1995), *Darwin’s dangerous idea*. New York: Simon & Schuster, pp. 213 & 228.

2.1 On the basic assumption(s) required

George V. Lauder in a very useful discussion⁴ makes some points which persuasively casts doubt upon the suggestion that what I have described above as the reverse engineering strategy might be used in developing an account of function. His argument turns on the use of a striking example of the behaviour⁵ of salamanders during aquatic prey capture. Lauder quantifies three parameters in his case study: (i) physiological function of the head muscles (by recording muscle activity electromyographically); (ii) behaviour (by video recordings of prey capture); and (iii) and the structure of the head (by examining the musculoskeletal system of the head). These parameters were examined on four taxa of salamanders: *Abbystoma*, *Cryptobranchus*, *Necturus*, and *Siren*.

He notes some striking similarities and differences. For instance, at the level of physiological function, *Necturus* and *Cryptobranchus* are not significantly different; *Siren* and *Abbystoma* are significantly different from both each other and the *Necturus* and *Cryptobranchus* pair. At the structural level, *Siren* and *Abbystoma* are similar; *Necturus* and *Cryptobranchus* are morphologically different from both each other and the *Siren* and *Abbystoma* pair. While at the behavioural level, *Abbystoma* and *Necturus* are similar, *Cryptobranchus* and *Siren* show a differentiation.

Strikingly, despite having a similar physiological structure, *Siren* and *Abbystoma* possess significantly different feeding behaviours and physiological function in motor

⁴ George V. Lauder (1996), "Argument from design", in M. R. Rose & G. V. Lauder (Eds.), *Adaptation*. San Diego: Academic Press, 55-91.

⁵ Here Lauder understands behaviour as follows: "Behavior results from patterned output from the central nervous system to musculature. This output, in conjunction with physiological properties of the musculature and the mechanics of the arrangement of muscles and bones, determines the observed pattern of movement that we call behavior [sic]". Lauder (1996), *op cit.*, p. 66.

patterns (muscle activity). And despite differing in both morphology and muscle function, *Abbystoma* and *Necturus* produce similar behaviours. From this study, Lauder thus concludes:

It is clear that [of the three levels mentioned above] an analysis of any one level alone is an insufficient description of the design of the feeding system in salamanders, and that prediction of behavior [sic] or physiological function from structure alone in this case study is effectively impossible.⁶

These findings do not mean that the reverse engineering strategy is futile; it does mean, however, that a genuine seed of doubt is sprouted. Certainly the conclusion Lauder draws ought to give the naturalist tempted by a reverse-engineering strategy some pause. For it is not at all clear whether there is the connection between structure and function that permits an entirely objective science-based enterprise to solve the qualitative and quantitative issues of functions from structures alone. The worry that the naturalist may be putting heavy weight on a dubious assumption is legitimate.

Demonstrating the falsity of a position, of course, requires more than pointing out dubious assumptions. Moreover, what is true of salamanders need not be true of humans. In any case, I think there are reasons that force a naturalist concerned with developing an account of health and disease in these foundations to echo Lauder's conclusion regarding humans, reasons which I shall aim to make clear in due course.⁷ To that end, let us now examine the extent to which, if at all, the reverse engineering strategy is correct to at least start from the possibility that the requisite connection between structure and function

⁶ Lauder (1996), at p. 70.

⁷ While I understand Lauder to be challenging the relationship between morphology and function i.e., the solutions to the qualitative issues about function(s), I think his conclusion applies to normal functioning i.e., the solutions to the quantitative issues about function(s). I expand upon this notion below.

exists in a way useful for the development of naturalist accounts of function and normal function. If this basic idea proves misguided, then equally will the naturalist's project of seeking to stake its conception of health and disease to the reverse engineering strategy.

There is another basic assumption, which as it turns out, is of great import.⁸ Dennett emphasizes the assumption that the reverse engineering strategy, and thus the naturalist, must make when he writes,

Still, optimality must be the default assumption; if the reverse engineers can't assume that there is good rationale for the features they observe, they can't even begin their analysis.⁹

The notion of optimality here is, of course, a tricky notion to come to terms with, but an evolutionary view of biological fitness is usually taken in the literature to provide a well-grounded scientific vantage from which the naturalist can view structures operating in terms of that notion. So suppose the naturalist turns to the "reverse engineering" strategy to identify functions based primarily or, at least in the first instance, solely on those current structures that according to the best hypotheses about the species under investigation seem to contribute to individual survival and reproduction. Concerned with human functioning, the naturalist must employ only the methods of the natural sciences to account for what structures count as "normal" functions and what level of functioning counts as "normal"

⁸ It is difficult to overstate the importance of a default assumption of "optimality" for the naturalist. Most accounts of function view "function" in terms of a conception of optimality—though, they may not express it as "optimality", *per se*,—with which, to quote Searle, "a whole vocabulary of success and failure is now appropriate that is not appropriate to simple brute facts of nature. Thus we can speak of 'malfunction', 'heart disease' and better or worse hearts". Searle (1995), *op cit.*, p. 15.

⁹ Dennett (1995), *op cit.*, at p. 213.

variation. That is, it must offer solutions to the qualitative and quantitative issues of functions that is ontologically and semantically objective by (O*) and (S*). As we have seen, this will require principled grounds for distinguishing between instances where one's biological structure is performing its function, i.e., "functioning", and "functioning successfully", construing the latter, presumably, as, "functioning normally". This is, in schematic outline, the project. I will be arguing that both in outline and in the details such a project built on a union of the ideas of reverse engineering and evolutionary history will run into some intractable difficulties.

2.2 On the solutions to the qualitative issues about functions

To motivate the roots of some of these difficulties, consider three obvious facts that the reverse engineering strategy must account for. First, the reverse engineering strategy must account for the obvious fact that a biological structure may adequately contribute to individual survival and reproduction in one sort of environment (internal and external) but not in a different sort of environment. An example of an "internal" environment which illustrates this is the example of (i) the human heart and the variety of reasons (e.g., toxins, hormonal abnormalities, blood volume, blood viscosity, and the resistance of the blood vessels) that would render the heart unable to adequately perform its function of circulating blood where, it seems to me, it would be a mistake to count the heart not to be normal or healthy. To further press this fact, recall that:

- (ii) An individual suffering from diabetes mellitus may have her blood sugar levels restored by an insulinoma—an islet cell tumour of the pancreas (Chap 5, Sec 4.2).

An example of an "external" environment, which illustrates this, is the famous example

of Sickle cell anemia. Recall that:

- (iii) An individual with the Sickle cell trait possesses a survival advantage in low altitude environments where malaria is a pandemic, mosquitoes are in abundance, and there is a complete lack of low-oxygen environment (Chap 4, Sec 4.3.3 & Chap 6, Sec 3.2).

And there are, to be sure, many other examples one could readily draw upon, but these three will suffice for our purposes.

Second, the reverse engineering strategy must clearly contend with the fact that, for any biological structure ascribed a “function”, there is an enormous amount of variation in the *actual* level of performance by that structure of the function. And, moreover, some of these levels will be such that they surely must be counted as *failing* to perform its function or as performing it at a level which endangers the very life of the organism or which makes procreation unlikely, while other levels surely must be counted as to performing its function at a high level of efficiency or effectiveness (various terms are fitting here). For brevity in the discussion to come (and even though the terms are less than ideal) I will refer to the first class of scenarios as “unsuccessful” and the second as “successful” functioning. It is important that both successful and unsuccessful functioning admit of degrees. As would be the case, for example, with those eyes that completely lack visual light perception, those eyes that have an aided visual acuity measurement of 20/20, and those eyes that have an unaided visual acuity measurement of 20/20, respectively.

And, finally, for all human functions, there is the fact that a successful level of functioning will vary between individuals given their age, sex, present activity, and the environment. That this is so is easy to illustrate with two noncontroversial examples. Consider the undeniable fact that newborn infants neither walk nor talk and cannot

reproduce yet the typical young adult can and that fertility in females declines with age and ends with menopause, while males typically remain fertile into old-age. Thus, if an account of function which at least begins with results developed using the idea of reverse engineering is to reflect this, then it must have the resources to be able to explain how a physiological structure's particular level of functioning is to be counted as *successfully* performing its function for some individuals (e.g., the legs of an infant) and to count the exact same level of functioning as *unsuccessfully* performing its function for other individuals (e.g., the legs of a young adult).

In light of these three facts, it therefore seems that, even if the reverse-engineering approach *might* in a manner reminiscent of Cummins make some interesting suggestions on how to address the *qualitative* issues concerning functions, it will have the (by now) familiar difficulty addressing the *quantitative* issues concerning functions. For it surely must be the case that a reverse engineering strategy can infer to function and normal functioning in a such a way that (A) must allow for the variations in *level* of functioning which can be the result of age and sex differences and which takes into account differences in the environment in which the individual lives its life—its “natural” environment; and (B) must have principled grounds for distinguishing instances where one and the same level of functioning in one environment contributes to biological fitness, i.e., promotes successful biological functioning, but in another environment threatens biological fitness, i.e., threatens biological functioning. That is, the reverse engineering strategy must have principled grounds for distinguishing between instances where the organism's biological structure is functioning “successfully” and instances where the structure is functioning “unsuccessfully”, even when the levels of functioning in the two cases are strictly

speaking the same.

With regards to (A), it is difficult to see how, in the absence of a statistical conception of normality, the reverse engineering strategy could ever do this. After all, it is not as if the examination of any one particular structure-*token* can by examination yield evidence of what is to count as the “normal” level of functioning for structures of this *type*—relativized to age and sex reference classes, or otherwise. That this is so casts legitimate doubt upon the very assumption on which the viability of the reverse engineering strategy here depends—a connection between structure and function exists which, from structures alone, permits an entirely objective science-based enterprise to specify the requisite conception of normal functioning. For, if the reverse engineer is unable to establish the normal level of functioning for the species-*type* that is relativized to reference classes, then the naturalist cannot appeal to any sort of claims about satisfactory species design when determining if a particular function is or is not functioning normally.

The lesson here, I suggest, is that the identification of the principled grounds required to specify normal functioning and hence to specify what is to mark successful functioning from unsuccessful functioning cannot be grounded in some sort of naturalist conception of species design. How then, a reasonable person might wonder, does the reverse engineering strategy effectively address the issue mentioned in (B)?

A pervasive difficulty is that the reverse engineering strategy cannot turn to empirical science in the same way we saw our group of naturalists in the “waligators” thought experiment (Chapter 6, Section 1.1) do so. Concerned with the waligator’s tusks, recall, our naturalists advanced a conception of the “normal functioning” of waligator’s tusks from reading off the “literally true” levels of actual performance of the waligator’s

tusks thus far observed. A significant challenge was then to specify a conception of the normal functioning of the waligator's tusks in the face of both the possibility that any part of an individual waligator's "regulating" systems (e.g., homeostatic mechanisms and biological structures that contribute to biological fitness as defined) may be "diseased", and the clear variability in successful functioning.

To address this issue, we saw the naturalists, very reasonably, determine whether a particular waligator's tusks are functioning *normally* by comparing its level of functioning against the level of functioning of other relevantly similar (i.e., same age, sex, etc.,) waligators. In effect, this statistical norm establishes the level of functioning requisite for a waligator's tusks to properly count as functioning *normally*. That is, the statistical norm serves as the principled standard that distinguishes the successful from the unsuccessful functioning. The problem with this way of addressing the issue, however, is that one ends up appealing to a statistical conception of normal functioning that is relativized to reference classes, which, for reasons that I hope are clear by now, is a conception that the naturalist now seeks to avoid.

Is there another way for the reverse engineering strategy to address the need for an explication of what counts as successful and unsuccessful functioning, i.e., to deal with the problem (B)? Unfortunately, in the absence of a statistical conception of normality, there are reasons to doubt that any conception of normal functioning with the requisite principled grounds can ever be developed.

Strong evidence comes from patients suffering from diabetes mellitus who have their blood sugar levels restored by an insulinoma—an islet cell tumour of the pancreas. Now here we have two states, one of which, diabetes mellitus, increases blood glucose

levels; and the other, an insulinoma, which raises insulin levels. The surprising result (recall from Chap 5, Sec 4.2) is that an insulinoma may serve to “normalize” the blood glucose levels in the diabetic patient and, as such, instead of causing hypoglycemia, the tumour effectively ameliorates diabetes.

To summarize the point rather provocatively in some cases an organism’s having two diseases may serve to promote biological fitness and, even more provocatively, to preserve the health of the patient. This said, modern medicine will count each state to be a disease¹⁰ and, as such, counts an individual with both states to be diseased. For the remainder of this section my aim is to show that the reverse engineering strategy lacks the resources to embrace this view of modern medicine without forcing the naturalist into an intractable position.

Let us begin with the following consideration. If, as Dennett claims, using the reverse engineering strategy forces us to bring to bear some conception of optimality which the naturalist, presumably, will have to cash out in terms of biological fitness, then it becomes problematic on what grounds the naturalist *could* offer when spelling out principled grounds for echoing modern medicine’s counting an individual with insulinoma and with diabetes as having two diseases. In other words, what are the principled grounds for not counting a structure to be functioning “normally” (read: successfully) when it contributes to the survival and reproduction of the individual in its particular environment?

¹⁰ The *ICD-10* differentiates between a malignant and a benign insulinoma with the codes *C25.4* (malignant neoplasm: endocrine pancreas) and *D13.7* (benign neoplasm: endocrine pancreas). It assigns the codes *E10-E14* to diabetes mellitus, differentiating between the type of diabetes mellitus, the body system affected, and the complications affecting that body.

A significant challenge, yet one that must be overcome, is to specify the normal or successful functioning of biological structures in the face of the ever-present possibility that a regulative system (e.g., homeostatic mechanisms, biological structures relevant to the biological fitness to the organisms) may be diseased and the clear variability in successful functioning.

And herein lies the crux of the problem, as I see it: the methods of empirical science cannot uncover the requisite conception of species-*type* “normal functioning” at least not simply by reading off the “literally true” objective facts of *token* biological structures without employing some sort of statistical conception of normal functioning. But for reasons stated above, this is precisely what the reverse engineer must insist upon, and, *a fortiori*, so too must the naturalist: a conception of health and disease that is ontologically and semantically objective by (O*) and (S*) cannot but depend on the “literally true” objective facts of *token* biological structures—there is simply no other naturalist game in town. Thus I conclude that, in rejecting a statistical conception of normality, one avoids the problematic statistical norms of reference classes; but, in doing so, one throws aside what seems to be the naturalist’s only principled way of specifying the normal level of functioning for the species-*type* of any function.

If the argument above is correct, then the reverse engineering strategy cannot refuse to count some particular biological structure (e.g., insulinoma, insulin secretion and/or absorption mechanisms) as functioning normally just because it compares unfavourably when considered against a conception of the “normal” level of functioning (e.g., insulin and blood glucose levels) for the species-*type*. At this point in the implementation of a reverse engineering strategy for identifying function, the species-*type* normal functioning

of any biological structure is not available—it is simply unknown. So it seems that, in the absence of a statistical conception of normality, the reverse engineering strategy winds up committed to saying that the species-*token* normal functioning must be relativized to the contribution to the survival and reproduction of the individual in its particular environment. In effect, this is a very similar commitment that we saw the naturalist proponent of a forward-looking account of function was forced to make as well.

At the least, this commitment, just as we saw was the case with forward-looking accounts of function, faces some serious difficulties. In fact, many of the most damaging arguments we saw raised against grounding the BST on a forward-looking account of function apply equally well, *mutatis mutandis*, against grounding a naturalist conception of health and disease in an account of function which starts from use of the reverse engineering strategy. Most notably, it also forces an obvious and significant departure from what modern medicine takes to be normal functioning, which (i) counts a disease—or two diseases!—*not* be a state of successful and hence of normal functioning¹¹ and (ii) counts states to be “diseases” (e.g., malaria resistant genetic disease α -Thalassemia, Sickle cell anemia, Type II diabetes mellitus, and cowpox) despite strong evidence that they confer a survival advantage in some environments.

If the naturalist wants to say, which surely one must (for reasons stated below), that a disease—or two diseases!—can *not* be a state of successful and hence of normal functioning, then it seems that the naturalist winds up committed to insisting that a

¹¹ Furthermore, there is the strong intuition—which modern medicine echoes—that “normal aging” is not a disease, which surely demands the view that disease is a deviation from (some sense of) normal functioning.

substantial disconnect exists between, on the one hand, the current structures *of a particular individual* which contribute to one's survival and reproduction and, on the other hand, what is to be classed as "normal" functioning (i.e., the solutions to the quantitative issues of functions). This provides further reason to insist that the conclusion Lauder draws from his study of salamanders also applies to humans.

For the naturalist to insist as much, however, runs against the very assumption on which the viability of the reverse engineering strategy depends: That a connection between structure and function exists which, from structures alone, permits an entirely objective science-based enterprise to yield the right sort of function ascriptions. If this is correct, then the naturalist finds oneself caught in a dilemma: Either the naturalist ends up committed to saying that there is a substantial disconnect between the accounts of *current* structures and those of *normal* functioning and thus denies the needed assumption on which the viability of the entire reverse engineering strategy depends. Or the naturalist ends up committed to saying that, given the right sort of internal and/or external environment, one or more disease state can be classed as a state of normal functioning, and thus advances an account of function that explains away or overrules what are surely some of modern medicine's most noncontroversial health and disease claims—an implication which is of course, at bottom unacceptable given the naturalist's aim to underpin medicine with a theoretical, value-free science of health and disease.

Given these two options, the naturalist might well elect to bite the bullet and advance health and disease claims that are at odds with the methods of modern medical practice. Here we would do well to keep in mind that medical practice has a long and troubling history of mistaken health and disease claims that we are thankful to have

outgrown. And, *a fortiori*, there is no reason to think that the modern medicine is immune from advancing—wittingly or unwittingly—mistaken health and disease claims that do not deserve the same fate.

To such a move, I have the following response. More troubling than advancing a contentious biomedical account of normal functioning is that the reverse engineering strategy for identifying “normal” functioning now seems unable to avoid the Cambridge-change objections. This is because, as stated above, when the conception of normal functioning (i.e., the solutions to the quantitative issues of functions) becomes relativized to an individual’s “natural” environment, then the naturalist is forced to concede that: (I) it could be the case where an individual in one sort of environment is properly counted to be functioning successfully or normally (read: healthy) and another individual in a different sort of environment is properly counted to be functioning unsuccessfully or subnormally (read: diseased), *despite there being no physiological difference between the two individuals*; and (II) that an appropriate sort of environmental change (e.g., malaria becoming endemic, eradication of malaria) would warrant an individual to go from being diseased to healthy, or vice versa, without any physiological change in that individual. Either concession leaves a reverse engineering account of function wide open to the Cambridge-change objection.

2.3 Section conclusion

What the preceding shows is that there is substantial reason to doubt that a satisfactory naturalist account of function will emerge from the implementation of a reverse engineering strategy in the way outlined above. Equally clear, I submit, is the fact that

there is no reason to think that a reverse engineering strategy for identifying the requisite account of functioning will enable a naturalist conception of health and disease to avoid the Cambridge-change objection. There is, however, good reason to think that it will not.

Thus I conclude: If there is a way for the naturalist to advance the requisite account of normal functioning without making use of a statistical conception of normality of some sort, I cannot see it. What can we draw from this admittedly less than decisive conclusion?

I think that, following the arguments set out in the previous chapters, we now have a further compelling reason both to reject the candidacy of Boorse's BST to furnish medicine with a basic scientific concept of health disease, and to doubt the resources of naturalism to advance a satisfactory value- and norm-independent conception of health and disease. In any case, we have substantial reasons to reject the naturalist's leading offer to underpin medicine with a theoretical, value-free science of health and disease.

Considerable comfort can be found not so much on the rejection of naturalism's main contender—for there could conceivably be better naturalist offerings toeing the line—but in the force of the rejection. Indeed, it seems plausible to say that we are rejecting the most promising naturalist conception of *normal functioning*.¹² After all,

¹² It is difficult to overstate the fundamental rôle Boorse and others assign to a conception of *normal functioning*. Boorse, for instance, repeatedly claims that the normal-pathological distinction is the basic theoretical concept of Western Medicine. See Boorse (1987), *op cit.*, pp. 364-365 and (1997), *op cit.*, p. 7. Accordingly, he insists often that the departure from "normal functioning" is a necessary condition for disease. See Boorse (1975), *op cit.*, pp. 57-58; (1977), *op cit.*, pp. 550 & 571; (1987), pp. 370 & 385; and (1997), pp. 64, 98, & 99. Others insisting as much are, for instance, Szasz (1960) *op cit.*; Wakefield (1992) *op cit.*; Allen Buchanan, Dan W. Brock, Norman Daniels, & Daniel Winkler (2001), *From chance to choice: Genetics and justice*. Cambridge: Cambridge University Press; Norman Daniels (2008), *Just health: Meeting*

ultimately what we are rejecting is defining health (and, by implication, disease) in terms of a statistical conception of normal biological functioning grounded on evolutionary biological fitness. In any case, if there is a better naturalist account of normal functioning waiting in the wings, then it ought to be put forth. Unless and until this is done, we would do well to begin to appreciate the ways in which a satisfactory conception of health and disease will be value- and/or norm-dependent.

3. Further implications and strategies for change

3.1 *Taking naturalism seriously*

Though I am convinced that the best articulated and defended conception of health and disease will be not be value- and norm-independent, it is imperative that we continue both to encourage and to pursue the naturalist endeavour. Taking naturalism seriously repays a better understanding of exactly when and the extent to which a satisfactory conception of health and disease *must* have purportedly value- and norm-independent criteria give way to normativist elements. And, as we shall see, it is difficult to overstate the importance of acquiring an accurate assessment of what our commitment to normativism minimally requires.

It is both alarming and obvious that a commitment to a normativist conception of

health needs fairly. Cambridge: Cambridge University Press; and Thomas Schramme (2010), “Can we define mental disorder by using the criterion of mental dysfunction? *Theoretical Medicine and Bioethics* 31: 35-47. For further reasons, a conception of normal functioning is no less important to Dan Brock who claims that, “quality of life must always be measured against normal, primary functional capacities for humans...”; and to Norman Daniels who defines his crucial accounts of ‘health needs’ and ‘normal opportunity range’ in terms of Boorse’s conception of normal functioning. See Dan W. Brock (1993), *Life and death*. Cambridge: Cambridge University Press, p. 308; and Daniels (2008), *op cit.*, especially at pp. 36-46.

health and disease travels with profound dangers, provided we are willing to see it. To put the matter starkly, there is always the legitimate worry that our conception of health and disease may be corrupted by values and norms that are in some important senses “illicit”, not only scientifically, but also socially, morally, ethically, and politically. Even more troubling, there is genuine reason to think that our present health and disease judgements *are* being skewed to the ideology and interests of a select few powerful and influential stakeholders.

The discussion (in Chapter Two) of the way in which GlaxoSmithKline (GSK) went about changing the cultural understanding of depression to market its drug *Paxil* is but one instance of a pharmaceutical giant simply manufacturing “conditions” to sell drugs to further—and let’s call a spade a spade—an ideologically tendentious corporate mandate. This one instance, however, is indicative of an underlying tension between “realizing the goals of healthy people and healthy communities” and “realizing the goals of for-profit companies and shareholders”. Unfortunately, this is not an isolated case.

Instances where our health and disease judgements seem to be under the thumb of an 800-pound gorilla are ubiquitous in modern society and perhaps most noticeably in modern psychiatry.¹³ To my mind, nowhere is this more evident than with the fourth edition of the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*. Consider, for example, some of the proposed disorders for

¹³ See, for instance, the two recent and very provocative articles by Marcia Angell (the erstwhile editor-in-chief of the *New England Journal of Medicine*): Marcia Angell (2011a), “The epidemic of mental illness: Why?”, *The New York Review*, June 23, 2011. <<http://www.nybooks.com/articles/archives/2011/jun/23/epidemic-mental-illness-why/>> Accessed July 4, 2011; and (2011b), “The illusions of psychiatry”, *op cit*.

DSM-V (to be published in May 2013), that are not currently listed in *DSM-IV*:

- (A) Disruptive Mood Dysregulation Disorder; Posttraumatic Stress Disorder in Preschool Children; Unspecified Trauma- or Stressor-Related Disorder; Sexual Dysfunction Associated with a Known General Medical Condition; Sexual Interest/Arousal Disorder in Women; Major & Minor Neurocognitive Disorder Associated with Substance Use; Hoarding Disorder; etc., etc.,

and some of the currently listed disorders with proposed revisions:

- (B) Oppositional Defiant Disorder; Antisocial Personality Disorder; Erectile Disorder; Female Orgasmic Disorder; Early Ejaculation; Premenstrual Dysphoric Disorder; Depersonalization/Derealization Disorder; Alcohol Use Disorder; Cannabis-Use Disorder; Tobacco Use Disorder; Other (or Unknown) Substance-Use Disorder; Gambling Disorder; Generalized Anxiety Disorder; Nightmare Disorder; Illness Anxiety Disorder; etc., etc.,¹⁴

By no means is this meant to be an exhaustive list; many more “disorders” could easily be added.

In effect the list of “disorders” outlined in (A) and (B) is a mirror of the medicalization¹⁵ of perceived problems. This drive to medicalize is troubling for a number of reasons. Not the least of which is the reality that there are profound consequences—indeed life and death consequences!¹⁶—for both society and the individual, from the

¹⁴ American Psychiatric Association. *DSM-5: The future of psychiatric diagnosis* <<http://www.dsm5.org/Pages/Default.aspx>> Accessed July 4, 2011.

¹⁵ Here I am using the term ‘medicalization’ as Peter Conrad does when he says: “Medicalization consists of defining a problem in medical terms, using medical language to describe a problem, adopting a medical framework to understand a problem, or using a medical intervention to “treat” it. This is a sociocultural process that may or may not involve the medical profession”. See Peter Conrad (1992), “Medicalization and social control”, *Annual Review of Sociology* 18: 209-232, at p. 211.

¹⁶ In a recent article about the famous mathematician Alan Turing, Thomas Szasz discusses a provocative real-life example of the profound life and death consequences that can and do result from improper medicalization that merit some words. Szasz tells of how Turing, after confessing to his homosexual affair, was arrested and charged with the crime of “gross indecency” in 1952—A crime punishable by two years imprisonment. To avoid jail, Turing was obliged to undergo “organo-therapy” for the same period. Szasz writes: “In April 1952 he [Turing] wrote to a friend, ‘I am both bound over for a year and obliged to take this

inability to curtail improper medicalization. This is an old, familiar problem, yet regrettably it remains a perennially pertinent problem.

The literature is often quick to point out shameful instances where disease-status was surely incorrectly given to behaviours viewed to be deviant and undesirable, some of which (e.g., drapetomania, masturbation and homosexuality) I spoke of earlier (Chapter 2, Section 4.1). In this context, instances of improper medicalization and treatment, while often an apt reminder that normal people suffer from perverse treatments when perverse treatments become normal, seem more like expressions of mistaken scientific facts than the need to circle the wagons and bulwark against illicit values and norms arranging marriages between disease-status and behaviours simply viewed by some to be “deviant” and “undesirable”.

Old problems can indeed be so stubbornly resistant to change. It is telling, for instance, that up for a proposed revision in the *DSM-V* is a “condition” called “Oppositional Defiant Disorder”. After reading the proposed revisions (see Appendix A below), one wonders whether the American Psychiatric Association (APA) and its stakeholders are oblivious to or inspired by the Soviet-era practice—a practice which is commonly cited in the literature—to revise definitions of mental illness such that political dissidents were deemed mentally ill and, as such, forcibly detained in mental institutions.

organo-therapy for the same period. It is supposed to reduce sexual urge whilst it goes on, but one is supposed to return to normal when it is over. I hope they're right.' Turing was never the same again. His body became feminized. He grew breasts...On June 8, 1954, Turing was found dead by his housekeeper, a partly eaten apple laced with cyanide next to his bed". See Thomas Szasz (2009), "The shame of medicine: The case of Alan Turing", *The Freeman* 59(4) <<http://www.thefreemanonline.org/columns/thetherapeutic-state/the-shame-of-medicine-the-case-of-alan-turing/>> Accessed July 4, 2011.

(Unfortunately, this may not be a vestige of the past as several notable news agencies have recently reported that Russia has revived this practice¹⁷).

If one accepts the proposed criteria of “Oppositional Defiant Disorder”, then likely there isn’t a teenager in the world that, at one time or another, couldn’t be counted to have this “disorder”. Importantly, whether teenagers are properly counted to have a “genuine” disorder or to be simply disorderly (as surely so many ordinary teenagers are) will depend on how the disorder is defined. Without denying that some teenagers suffer from a genuine mental disorder, the broadness of the criteria and its vulnerability to improper medicalization must be acknowledged. And because of this I challenge anyone following the proposed criteria to a tee to show how an individual deemed to be a political dissident wouldn’t *ipso facto* properly count to be suffering from oppositional defiant disorder.¹⁸

As always, the real fear of course is that we are on a slippery slope to where health and disease becomes a social engineering control of a population’s behaviour. In fact, I believe we have slid much further down the slope than many of us would like to believe. For what else remains to revive the Soviet model of institutional confinement but to establish that “the symptoms” of oppositional defiant disorder are such that individuals

¹⁷ See, for instance, Alastair Gee, “Russian dissident ‘forcibly detained in mental hospital’”. *The Independent* July 30, 2007 <<http://www.independent.co.uk/news/world/europe/russian-dissident-forcibly-detained-in-mental-hospital-459539.html>> Accessed July 4, 2011. And Mark Franchetti, “Putin brings back mental ward torment” *The Sunday Times* August 26, 2007 <<http://www.timesonline.co.uk/tol/news/world/europe/article2327824.ece>> Accessed July 4, 2011.

¹⁸ It should be point out that IF such an individual did not sufficiently meet the criteria there is always the diagnosis of ‘Q04 Other Specified Disruptive or Impulse Control Disorder’, which the APA states: “This category is for disorders characterized by conduct or oppositional defiant behaviors [sic] that do not meet the criteria for Conduct Disorder or Oppositional Defiant Disorder. For example, include clinical presentations that do not meet full criteria either for Oppositional Defiant Disorder or Conduct Disorder, but in which there is clinically significant impairment”. See, American Psychiatric Association. <<http://www.dsm5.org/ProposedRevision/Pages/proposedrevision.aspx?rid=107#>> Accessed July 4, 2011.

are deemed to be at “serious” risk either to themselves or to society? Common sense certainly would not have such individuals forcibly committed to institutions on such broad and vague criteria (e.g., “The disturbance in behavior [sic] causes clinically significant impairment in social, educational, or vocational activities”) alone. But no one denies that common sense can be all too uncommon. Or that a country can be so massively cruel to its citizens.

To focus on “operational defiant disorder” is to reveal just the very tip of the *DSM* iceberg of highly contentious “disorders”. A recent *Globe and Mail* article notes that the *DSM-II* listed 182 disorders over 150 pages, while the current *DSM-IV* lists 365 disorders and contains over 900 pages¹⁹—and the *DSM-V* proposes to add even more disorders! It is impossible to ignore that the drive to medicalize new “conditions” is not without serious and profound consequences. Even the former editor of the *DSM-IV*, Allen Francis, admits as much:

Because of the power of drug-company marketing and the Internet and consumer-advocacy groups, there have been a number of false epidemics, of fads in psychiatric diagnosis that have resulted in tremendous diagnostic inflation and much higher rates of mental disorder than ever before...And many more people getting medication, which in many cases is not useful and may be harmful.²⁰

There is an important lesson here. What is true, at most here, is that our health and disease judgements may be *said* to be built upon evidence-based medicine and scientific

¹⁹ Ian Brown. “Where is its mind? What the battle over the ‘bible’ says about psychiatry”, *Globe and Mail*, 09 July, 2011 <<http://www.theglobeandmail.com/life/health/new-health/conditions/addiction/mental-health/where-is-its-mind-what-the-battle-over-the-bible-says-about-psychiatry/article2091844/page1/>> Accessed July 10, 2011. My thanks to James Ash who brought to my attention this revealing article

²⁰ Ibid.

facts—but it is, to be sure, values and norms that paves the way. Increasingly it is powerful and influential stakeholders blurring the boundaries between sophisticated marketing and medical science. But such stakeholders are by no means limited to the pharmaceutical industry as Carol Bernstein, the president of the APA last year, in effect concedes:

It became necessary in the 1970s, to facilitate diagnostic agreement among clinicians, scientists, and regulatory authorities given the need to match patients with newly emerging pharmacologic treatments.²¹

Clearly there is genuine reason to think that our present health and disease judgements *are* being skewed to the ideology and interests of a select few powerful and influential stakeholders. What this means is that those of us truly concerned not to advance health and disease claims that are scientifically dubious and ideologically tendentious must be vigilant about insisting that value- and norm-independent science informs our health and disease judgements—including the essential values or norms on which a satisfactory conception of health and disease depend!—to the greatest extent possible. This, of course, requires taking naturalism seriously.

3.2 *Taking normativism seriously*²²

I have argued, *pace* naturalist theorists concerning health and disease, that a satisfactory conception of normal functioning will not be value- and norm-independent. The recognition that the best conception of health and disease available to guide responsible²³

²¹ Angell (2011b), *op cit.*

²² Portions of Sections 3.2 & 3.3 have been published as Guerrero (2010a), *op cit.*

²³ Clearly, further elaboration and qualification is called for. However, for the present purposes, it is enough to distinguish a responsible health approach from a health approach that stands content to build upon a

health research, policy, and practice *will* be crucially a function of human values or norms should provide the impetus for a serious encounter with normativism. That being said, I suspect many will be resistant to openly embracing a normativist conception of health and disease. I am not blind to the allegiance that many (probably most) physicians, scientists and, more generally, proponents of “evidence-based” medicine and healthcare have to purportedly “objective” science.

I have the following response to such resistance: Without discounting the genuine dangers of normativism, there is a decisive reason to openly embrace a value- and/or norm-dependent conception of health and disease. Though I have not explicitly mentioned it until now, there is, as it stands, an unmovable naturalist elephant in the room. This naturalist elephant is easy to see—deceptively so—when we are willing to appreciate what is surely an undeniable fact of naturalism. A naturalist conception of health and disease, and hence of normal functioning (i.e., a conception that is ontologically and semantically objective by (O*) and (S*)), must be entirely dependent on the “literally true” biological facts of nature. In effect, this means that a naturalist conception of normal functioning only tracks how we are (or perhaps, in the case of our close ancestors, how we once were) in fact functioning. This tenet of naturalism is what I want to call the naturalist elephant in the room.

The problem can be summarized like this: The naturalist must insist that what

clearly inadequate conception of health. Part of the idea here is that, given the tremendous practical implications involved, so many of us want to be able to say that it would be irresponsible not to employ the best conception of health and disease that could be employed; and, moreover, that we *should not* employ a conception of health and disease that clearly undermines our alleged health aims.

essentially matters for health is, *ipso facto*, statistically normal functioning. But a naturalist conception of statistically normal functioning only tracks how we are (or perhaps how we once were) in fact functioning. There is simply no guarantee, however, that how we are in fact functioning is sufficient to be functioning well or even how we *should* be functioning. It is not a sufficient condition for health because, simply put, an unhealthy individual can be functioning at a level that is, statistically speaking, normal. Even if all humans were to become grossly obese diabetics, there would still be people functioning at or above what is statistically speaking “normal”; yet insofar as gross obesity and diabetes clearly undermines an individual’s ability to function and *a fortiori* to survive, we should surely not want to count such people as healthy—statistically normal or not.²⁴ Hence, operationalizing a naturalist conception of health and disease, wedded as it is to the above naturalist tenet, is fraught with dangers.

Importantly and significantly, there is reason to think that many individuals are not in fact functioning how they should be. One way to see this is to recall (from Chapter 6, Section 3.1.1) what I called the “increasing population problem”. In motivating this problem, a conclusion was emphasized, which Sir Michael Marmot and others have persuasively drawn—that economic and social inequities are related to causation of ill-health. It was further noted that both the Canadian Public Health Association and the Public Health Agency of Canada have long insisted that income and social status are

²⁴ This is not to say that we may never have legitimate reasons to count a grossly obese individual to be more “healthy” in comparison to another individual (e.g., a grossly obese individual with terminal cancer). However, in such instances I think we could just as easily, and more accurately, say such an individual is less diseased.

strong determinants of health. Unless one wants to deny the fundamental principles of the population/public health approach—an approach most countries officially support—then one must acknowledge that individuals from countries with *avoidable* socio-economic inequalities and inequities may not be functioning as well as they could and, perhaps, *should* be.

All of this strongly suggests that a fundamental problem with a naturalist conception of health and disease is that its best conception of normal functioning is hopelessly interwoven with existing health inequalities and inequities. Imagine if Canadian health research, policy, and practice were to champion a naturalist conception of health, then it will be led to perpetuate the very health disparities that key stakeholders have acknowledged engenders ill-health and have pledged to tackle.²⁵ This *is* unacceptable. In my view, a striking statistic (introduced in Chapter 6) which drives this point home is that, to quote the Senate of Canada, “[f]ully 50% of the health of the population can be explained by socio-economic factors”!

This becomes an especially significant claim because the unfortunate fact of the matter is that Canada offers a paradigm example of socio-economic hierarchies having a determining impact on ill-health. It is no coincidence that the socio-economic status of First Nations peoples is lower than that of non-Aboriginal Canadians on virtually every measure²⁶ and (i) registered First Nations peoples can expect to live over seven years

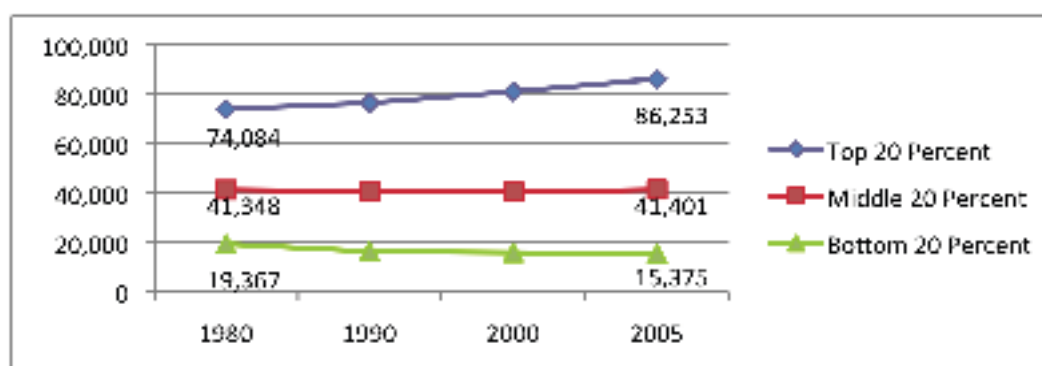
²⁵ Consider, for instance, Canada’s pledge to the *Ottawa Charter for Health Promotion* and, in particular, “to respond to the health gap within and between societies, and to tackle the inequities in health produced by the rules and practices of these societies”. World Health Organization (1986), *op cit.*, at p. 4. Consider also the two standard objectives of a population health approach cited below (footnote #37).

²⁶ Canadian Institute for Health Information (2004), *Improving the health of Canadians*. Ottawa: Author, p.

less;²⁷ (ii) the infant mortality rate and the prevalence of heart disease is over 1.5 times higher;²⁸ (iii) the obesity rate is twice as high²⁹; and (iv) the tuberculosis infection rates are 6 times higher than that of the general Canadian population.³⁰ Indeed, there is a very clear message here: *poorer Canadians have poorer health*.

To their credit, as stated above, key stakeholders in Canada acknowledge this message. Despite this, however, the reality is that the majority of Canadians are getting poorer. As Table 1 shows, disparities in income are growing in Canada, as the rich are getting increasingly richer while the poor are getting increasingly poorer.

Table 7.1: Increasing Disparity in Income in Canada (1980-2005)



Source: Statistics Canada. Earnings and incomes of Canadians over the past quarter century, 2006 Census, May, 2008. Catalogue no. 97-563-X.

This data reveals some stark underlying facts. Most notably, between 1980 and 2005, median earnings among the top one-fifth of full-time wage earners increased by

80. See also, Senate of Canada, 2009, *op cit.*, p. 9.

²⁷ Health Council of Canada (2005), *op cit.*, p. 43. See also, Senate Canada (2009), p. 9.

²⁸ Canadian Institute for Health Information (2004), *op ct.*, p. 8. See also, Health Council of Canada (2005), p. 45; and Health Canada (2008), *Healthy Canadians—A federal report on comparable health indicators 2008*. Ottawa: Author.

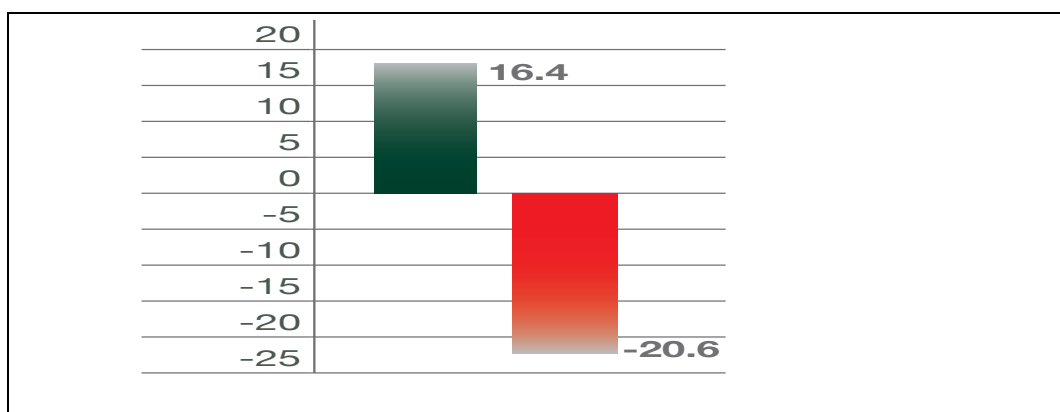
²⁹ Health Council of Canada (2005), p. 6.

³⁰ *Ibid.*

16.4%. In contrast, the median earnings among those in the middle 20% increased by only 0.1%; while for those among the bottom one-fifth of wage earners, their median earnings fell by 20.6%. As Table 2 (below) graphically shows, the rich (top 20%) have gotten significantly richer, while the poor (bottom 20%) significantly poorer!

Canada is a country with significant—and growing—socio-economic disparities. This is troubling for a variety of reasons; not the least of which is the overwhelming evidence that economic and social inequalities engender ill-health and *a fortiori* is a health issue. Quite simply, there is thus a substantial element of complaint with the increasing economic and social disparities amongst Canadians that is justified.

Table 7.2: Percentage of Change in Income in Canada (1980-2005)



Source: Statistics Canada. Earnings and incomes of Canadians over the past quarter century, 2006 Census, May, 2008. Catalogue no. 97-563-X.

Considered from the point of view of health, what can be said—and what should be said!—is that our current economic, political and social practices are *avoidably* perpetuating the ill-health of many Canadians. Certainly, it is difficult to insist otherwise given that, in 2001, almost a third (27.7%) of on-reserve aboriginal houses failed to satisfy

at least one core housing standard.³¹ Close to a quarter (23%) of First Nations on-reserve housing units were identified to have inadequate (in terms of volume and/or health requirements) or no water services and nearly one-fifth (19%) to have inadequate or no sewage services.³² This is simply shameful. We report these statistics, we explicitly acknowledge that inadequate housing, water and sanitation systems engender ill-health³³ and, worse still, we could surely right these deplorable conditions if we collectively chose to.

Indeed, it is simply not the case that Canada lacks the requisite resources to improve these conditions. After all, Canada is a high income OECD member³⁴ that has recently demonstrated the ability to marshal what is estimated to be as high as \$25-billion (US) to bailout the auto industries.³⁵ This is money that could have instead been used to bailout, say, the one in four children through no fault of their own, currently living in poverty in First Nation communities.³⁶

Choices were made; and we could have chosen otherwise. One could say that the

³¹ Health Canada (2009), *A statistical profile on the health of First Nations in Canada — Determinants of health, 1999 to 2003*. Ottawa: Author, pp. 33-34. <<http://www.hc-sc.gc.ca/fniah-spnia/pubs/aborig-autoch/index-eng.php>> Accessed July 4, 2011.

³² Health Canada (2009), *op cit.*, pp. v & 34-35.

³³ This view can be found in several notable publications. See, for example, Health Canada (2001), *op cit.*, p. 12; Public Health Agency of Canada. "What is the population health approach: Key elements of a population health approach" <http://www.phac-aspc.gc.ca/ph-sp/approach-approche/appr-eng.php#key_elements> Accessed July 4, 2011; and World Health Organization (1986), *Ottawa Charter for Health Promotion*. Geneva: Author. <www.who.int/hpr/NPH/docs/ottawa_charter_hp.pdf> Accessed July 4, 2011.

³⁴ The explicit mandate of the Organisation for Economic Co-operation and Development (OECD) is "to promote policies that will improve the economic and social well-being of people around the world". <http://www.oecd.org/pages/0,3417,en_36734052_36734103_1_1_1_1_1_1_1,00.html> Accessed July 4, 2011.

³⁵ Karen Howlett, Greg Keenan, & Shawn McCarthy (2008), "Auto bailout tab pegged at \$25-billion." *Globe & Mail* 17 Dec. 2008: B1. *Canadian Periodicals Index Quarterly*. Web. Accessed July 4, 2011.

³⁶ Campaign 2000 (2010), *2010 report card on child and family poverty in Canada: 1989-2010*, p. 1. <<http://www.campaign2000.ca/reportcards.html>> Accessed July 4, 2011. Here it is also noted that, in Canada, nearly one in ten persons, including one in ten children lives in poverty!

correct choices were made. But this requires further and substantive argument. In any case, I think such argument, even if successful, would only explain away an undeniable fact of the matter: That current economic, political and social practices prevent many Canadians from being as healthy as they could (and I would add, as they *should*) be. And so long as this is the case, I see no way for a responsible Canadian health approach to reasonably champion a naturalist conception of health and disease, in general, and a value- and norm-independent conception of normal functioning, in particular.

Aside from advancing a conception of normal functioning interwoven with health inequalities and inequities, embracing naturalism has a different problem: It fails to avoid the Cambridge-change objection. The cost of this, recall, is that the minimal standard of health will be dynamic and may change as the various individuals who presently form the various reference classes change individually, or as the membership of the reference class changes.

To see why is so troubling take a population health perspective as an example. To appreciate the force of the problem, it is worth explicitly noting that the two standard objectives of a population health approach are to improve the health of the entire population and to reduce health inequalities among population groups.³⁷ On this approach, then, an unacceptable conclusion emerges just as soon as one recognizes that a significant increase in “health disparities” between populations could, in fact, lead to a decrease in

³⁷ Public Health Agency of Canada (2011), *op cit.*, Accessed July 4, 2011. This two-fold objective is stated in several notable Canadian publications. See, for instance, the Senate of Canada (2008a), *op cit.*, p. 3 and Health Canada (1998), *Taking action on population health: A position paper for Health Promotion and Programs Branch staff*. Ottawa: Author, p. 1.

level of functioning counted to be requisite of health (i.e., the solution to the quantitative issues of normal functions) such that more—and not fewer!—individuals would properly count as being “healthy”. Surely a population health approach that would, for example, intentionally seek to increase inequalities between or amongst populations in order to count individuals to be “healthy” is not merely factually wrong-headed; it is unjust.

In sum, the preceding shows that there is a decisive reason to openly embrace a value- or norm-dependent conception of health and disease. The fact of the matter is that Canada is a country fraught with existing and significant health inequalities and inequities. And so long as this is the case, the elephant in the room ensures that the best naturalist account of normal functioning is hopelessly interwoven with existing health inequalities and inequities. Quite simply, responsible health research, policy, and practice cannot afford to turn a blind eye to this naturalist elephant—there is too much at stake. Avoiding the unacceptable costs of naturalism, I submit, provides reason to openly embrace a less-problematic normativist conception of health and disease. This, of course, requires taking normativism seriously.

With this in mind, I would like to return a point I mentioned above, i.e., the importance of acquiring an accurate assessment of what our commitment to normativism *minimally* requires. To illustrate and motivate this point, it will be worthwhile to briefly examine two notable accounts of health, which are said to ground a Canadian population health approach. By doing so, I hope to provide a compelling glimpse into how the mechanisms of the framework advanced in this dissertation provides an important and useful way for those of us concerned to identify and bring to light the extent to which a

conception of health and disease depends on values and norms, especially when denied or not explicitly stated. Further still, I hope to show that such a framework is a welcome resource. This is because there are at least two notable accounts of health and disease presumably being operationalized in Canada that are particularly vulnerable to be a function of clandestine values and norms.

3.3 Health: A Canadian perspective

Here it is important to pause and note two things: first, there are several very different accounts of health that seem to be in operation in Canada and, second, for obvious reasons, I can discuss only a couple here.

3.3.1 Account of Health #1

Keeping these two points in mind, an obvious place to start is with the Public Health Agency of Canada (PHAC). On their website, one can find a promising heading entitled “Population Health: Defining Health”. Here we are told that there has been a shift in the thinking of the population health approach such that it no longer embraces the WHO definition of health and that:

The population health approach recognizes that health is a capacity or resource rather than a state, a definition which corresponds more to the notion of being able to pursue one’s goals, to acquire skills and education, and to grow.³⁸

Let us briefly pause here to recall our discussion (Chapter 2, Section 3) of the WHO definition of health. There we saw that a serious problem with the WHO definition of

³⁸ Public Health Agency of Canada (2011), *op cit.*, Accessed July 4, 2011. This exact wording also appears in Health Canada (1998), *op cit.*, at p. 7.

health since is that it embraces *any* alleged criteria of well-being as integral components of health. In other words, to put it bluntly, it is too inclusive.

But, with this in mind, it is difficult to see how the above definition of health—“a definition which corresponds more to the notion of being able to pursue one’s goals, to acquire skills and education, and to grow”—is in and of itself necessarily more limiting. After all, is it immediately obvious why one’s goal could not be to grow into a state of complete physical, mental, and social well-being? Surely not.

In order to avoid an unfair characterization, it is important to note that after stating that their adopted definition of health recognizes the various broad factors that impact health, the PHAC, quoting Frankish et al., goes on to state:

The best articulation of this concept of health is “the capacity of people to adapt to, respond to, or control life’s challenges and changes”.³⁹

Framing health as a capacity in terms of “life’s challenges and changes” is an improvement up to a point; it does, I think, serve to limit the broadness of health to some extent, but exactly to what extent is an open question. Presumably, it would serve to eliminate some unrealistic goals (e.g., for me to be the tallest Canadian) and implausible skills (e.g., time travel) from becoming a central component of health. And insofar as the goal to grow into a state of complete physical, mental, and social well-being could be considered to be unrealistic and/or implausible, then this view of health will be significantly more limiting than the WHO definition.

But surely a reasonable person may wonder if it is still, in fact, too broad a

³⁹ Public Health Agency of Canada (2011), *op cit.*, Accessed July 4, 2011.

conception of health. After all, even a blind diabetic man who has just been poisoned and stabbed in the leg by a scorned lover and bitten in the other leg by a rabid dog has some capacity to respond to life's challenges and changes; yet I think no reasonable person, the PHAC included, would consider this man to be "healthy". Thus a qualification must be added: To properly count an individual (or population) to be healthy we must insist that this individual (or population) possesses not just *any* capacity but some minimal capacity "to adapt to, respond to, or control life's challenges and changes".

The PHAC, of course, does in fact count people and populations to be healthy and unhealthy. And to the extent that they do so, they surely must be employing some conception of a minimal standard of health. Now I confess not to know the PHAC's minimal standard of health. If it ever has been stated, I have never been able to find it and have certainly never come across it. In any case, the minimal standard of health that the PHAC employs is obviously an important and substantive issue, since it—explicitly or implicitly, wittingly or unwittingly—demarcates the healthy from the unhealthy. In light of this, a reasonable person might wonder what exactly the minimal capacity to adapt to, respond to, or control life's challenges (any challenges in life?) and changes the PHAC counts to be requisite for "health". What must be stressed here is that different values and norms will confer legitimacy upon different conceptions of a minimal capacity of "health".

3.3.2 *Account of Health #2*

Another notable account of health can be found in Health Canada's *The Population Health*

*Template: Key Elements and Actions that Define a Population Health Approach.*⁴⁰

Here, we find one notable difference in that the authors explicitly state both (a) their conception of health and (b) that their conception of health provides the foundation for their understanding of the population health approach. The claim, in short, is that a Canadian population health approach rests ultimately on the following understanding of health:

Health is a capacity or resource for everyday living that enables us to pursue our goals, acquire skills and education, grow, and satisfy personal aspirations.⁴¹

This definition is an upgrade at least insofar as “everyday living” may be persuasively thought to serve as some sort of limiting criteria. But this definition of health is still far from being satisfactory because it leaves far too many substantial questions open—questions whose answers carry serious practical implications. For example: to what extent must we have a capacity or resource to be satisfactorily “enabled” to pursue *our* goals and acquire skills and education? Is there a principled standard of “everyday living” or will it vary from individual to individual and/or population to population? If it is the former, then who sets it?

And if it is the latter, then, given that different views of “everyday living” will demand the satisfaction of different levels of skills, education, and growth that will surely require different capacities or resources, it is difficult to see how this conception of health

⁴⁰ It may be worth explicitly noting here the following note found on the PHAC’s website: “Production of this resource has been made possible through a financial contribution by Health Canada prior to the announcement of the establishment of the Public Health Agency of Canada on September 24, 2004. Any reference to Health Canada should be assumed to be to the Public Health Agency of Canada”.

⁴¹ Health Canada (2001), *op cit.*, at p. 2.

would not, in fact, perpetuate some “health” inequalities. After all, different people do in fact want different—and crucially opposing—“goals” and “personal aspirations” satisfied. And if this were the case, then a serious worry would be that *any* whim, unrealistic goal, or fiat could demarcate health. A related and very serious worry here is that it might be claimed that we can improve the overall health of a particular population if we were to have them, say, set different or lower standards when it comes to “*their* goals and personal aspirations”.

3.3.3 Identifying values and norms: A proposal

In my view, there are genuine and serious concerns with each of the above accounts of health, not the least of which is that both of these conceptions of health, in their current formulation, make health claims a function of all-too vague criteria. In effect, both easily embrace quite different—and opposing—sorts of content. After all, one can hardly deny that there are many possible and plausible ways to cash out the above criteria counted to be requisite for health. Most importantly, there is thus the legitimate concern that hiding in the vagueness of these accounts of health are illicit values and norms. One would, therefore, hope that a responsible population health approach would lead the charge in addressing these concerns.

I would now like to motivate some first steps a proactive response would do well to take by quickly viewing the above of accounts of health through the lens of our framework. It will suffice to be brief since I will proceed in much the same way proposed, with some added details, in Chapter 2 of this dissertation.

The first thing to notice is that both the above accounts define “health” in terms of

a “capacity” and a “resource” to “pursue one’s/our goals”, “acquire education” and “to grow”. Further reflection reveals that this key criteria for health, for reasons that became evident in the earlier examination of the WHO definition of health, make health and disease claims a function of semantically subjective statements about ontologically subjective entities, as defined by (S*) and (O*). Accordingly, with each account of health, just as was the case with the WHO’s conception of health, we can expect to find values and/or norms playing an active role in these two distinct ways.

Firstly, we can expect to find (S*) subjectivity. That is, we can expect the truth or falsity of the judgement that “ p_1, \dots, p_n is a capacity or resource requisite for health” to be a function of the accepted values or norms of an individual, a group, a society, a culture, etc., and hence is, semantically speaking, subjective. Recall the two parameters of (S*) subjectivity and that there are at least two levels—a first-order and a second-order—in which objectivity and subjectivity may be at play. Since this was spelled out in some detail in Chapters 1 and 2, I hope that it is by now clear that the health judgments of both of the above accounts of health will report a semantically subjective fact of the matter to the effect that, for example, the properties p_1, \dots, p_n or the conditions C_1, \dots, C_n are the criteria requisite for health.

Let me merely say here that those concerned to identify and challenge questionable values or norms would do well to remain keenly aware of the way in which the health judgements of both the above accounts are being made true or false by *a particular criteria* which states whatever amount of “a capacity” or “a resource” is counted to be requisite to “pursue or acquire x ” (e.g., goals, education, and so on) in virtue of which an individual (or population) is said to be healthy or not.

And, secondly, we can expect to find (O*) subjectivity. That is, we can expect the existence of at least one or more of the properties p_1, \dots, p_n (e.g., education, a goal) to depend on particular human beliefs about p_i or attitudes to p_i , and/or to depend on the acceptance of one or more cultural norms that do not exist in the world independent of human attitudes and feelings. Note, for instance, the role that the above accounts of health give to conceptions of “one’s/our goals”, “education”, “skills”, “to grow”, “personal aspirations”, “life’s challenges”—these “health needs” are not the kinds of facts that exist in the world independent of human beliefs, attitudes, feelings and values.⁴² Hence each one of these conceptions will crucially depend on ontologically subjective properties or norms (by O*).

In sum, there is an important lesson for those of us concerned to safeguard our health judgments against illicit values and norms. Whenever health judgements are, explicitly or otherwise, dependent on (S*) and (O*) subjectivity there is legitimate danger of it being the case that different values and norms are accepted and in force, despite—and perhaps against—the accepted values or norms of any one individual or group. A responsible population health approach would thus do well to look closely into the two distinctly different ways (i.e., (S*) and (O*) subjectivity) in which values or norms *must* be playing an active role in any health judgements that are a function of either of the above two accounts of health. If the above accounts of health are a function of illicit values or norms, they are to be found in (S*) and (O*) subjectivity.

⁴² Here I am, of course, assuming that the requisite conceptions of “goals”, “skills” and “grow” go beyond the biological goals of survival and reproduction—a surely uncontroversial assumption given the stated goals of the Canadian population health approach.

3.4 *A concluding challenge*

In my view, the best conception of health and disease will essentially be a function of value- or norm-dependent *subjectivity* in the sense defined by (O*) and/or (S*). I am thus aligning myself with the normativist side of the modern debate over health and disease.

If my arguments are convincing, then there are substantial normativist challenges facing Canadian health research, policy, and practice—challenges that I believe have not received the attention they deserve. For instance, we need to become much clearer on what values and norms—as well as whose—ought (and ought not) to play a fundamental rôle in responsible health and disease judgements. Most importantly, we need to identify, to weed out, and to do our best not to allow illicit values and norms to take root again. These challenges are related and, if they are to be effectively confronted, then they must be tackled simultaneously.

To be sure, the challenges before us are daunting and will not be easy to deal with; but no reasonable person should expect otherwise. Equally clear is that these challenges are not just the theoretical fare of philosophers and health theorists. All Canadians have a vested interest here, although they may not express it or recognize it. Regardless of whether one appreciates it or not, how we understand health and disease carries profound consequences—medical, legal, ethical, political, philosophical, economic, environmental, and social—that impact *every* Canadian. This means that we need to ensure that our health and disease claims are not being driven by clandestine values and norms skewed to the ideology and interests of only a minority of Canadians.

To circle the wagons, I have tried to show that as it stands one doesn't always know whose interests are being served—or ignored—when it comes to current health and

disease judgements. I have argued that there is an underlying tension between realizing the goals of healthy people and healthy communities and realizing the goals of for-profit companies and shareholders, psychiatric associations, and governments. In my view, a substantial problem is the lack of clarity and precision of the accounts of health and disease purporting to underpin Canadian health research, policy, and practice. That different—and opposing—values and norms so easily reside and flourish within vague and ambiguous accounts of health underscores this point.

This is a point that cannot be overstated. When we allow our conception of health and disease to be a function of ambiguous value- or norm-dependent criteria we leave our health and disease judgements open to be skewed to the ideology and interests of a select few powerful and influential stakeholders. Our brief examination of two notable accounts of health purporting to guide Canadian population health should, it seems to me, serve to show that there *is* a pressing need to acquire a much better grasp of the values or norms that *are*—wittingly or unwittingly—driving notable health and disease judgements.

To that end, I hope to have laid the groundwork for the sort of framework that may equip philosophers, health researchers, health educators, health practitioners, political theorists, health activists, health lobbyists, and others to better recognize the distinct and different ways in which individuals, groups, societies, and/or cultures may reasonably be said to depend—explicitly or implicitly, wittingly or unwittingly—on values and norms to justify and sustain health and disease judgements. Perhaps with further enhancement this framework may even provide the impetus for better health outcomes.

Undermining the ability to achieve many (probably most) of Canada's stipulated health objectives is the reluctance of key stakeholders to accept and confront the

challenges of normativism. Though I am confident that the majority of individuals tasked with stewarding the health of Canadians are well-intentioned, I am not blind to the fact that, in effect, they are mostly toothless. The inability to translate knowledge into action has not gone unnoticed by some; the Senate of Canada aptly describes our dismal practical impact:

Sadly, the great majority of those fine policy statements that have been produced by the federal, provincial and territorial governments since the Lalonde report, 30 years ago, to foster greater emphasis on the non-medical determinants of health, population health, and health disparities remain little more than well-meaning but empty rhetoric. Canadians deserve better!⁴³

Yet, less than five months in the wake of this Senate of Canada report, the Canadian Public Health Association (CPHA) affirmed that “Canada can be proud of its overall health achievements”.⁴⁴ Such pride is especially disturbing because, after reporting that, of 30 countries surveyed, no country spends more money on health care to achieve worse results than Canada, the Senate of Canada pulls no punches in stating “[t]hese sobering numbers tell us we are doing something terribly wrong regarding health and the health care delivery system”.⁴⁵ Simply put, we can no longer afford to mask this reality.

3.4.1 Constrained normativism

In my view, if we are truly concerned to achieve better health results, then we must act to secure a minimal standard of health. And if we are truly concerned not to advance health claims that are scientifically dubious and ideologically tendentious, then we must act to

⁴³ Senate of Canada (2008b), *Population health policy: Federal, provincial and territorial perspectives*. Ottawa: Author, at p. 91.

⁴⁴ Canadian Public Health Association (2008), *Canadian Public Health Association response to the World Health Organization (WHO) Commission’s report*. Ottawa: Author, at p. 5.

⁴⁵ Senate of Canada (2008b), *op cit.*, at p. 91.

operationalize a conception of health and disease that is, as much as possible without being open to Cambridge-change objections, value- and norm-independent.

To put the matter starkly, a normativist conception of health—involving as it does an assertion, sometimes explicit but other times without discussion, of values or norms—is fraught with dangers. Those of us concerned to insulate our health judgments from illicit values and norms would do well to seize upon an operational conception of health that is equipped (as much as is possible) to clearly and precisely explicate the minimal standards requisite for health.⁴⁶ Not only would this improve upon the failings of the above vague accounts of health, but it may also equip health initiatives with the needed teeth to achieve better health results.

To illustrate, consider the fact that the Senate Subcommittee on Population Health, (echoing a view purportedly held by many powerful and influential stakeholders, including most countries) has recently explicitly stated that “[h]ealth is a fundamental human need and, therefore, a basic human right”.⁴⁷ Presumably, if this is a right that every Canadian is in some sense entitled to as a matter of justice, then insofar as our social policy choices perpetuate inequalities and inequities in health they could be considered to be unjust and,

⁴⁶ It is important to be mindful that health, it does seem, is a paronymous concept with complex extensions that will not be defined by a crisp set of necessary and sufficient conditions. Nevertheless, the fact that there is a plurality of (opposing) values among different populations underscores the need to take very seriously Aristotle’s call in the *Nicomachean Ethics* “to look for precision in each class of things just so far as the nature of the subject admits”. Aristotle (trans., 1984), in J. Barnes (Ed.), *The complete works of Aristotle: The revised Oxford translation*. (Vols. 1-2). Princeton: Princeton University Press, at p. 1730. Here, I also note the common standard of using Bekker numbers to reference Aristotle: *Nicomachean Ethics* Book I: 1094b24-25. Interestingly, many of our contemporary views about health (e.g., the idea that health is a necessity for a flourishing life, the tendency to identify health narrowly in terms of bodily excellence, or much more widely with well-being) are to be found in the writings of Aristotle. See, for instance, *Politics* Book VII: 1330a38-b17 and *Physics* Book VII: 246b5.

⁴⁷ Senate of Canada (2008a), *op cit.*, p. 10.

as such, warrant change.

But as it stands what exactly does this basic human right entail? Does it entail living in better conditions than Vancouver's Downtown East Side? Or is it not being exposed to the significant risk of unsafe drinking water, as 75 percent of First Nation communities across Canada are?⁴⁸ Making sense of what it means to say that health is a basic human right requires justifying a minimal standard of health. Unless and until this is done, it is difficult both to accurately assess how many individuals and populations are "being wronged" and to effectively parlay the claim that health is a human right into positive change.

This alone provides compelling reason to secure and operationalize a minimal standard of health that is a function of (S*) and (O*) objectivity, to the extent that a satisfactory conception of health and disease allows.

To that end, I would like to conclude this dissertation with an outline, in very broad strokes, of what I envision the most promising conception of health and disease to be. I will sketch the sort of conception of disease I have in mind first, with a conception of health to follow.

(I) A constrained normativist conception of disease

It seems to me that we ought to operationalize a conception of disease that is *ontologically*

⁴⁸ Randy Christensen (2006), *Waterproof 2: Canada's drinking water report card*. Vancouver: Sierra Legal Defence Fund, p. 30. Christensen recently reports that as of April 30th, 2010, there were 116 First Nations communities across Canada under a Drinking Water Advisory for risk of waterborne contaminants. He notes that, between 2003 and 2007, the mean average duration of a Drinking Water Advisory in First Nations communities was 343 days, with the longest in place for close to 13 years. See, Randy Christensen (2010), *Seeking water justice: Strengthening legal protection for Canada's drinking water*. Vancouver: Ecojustice, p. 10.

objective (as defined by O*) and *semantically subjective* (as defined by S*).

Recall that to say that some organism is diseased on this account is to say something like the following: That, on one hand, the organism possesses properties p_1, \dots, p_n where that the organism possesses each property p_i is a *brute* fact, but, on the other hand, that the truth or falsity of this judgement (i.e., that *these* particular properties p_1, \dots, p_n are requisite for disease) is a function of at least *two* parameters: (i) there exists in the society in which health is being ascribed some *social construction*, a construction which specifies certain conditions C_1, \dots, C_n for what is to count as a *diseased* state and (ii) the particular organism said to be diseased satisfies C_1, \dots, C_n in virtue of possessing properties p_1, \dots, p_n .

Note an important point from Chapter 2: There are at least two levels—a first-order and second-order—with distinct and different issues in which objectivity and subjectivity may be at play. Hence, the second-order insistence that the properties p_1, \dots, p_n and/or the conditions C_1, \dots, C_n are the criteria for D^1 is subjective by (S*) is perfectly compatible with the first-order insistence that an individual A^i has the disease D^1 is objective by (S*). Following the “local” sense of the subjective/objective distinction proposed in Chapter 2 (Section 3.2), the sort of semantic subjectivity I have in mind here may be further clarified to be *local-objectivist*. That is, the criteria specified to be requisite for disease is adopted as something to be counted as a diseased-state *for all* relevantly similar individuals.

Now what I want to propose is that we take this sort of a conception of disease and inform it with the leading naturalist conception of dysfunction. I am sympathetic to Boorse’s conception but I see no good reason why Wakefield’s view of “dysfunction”, as discussed in Chapter 2, would not work equally as well here. As does Boorse (and others), recall that I am using “disease” as a technical definition that is meant to cover the

multifarious conditions (e.g., cancers, physiological injuries, mental disorders, genetic disorders, traumas, and so on) that medical science counts to be inconsistent with perfect health.

There is, to my mind, a clear and obvious three-prong upshot to this sort of conception of disease. It may become an important and effective bulwark against (i) improper medicalization; and, what inevitably follows, (ii) over-prescription of “treatments”—many of which are harmful; and (iii) “disease” becoming a social engineering control of a population’s behaviour. For when we insist that the content of our disease judgements does not depend on any properties, facts, states, and so on the existence of which is dependent of the beliefs, attitudes or feelings of any perceiver about the properties we are in effect demanding that an individual is diseased if and only if there is a genuine physiological (which includes psychological) dysfunction verifiable and falsifiable by medical science. Operationalizing a conception of disease that requires the presence of demonstrable somatic pathology, would have significant and, in my opinion, welcome implications for health and disease judgments, in general, but for mental health judgements, in particular.

It is worth mentioning that Boorse in effect acknowledges a need to introduce value- or norm-dependent criteria with what he calls a “disease-plus” concept, which he says is “disease plus extra criteria of severity or disvalue”.⁴⁹ According to Boorse:

[The BST] provides a theoretical, value-free concept of disease or pathological condition. But on this foundation one can build value-laden disease concepts, by adding evaluative criteria, to taste. Starting from the basic disease concept...one can use different "disease-plus" concepts for

⁴⁹ Boorse (1997), *op cit.*, p. 55.

different purposes. Yet the value-free scientific concept of disease remains a bedrock requirement to block the subversion of medicine by political rhetoric or normative eccentricity.⁵⁰

I am sympathetic with Boorse's disease-plus concepts; but I disagree with him on where the evaluative criteria (read: subjectivity as defined by (O* or (S*)) will come in. I have argued that a value- and norm-independent conception of disease fatally succumbs to the Cambridge-change objections. If my arguments have been convincing, then a satisfactory conception of disease must itself be a function of value- or norm-dependent criteria. Hence, Boorse is mistaken to think that a value-free concept of disease remains a bedrock on which we create disease-plus concepts by “adding evaluative criteria, to taste”—a satisfactory conception of disease is much trickier than that.

(II) A constrained normativist conception of health

In this dissertation, we have come across several notable accounts of health that our framework reveals to be are ontologically and semantically subjective as defined by (O*) and (S*). Though I am convinced that an adequate conception of health must be a function of (S*) subjectivity, I am less confident that (O*) subjectivity is required. Yet, given multifarious Canadian health research, practice, and policy initiatives, I am sympathetic to the view that a health initiative may be better served by a conception of health that is to some extent a function of (O*) subjectivity. At this point I see a population/public health approach being such a candidate. Hence, I'm inclined to think that different health initiatives may be better served by operationalizing different

⁵⁰ Ibid., p. 100.

conceptions of health. But even if different conceptions of health are legitimate, our task should be to operationalize a single *minimal standard of health*.

In doing so, why not aim to advance a minimal standard of health that is *ontologically objective* (as defined by O*) and *semantically subjective* (as defined by S*)? A minimal standard falls short of the sort of conception of health that I believe is needed to guide health research, policy, and practice, but it does provide an important first step towards securing a more comprehensive conception. In effect, such a minimal standard of health, could serve as the basis upon which further normativist elements may be added as necessary. This process, involving as it does a benchmark minimal standard of health, would surely make the identification of any extraneous values and norms driving health judgments much easier. When coupled with the sort of conception of disease I have been arguing for, I envision an (O*) objective and (S*) subjective minimal standard of health becoming a very important bulwark against the corruption of health judgments by illicit values and norms.

Further still, the sort of minimal standard I am proposing could provide some much needed teeth to better achieve positive health outcomes. Importantly, it provides a tangible benchmark with which stewards of health may insist that an individual or group is demonstrably not healthy, where the first-order insistence is objective by (S*).

Obviously there is much more that could be said, but, as I stated from the outset my aim here is limited. Using broad strokes, it is merely to outline what I take to be most promising conception of health and disease. The next step requires that we specify which properties are requisite for health and disease. This is a matter that I must defer to medical science and to others more sensitive to the task.

But in doing so, we must be careful here. I have argued that which properties are counted to be requisite will depend on which values or norms win the day and are in force to specify what is to count as a *healthy* and *diseased* state. It is here that a commitment to normativism forces us into a dangerous sea of tendentious and clandestine values and norms.

Though no one denies that empirical science must inform our conception of health and disease, the view that science *ipso facto* debunks ideology is hardly unanimous. And, what is more, there seems to be growing contempt for science percolating within a small, but increasingly influential segment of Canadian society. In the end this means, whenever science and ideology clash (and it will), we must act to ensure that medical science is allowed to prevail—and *a fortiori* that our health judgements do not run against the leading and ever-growing scientific knowledge of the day!

This requires having mechanisms in place with the capacity to have any (O*) and (S*) subjectivity our health judgements may depend on, in some important sense, constrained by medical science, and not the other way around. Perhaps the conception of health and disease this dissertation has been arguing for may prove to be such a mechanism.

This dissertation has argued for a conception of health and disease that will, in some important way, be a function of ontological and/or semantic *subjectivity* in the sense defined by (O*) and (S*). But it will also be a conception that is a function of (S*) and (O*) objectivity, to the extent that empirical science legitimately furnishes without, of course, being open to Cambridge-change objections. That is to say, I think Canadian health research, policy, and practice should be guided by a normativist conception of health and

disease, but one that is, as much as possible, *constrained* by naturalism. In short, I am calling for a constrained normativist conception of health and disease.

It will be no easy task to weed out and not allow illicit values and norms to take root again. Importantly, we should appreciate and take advantage of the fact that any satisfactory conception of health and disease we employ *is* essentially tied to human values or norms. If, for instance, we find ourselves employing a conception of health that does little more than express the entrenched interests of a select few, then we ought to take issue with the values and norms that essentially buttress such a conception. Accordingly, I submit that Canadian health research, policy, and practice should immediately aim to secure a minimal standard of health that can legitimately aim to reduce health disparities, especially those rooted in discrimination and oppression.

To be sure, there will be those—many of which will be powerful and influential stakeholders—that will be resistant and in some cases outright hostile against operationalizing the sort of constrained normativist conception of health and disease this dissertation argues for. Cui bono?

References

- Abraham, Carolyn. (2010). "Failing boys: Part 3: Are we medicating a disorder or treating boyhood as a disease?", *Globe and Mail*, 18 Oct. 2010.
- Allen, Colin, Marc Bekoff, & George Lauder. (Eds.), (1998). *Nature's purposes: Analysis of function and design in biology*. Cambridge, Mass: The MIT Press.
- American Psychiatric Association *DSM-5: The future of psychiatric diagnosis*.
<<http://www.dsm5.org/Pages/Default.aspx>> Accessed July 4, 2011.
- Amundson, Ron. (2000). "Against normal function", *Studies in the History and Philosophy of Biological and Biomedical Sciences* 31(1): 33-53.
- Amundson, Ron, & George V. Lauder. (1994). "Function without purpose: The uses of causal role function in evolutionary biology", *Biology and Philosophy* 9(4): 443-469.
- Ananth, Mahesh. (2008). *In defense of an evolutionary concept of health*. Aldershot: Ashgate Publishing Co.
- Angell, Marcia. (2011a). "The epidemic of mental illness: Why?", *The New York Review*, June 23, 2011. <<http://www.nybooks.com/articles/archives/2011/jun/23/epidemic-mental-illness-why/>> Accessed July 4, 2011
- (2011b). "The illusions of psychiatry", *The New York Review*, July 14, 2011.
<<http://www.nybooks.com/articles/archives/2011/jul/14/illusions-of-psychiatry/?page=1>> Accessed July 22, 2011.
- Anscombe, G. E. M. (1958). "On brute facts", *Analysis* 18(3): 69-72.
- Ariew, André, Robert Cummins, & Mark Perlman (Eds.), (2002), *Functions: New essays in the philosophy of psychology and biology*. Oxford: Oxford University Press.
- Arioglu, Elif, Nicole A. Gottlieb, Chroastian A. Koch, John. L. Doppman, *et al.*, (2000), "Natural history of a proinsulin-secreting insulinoma: From symptomatic hypoglycemia to clinical diabetes", *The Journal of Clinical Endocrinology & Metabolism* 85(10): 3628-3630.
- Aristotle. (trans., 1984). in J. Barnes (Ed.), *The complete works of Aristotle: The revised Oxford translation*. (Vols. 1-2). Princeton: Princeton University Press.
- Associated Press. "India's health minister calls homosexuality a 'disease'" July 5, 2011
<<http://www.cbc.ca/news/world/story/2011/07/05/india-gay-slur.html>> Accessed July 5, 2011.

- Ayers, Michael. (1982). "Locke versus Aristotle on natural kinds", *The Journal of Philosophy* 78(5): 247-272.
- Bechtel, William. (1985). "In defense of a naturalistic concept of health", in J. Humber & R. Almeder (Eds.), *Biomedical ethics reviews*. Clifton, NJ: Humana, 131-170.
- Bigelow, John & Robert Pargetter. (1987). "Functions", *The Journal of Philosophy* 84(4): 181-196.
- Bird, Alexander, & Emma Tobin. (2009). "Natural kinds", in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy* <<http://plato.stanford.edu/archives/spr2009/entries/natural-kinds/>> Accessed July 4, 2011.
- Blute, Marion. (2008). "Is it time for an updated 'Eco-Evo-Devo' definition of evolution by natural selection?", *Spontaneous Generations: A Journal for the History and Philosophy of Science* 2(1): 1-5.
- Boorse, Christopher. (1975). "On the distinction between disease and illness", *Philosophy and Public Affairs* 5(1): 49-68.
- (1976a). "What a theory of mental health should be", *Journal for the Theory of Social Behaviour* 6: 61-84.
- (1976b). "Wright on functions", *The Philosophical Review* 85(1): 70-86.
- (1977). "Health as a theoretical concept", *Philosophy of Science* 44: 542-573.
- (1987). "Concepts of health", in & T. Regan D. Van De Veer (Eds.), *Health care ethics: An introduction*. Philadelphia: Temple University Press, 359-393.
- (1997). "A rebuttal on health", in R. F. Almeder J. M. Humber (ed.), *What is disease?*, Towata, New Jersey: Humana Press, 3-134.
- (2002). "A rebuttal on functions", in Andrew Ariew, Robert Cummins & Mark Perlman (Eds.), *Functions: New essays in the philosophy of psychology and biology*. Oxford: Oxford University Press, 63-112.
- (2010). "Disability and medical theory", in D. Christopher Ralston & Justin Ho (Eds.), *Philosophical reflections on disability*. Dordrecht: Springer, 55-88.
- Boyd, Richard. (1999). "Homeostasis, species and higher taxa", in Robert A. Wilson (Ed.), *Species: New interdisciplinary essays*. London: MIT Press, 141-186.
- Brink, David O. (2001). "Legal interpretation, objectivity, and morality", in Brian Leiter (Ed.), *Objectivity in law and morals*. New York: Cambridge University Press, 12-65.

- Brock, Dan W. (1993). *Life and death*. Cambridge: Cambridge University Press.
- Brown, Ian. "Where is its mind? What the battle over the 'bible' says about psychiatry", *Globe and Mail*, 09 July, 2011 <<http://www.theglobeandmail.com/life/health/new-health/conditions/addiction/mental-health/where-is-its-mind-what-the-battle-over-the-bible-says-about-psychiatry/article2091844/page1/>> Accessed July 10, 2011.
- Brown, W. Miller. (1985). "On defining disease", *Journal of Medicine and Philosophy* 10(4): 311–328.
- Buchanan, Allen, Dan W. Brock, Norman Daniels, & Daniel Winkler. (2001). *From chance to choice: Genetics and justice*. Cambridge: Cambridge University Press.
- Buller, David J. (1998). "Etiological theories of function: A geographical survey", *Biology and Philosophy* 13: 505-527.
- Buss, David M., Martie G. Haselton, Todd K. Shackelford, April L. Bleske, & Jerome C. Wakefield. (1998). "Adaptations, exaptations, and spandrels", *American Psychologist* 53(5): 533-548.
- Campaign 2000. (2010). *2010 report card on child and family poverty in Canada: 1989-2010*. <<http://www.campaign2000.ca/reportcards.html>> Accessed July 4, 2011.
- Canadian Cancer Society's Steering Committee. (2009). *Canadian cancer statistics 2009*. Toronto: Canadian Cancer Society.
- Canadian Institute for Health Information. (2004). *Improving the health of Canadians*. Ottawa: Author.
- Canadian Mental Health Association. "Statistics" <http://www.cmha.ca/bins/site_page.asp?cid=284-285-1258-1404&lang=1> Accessed July 4, 2011.
- Canadian Public Health Association. (2008). *Canadian Public Health Association response to the World Health Organization (WHO) Commission's report*. Ottawa: Author.
- Canguilhem, Georges. (1978). *On the normal and the pathological*. Dordrecht, Holland: D. Reidel Publishing Co.
- Cannon, Walter B. (1926). "Physiological regulation of normal states: some tentative postulates concerning biological homeostatics", in, *Jubilee volume to Charles Richet*, Paris Editions Medicales.
- (1929). "Organization for physiological homeostasis", *Physiological Reviews* 9(3): 399-431.

- Caplan, Arthur L. (1993). "The concepts of health, illness and disease", in W. F. Bynum and R. Porter (Eds.), *Companion encyclopedia of the history of medicine*. London: Routledge, 233-248.
- Carretero, Oscar A., & Suzanne Oparil. (2000). "Essential hypertension: Part I: Definition and etiology", *Circulation* 101: 329-335.
- Christensen, Randy. (2006). *Waterproof 2: Canada's drinking water report card*. Vancouver: Sierra Legal Defence Fund.
- (2010). *Seeking water justice: Strengthening legal protection for Canada's drinking water*. Vancouver: Ecojustice.
- Clarke, Michael F. & Margaret Fuller. (2006). "Stem cells and cancer: two faces of Eve", *Cell* 124: 1111–1115.
- Conrad, Peter. (1992). "Medicalization and social control", *Annual Review of Sociology* 18: 209-232.
- (2007). *Medicalization of society: On the transformation of human conditions into treatable disorders*. Baltimore: The Johns Hopkins University Press.
- Cooper, Rachel. (2002). "Disease", *Studies in History and Philosophy of Biological and Biomedical Sciences* 33(2): 263-282.
- (2005). *Classifying madness: A philosophical examination of the diagnostic and statistical manual of mental disorders*. Dordrecht: Springer.
- Cooper, Steven J. (2008). "From Claude Bernard to Walter Cannon: Emergence of the concept of homeostasis", *Appetite* 51: 419–427.
- Crimmins, Mark. (1998). "Semantics", in E. Craig (Ed.), *Routledge Encyclopedia of Philosophy*. <<http://www.rep.routledge.com/article/U036>> Accessed July 4, 2011.
- Cropper, William H. (2001). *Great physicists: The life and times of leading physicists from Galileo to Hawking*. New York: Oxford University Press, Inc.
- Cummins, Robert. (1975). "Functional analysis", *The Journal of Philosophy* 72(20): 741-765.
- D'Amico, Robert. (1995). "Is disease a natural kind?", *The Journal of Medicine and Philosophy* 20: 551-569.
- (2007). "Disease and the concept of supervenience", in Harold Kincaid and Jennifer McKittrick (Eds.), *Establishing medical reality*. Dordrecht: Springer, 35-45.

- Dalerba, Piero, Robert W. Cho, & Michael F. Clarke. (2007). "Cancer stem cells: Models and concepts", *Annual Review of Medicine* 58: 267-384.
- Daniels, Norman. (2008). *Just health: Meeting health needs fairly*. Cambridge: Cambridge University Press.
- Daniels, Norman, Bruce Kennedy, & Ichiro Kawachi. (2000). *Is inequality bad for our health?* Boston, Mass: Beacon Press.
- Darwall, Stephen. (2001), "Normativity", in E. Craig (Ed.), *Routledge Encyclopedia of Philosophy*. <<http://www.rep.routledge.com/article/L135SECT6>> Accessed July 4, 2011.
- Darwin, Charles. (1872). *The origin of species*. 6th ed. London: John Murray.
- (1874). *The descent of man and selection in relation to sex*. 2nd ed. Chicago: Rand, McNally Co.
- Davis, Phillip V. & John G. Bradley. (1996). "The meaning of normal", *Perspectives in Biology and Medicine* 40(1): 68-77.
- Dawkins, Richard. (1986). *The blind watchmaker: Why the evidence of evolution reveals a universe without design*. New York: W. W. Norton & Co.
- (2009). *The greatest show on earth*. New York: Free Press.
- Dennett, Daniel C. (1995). *Darwin's dangerous idea*. New York: Simon & Schuster.
- Devitt, Michael. (2008). "Resurrecting biological essentialism", *Philosophy of Science* 75: 344-382.
- Diamond, Jared. (2007). "The double puzzle of diabetes", *Nature* 423: 599-602.
- Dragulinescu, Stefan. (2010). "Diseases as natural kinds", *Theoretical Medicine and Bioethics* 31(5): 247-369.
- Duke, Richard C., David M. Ojcius, & John Ding-E Young. (1996). "Cell suicide in health and disease", *Scientific American* 275(6): 80-87.
- Dunn, J. Michael. (1990). "Relevant predication 2: Intrinsic properties and internal relations", *Philosophical Studies* 60(3): 177-206.
- Dupré, John. (1995). "Wilkerson on natural kinds", *Philosophy* 64: 248-251.

- Dworkin, Ronald. (1977). *Taking rights seriously*. Cambridge, Mass: Harvard University Press.
- Elder, Crawford. (1994). "Higher and lower essential natures", *American Philosophical Quarterly* 31(3): 255-265.
- Ellis, Brian. (2001). *Scientific essentialism*. Cambridge: Cambridge University Press.
- Engelhardt, Jr., H. Tristram. (1976). "Ideology and etiology", *Journal of Medicine and Philosophy* 1(3): 256-268.
- (1986). *The foundations of bioethics*. New York: Oxford University Press.
- Ereshefsky, Marc. (2009a). "Defining 'health' and 'disease'", *Studies in the History and Philosophy of Biological and Biomedical Sciences* 40(3): 221-227.
- (2009b). "Natural kinds in biology", in E. Craig (ed.), *Routledge Encyclopedia of Philosophy*. <<http://www.rep.routledge.com/article/Q124SECT1>> Accessed July 4, 2011.
- (2010). "What's Wrong with the New Biological Essentialism", *Philosophy of Science*.
- Feinberg, Andrew P. (2007). "Phenotypic plasticity and the epigenetics of human disease", *Nature* 447: 433-440.
- Flam, Faye. (1994). "Hints of a language in junk DNA", *Science* 226: 1320
- Flint, J., A. V. S. Hill, D. K. Bowden, & S. J. Oppenheimer, et al. (1986). "High frequencies of α -thalassaemia are the result of natural selection by malaria", *Nature* 321: 744-750.
- Franchetti, Mark .(2007). "Putin brings back mental ward torment", *The Sunday Times*, August 26, 2007.
- Friedlander, Robert M. (2003). "Apoptosis and caspases in neurodegenerative diseases", *The New England Journal of Medicine* 348: 1365-1375.
- Fulford, K. W. M. (1989). *Moral theory and medical practice*. Cambridge: Cambridge University Press.
- Garten, Samuel, & R. Victor Falkner. (2003). "Continual smoking of mentholated cigarettes may mask the early warning symptoms of respiratory disease", *Preventive Medicine* 37: 291-296.
- Geach, Peter .(1969). *God and the soul*. London: Routledge & Kegan Paul.

- (1972). *Logic matters*. Berkeley: University of California Press.
- Gee, Alastair. (2007). "Russian dissident 'forcibly detained in mental hospital'", *The Independent*, July 30, 2007. <<http://www.independent.co.uk/news/world/europe/russian-dissident-forcibly-detained-in-mental-hospital-459539.html>> Accessed July 4, 2011.
- Gilbert, Scott F., & David Epel. (2009). *Ecological Developmental Biology: Integrating Epigenetics, Medicine, and Evolution*. Sunderland: Sinauer Associates.
- GlaxoSmithKline. *GSK Annual Review 2000: It's about you*. <<http://www.gsk.com/investors/annual-reports-archive.htm>> Accessed July 4, 2011.
- . *GSK Annual Review 2004: New challenges, new thinking*. <<http://www.gsk.com/investors/reports/ar2004/annual-review-04/business.htm>> Accessed July 4, 2011
- . *GSK Annual Review 2008*. <<http://www.gsk.com/investors/annual-reports-archive.htm>> Accessed July 4, 2011
- . *GlaxoSmithKline legal update, July 15 2010*. <http://www.gsk.com/media/pressreleases/2010/2010_pressrelease_10076.htm> Accessed July 4, 2011
- Godfrey-Smith, Peter. (1994). "A modern history theory of functions", *Noûs* 28(3): 344-362.
- Goldman, Lee, & Dennis Ausiello. (Eds.), (2007). *Cecil textbook of medicine*. 23rd ed. Philadelphia: Saunders.
- Gould, Stephen Jay. (1997). "The exaptive excellence of spandrels as a term and prototype", *Proceedings of the National Academy of Sciences* 94: 10750-10755.
- Gould, S. J., & R. C. Lewontin. (1979). "The spandrels of San Marco and the Panglossian paradigm: A critique of the adaptationist programme", *Proceedings of the Royal Society of London. Series B, Biological Sciences* 205(1161): 581-598.
- Gould, Stephen Jay, & Elisabeth S. Vrba. (1982). "Exaptation—A missing term in the science of form", *Paleobiology* 8(1): 4-15.
- Greaves, Mel. (2001). *Cancer: The evolutionary legacy*. Oxford: Oxford University Press.
- . (2010). "Cancer stem cells: Back to Darwin?", *Seminars in Cancer Biology* 20(2): 65-70.
- Griffin, S. M., & S. A. Raimes (1998), "Proton pump inhibitors may mask early gastric cancer", *British Medical Journal* 317:1606–1607.

- Griffiths, Paul E. (1993). "Functional analysis and proper functions", *The British Journal for the Philosophy of Science* 44: 409-422.
- (1999). "Squaring the circle: Natural kinds with historical essences", in Robert A. Wilson (Ed.), *Species: New interdisciplinary essays*. London: MIT Press, 209–228.
- (2002). "What is innateness?", *Monist* 85: 70–85.
- Guerrero, J. David. (2010a). "The essential value(s) of health: Implications for Canadian population health research and policy", in T. McIntosh, B. Jeffery & N. Muhajarine. (Eds.), *Redistributing health: New directions in population health research in Canada*. Regina: CPRC Press, 3-19.
- (2010b). "On a naturalist theory of health: A critique", *Studies in the History and Philosophy of Biological and Biomedical Sciences* 41(3): 272-278.
- Haack, Susan. (2004). "Point of honor: On science and religion", *Skeptical Inquirer* 28(2): 56–62.
- Hacking, Ian. (1991). "A tradition of natural kinds", *Philosophical Studies* 61: 109-126.
- Hamed, Muhammad Fuad, Graeme E. Hole, & Zoe Muir (2006), "A mysterious case of normalising blood sugar: insulinoma in a long-standing diabetic patient", *Age and Ageing* 35: 317–318
- Hardcastle, Valerie Gray. (1999). "Understanding functions: A pragmatic approach", in V. G. Hardcastle (Ed.), *Where biology meets psychology: Philosophical essays*, Cambridge, Mass: The MIT Press, 27-43.
- (2002). "On the normativity of functions", in A. Ariew, R. Cummins and M. Perlman (Eds.), *Functions: New essays in the philosophy of psychology and biology*. Oxford: Oxford University Press, 144-156.
- Health Canada. (1998). *Taking action on population health: A position paper for Health Promotion and Programs Branch staff*. Ottawa: Author.
- (2001). *The population health template: Key elements and actions that define a population health approach*. Ottawa: Author.
- (2008). *Healthy Canadians—A federal report on comparable health indicators 2008*. Ottawa: Author.
- (2009). *A statistical profile on the health of First Nations in Canada—Determinants of health, 1999 to 2003*. Ottawa: Author. <<http://www.hc-sc.gc.ca/fniah-spnia/pubs/aborig-autoch/index-eng.php>> Accessed July 4, 2011.

- Health Council of Canada. (2005). *The health status of Canada's First Nations, Metis and Inuit Peoples*. Toronto: Author.
- Hempel, Carl G. (1965). "The Logic of functional analysis", in *Aspects of scientific Explanation and other essays in the philosophy of science*. New York: The Free Press, 297-330.
- (1966). *Philosophy of natural science*. Englewood Cliffs, N.J.: Prentice-Hall, Inc.
- Higgs, D.R., M.A. Vickers, A.O. Wilkie, I.M. Pretorius, et al., (1989). "A review of the molecular genetics of the human alpha-globin gene cluster", *Blood: The Journal of the American Society of Hematology* 73(5): 1081-1104.
- Hill, Adrian V.S. (1992). "Malaria resistance genes: a natural selection", *Transactions of the Royal Society of Tropical Medicine and Hygiene* 86: 225-226, 232.
- Hirst, Martin, & Marco A. Marra. (2009). "Epigenetics and human disease", *The International Journal of Biochemistry & Cell Biology* 41: 136–146.
- Hofmann, Bjørn. (2001). "Complexity of the concept of disease as shown through the rival theoretical frameworks ", *Theoretical Medicine* 21: 211–236.
- Horowitz, Allan V. (2010). "Pharmaceuticals and the medicalization of social life", in Donald W. Light (Ed.), *The risks of prescription drugs*. New York: Columbia University Press, 92-115.
- Hotchkiss, Richard S., Andreas Strasser, Jonathan E. McDunn, & Paul E. Swanson. (2009). "Cell death", *The New England Journal of Medicine* 361: 1570-1583.
- Howlett, Karen, Greg Keenan, & Shawn McCarthy. (2008). "Auto bailout tab pegged at \$25-billion", *Globe & Mail*, 17 Dec. 2008 B1. *Canadian Periodicals Index Quarterly*. Web. Accessed July 4, 2011.
- Humberstone, I. L. (1996). "Intrinsic/Extrinsic", *Synthese* 108: 205-267.
- Hume, David. (1739). *A treatise of human nature*. New York: Prometheus Books.
- Jemal, Ahmedin, Rebecca Siegel, Rebecca, Elizabeth Ward, et al. (2009). "Cancer statistics, 2009", *CA: A Cancer Journal for Clinicians* 59: 225-249.
- Joint National Committee. (2003). "The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure", *Journal of the American Medical Association* 289(19): 2560-2571.

- Jordon, Craig T. (2009). "Cancer stem cells: controversial or just misunderstood?", *Cell Stem Cell* 4: 203-205.
- Kass, Leon R. (1981). "Regarding the end of medicine and the pursuit of health", in A. L. Caplan, H. T. Engelhardt, Jr., & J. J. McCartney. (Eds.), *Concepts of health and disease: Interdisciplinary perspectives*. Reading, Mass: Addison-Wesley, 3-30.
- Kendell, R. E. (1975). "The concept of disease and its implications for psychiatry", *British Journal of Psychiatry* 127: 305–315.
- Khushf, George. (1997). "Why bioethics needs the philosophy of medicine: Some implications of reflection on concepts of health and disease", *Theoretical Medicine* 18: 145-163.
- (2001). "What is at issue in the debate about concepts of health" in Lennart Nordenfelt (Ed.), *Health, science and ordinary language*. Amsterdam: Rodopi Press, 123-169.
- (2007). "An agenda for future debate on concepts of health and disease", *Medicine, Health Care and Philosophy* 10: 19-27.
- King, Daley C. (1945), "The meaning of normal", *Yale Journal of Biology and Medicine* 17: 493-501.
- King, Lester S. (1981). "What is disease?", in A. L. Caplan, H. T. Engelhardt, Jr., & J. J. McCartney (Eds.), *Concepts of health and disease: Interdisciplinary perspectives*. Reading, Mass: Addison-Wesley, 107-118.
- Kingma, Elselijn. (2007). "What is it to be healthy?", *Analysis* 67: 128–133.
- (2010). "Paracetamol, poison, and polio: Why Boorse's account of function fails to distinguish health and disease", *British Journal for the Philosophy of Science* 61(2): 241–264.
- Kitcher, Philip. (1993). "Function and design", *Midwest Studies in Philosophy* 18: 379–397.
- Kovács, József. (1998). "The concept of health and disease", *Medicine, Health Care and Philosophy* 1(1): 31-39.
- Kripke, Saul. (1980). *Naming and necessity*. Oxford: Blackwell.
- Krohs, Ulrich. (2009). "Functions as based on a concept of general design", *Synthese* 166: 69-89.

- Lauder, George V. (1996). "Argument from design", in Michael R. Rose & George V. Lauder (Eds.), *Adaptation*. San Diego: Academic Press, 55-91.
- Lee, Soyoung I., Russell J. Schachar, Shirley X. Chen, et al. (2008). "Predictive validity of DSM-IV and ICD-10 criteria for ADHD and hyperkinetic disorder", *Journal of Child Psychology and Psychiatry* 49(1): 70-78.
- Leiter, Brian. (2001). "Introduction", in Brian Leiter (Ed.), *Objectivity in law and morals*. New York: Cambridge University Press.
- (2007). *Naturalizing jurisprudence: Essays on American legal realism and naturalism in legal philosophy*. Oxford: Oxford University Press.
- Lewens, Tim. (2004). *Organisms and artifacts: Design in nature and elsewhere*. Cambridge: The MIT Press.
- Lobo, Neethan A., Yohei Shimono, Dalong Qian, & Michael F. Clarke. (2007). "The biology of cancer stem cells", *Annual Review of Cell and Developmental Biology* 23(675-699).
- Locke, John. (1690). *An essay concerning human understanding*. Kitchener: Batoche Books, 2001.
- Mackie, J. L. (1977). *Ethics: Inventing right and wrong*. Harmondsworth, England: Penguin Books.
- Mameli, Matteo. (2005). "The inheritance of features", *Biology and Philosophy* 20: 365-399.
- Margolis, Joseph. (1976). "The concept of disease", *Journal of Medicine and Philosophy* 1(3): 238-255.
- Marmor, Andrei. (2001). *Positive law and objective values*. Oxford: Oxford University Press.
- Marmot, Michael. (2006). "Health in an unequal world: social circumstances, biology and disease", *Clinical Medicine* 6(6): 559-572.
- Marx, Karl. (1845). *Theses On Feuerbach*. Marx/Engels Internet Archive. <<http://www.marxists.org/archive/marx/works/1845/theses/theses.htm>> Accessed July 4, 2011.
- Mayo Foundation for Medical Education. <<http://www.mayoclinic.com/print/type-1-diabetes/DS00329/DSECTION=all&METHOD=print>> Accessed July 4, 2011.
- Mayr, Ernst. (1976). *Evolution and the diversity of life: Selected essays*. fifth printing ed. Cambridge, Mass.: Harvard University Press.

- McCall, John W., Claudio Genchi, Laura H. Kramer, Jorge Guerrero, & Luigi Venco. (2000). "Heartworm disease in animals and humans", *Advances in Parasitology* 66: 193-285.
- McGinn, Colin. (1991). *The problem of consciousness*. Oxford: Blackwell.
- McLaughlin, Peter. (2001). *What functions explain: Functional explanation and self-reproducing systems*. Cambridge: Cambridge University Press.
- Miller, Greg. (2010). "Beyond DSM: Seeking a brain-based classification of mental illness", *Science* 327: 1437.
- Millikan, Ruth Garrett. (1984). *Language, thought, and other biological categories: New foundations for realism*. Cambridge, Mass: MIT Press.
- Mordacci, Roberto, & Richard Sobel. (1998). "Health: A comprehensive concept", *Hastings Center Report* 28(1): 34-37.
- Muilenburg, Diego J., Jodi M. Coates, Subbulakshmi Virudachalam, & Richard J. Bold. (2010). "Targeting Bcl-2-mediated cell death as a novel therapy in pancreatic cancer", *Journal of Surgical Research* 163(2): 276-281.
- Muller, G. B. (2002). "Vestigial organs and structures", in Mark Pagel (ed.), *Encyclopedia of evolution*. New York: Oxford University Press, 1131-1133.
- Murphy, Dominic. (2006). *Psychiatry in the scientific image*. Cambridge, Mass: The MIT Press.
- (2009). "Concepts of disease and health", in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. <<http://plato.stanford.edu/archives/sum2009/entries/health-disease/>> Accessed July 4, 2011.
- Murphy, Edmond A. (1973). "The normal", *American Journal of Epidemiology* 98(6): 403-411.
- Nagel, Ernst. (1977). "Teleology revisited", *The Journal of Philosophy* 74: 261-301.
- National Institute of Mental Health. *The numbers count: Mental disorders in America* <<http://www.nimh.nih.gov/health/publications/the-numbers-count-mental-disorders-in-america/index.shtml>> Accessed July 4, 2011.
- National Institute of Neurological Disorders and Stroke. *NINDS Tay-Sachs Disease Information Page* <<http://www.ninds.nih.gov/disorders/taysachs/taysachs.htm>> Accessed July 4, 2011.

- National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC). (2008). *Medullary sponge kidney* <<http://kidney.niddk.nih.gov/kudiseases/pubs/medullaryspongekidney>> Accessed July 4, 2011.
- Neander, Karen. (1991). "Functions as selected effects: The conceptual analyst's defense", *Philosophy of Science* 58(2): 168-184.
- Neel, James V. (1962). "Diabetes Mellitus: A "thrifty" genotype rendered detrimental by "progress"?", *The American Journal of Human Genetics* 14: 353–362.
- (1999). "The "thrifty genotype" in 1998", *Nutrition Reviews* 75: S2-S9 75: S2-S9.
- Nesse, Randolph M. (2001). "On the difficulty of defining a disease: A Darwinian perspective", *Medicine, Health Care and Philosophy* 4: 37-46.
- Nesse, Randolph M., & George C. Williams. (1996). *Why we get sick: The new science of Darwinian medicine*. 1st ed. New York: Vintage Books.
- Nordenfelt, Lennart. (2001). *Health, science and ordinary language*. Amsterdam: Rodopi Press.
- (2004). "The logic of health concepts", in George Khushf (Ed.), *The handbook of bioethics*. Dordrecht: Kluwer Academic Publishers, 205-222.
- Nowell-Smith, Patrick H. (1954). *Ethics*. Harmondsworth, England: Penguin Books.
- Okasha, Samir. (2002). "Darwinian metaphysics: Species and the question of essentialism", *Synthese* 131(2): 191-213.
- Organisation for Economic Co-operation and Development (OECD). <http://www.oecd.org/pages/0,3417,en_36734052_36734103_1_1_1_1_1,00.html> Accessed July 4, 2011.
- Perlman, Mark. (2004). "The modern philosophical resurrection of teleology", *The Monist* 87(1): 3-51.
- Perlman, Robert L. (2005). "Why disease persists: An evolutionary nosology", *Medicine, Health Care and Philosophy* 8: 343-350.
- Pettit, Philip. (2001). "Embracing objectivity in ethics", in Brian Leiter (Ed.), *Objectivity in law and morals*. New York: Cambridge University Press, 234-286.
- Plantinga, Alvin. (1993), *Warrant and proper function*. New York: Oxford University Press.
- Pörn, Ingmar. (1993). "Health and adaptedness", *Theoretical Medicine* 14: 295-303.

- Progeria Research Foundation. "Progeria 101/FAQ" <www.progeriaresearch.org/progeria_101.html> Accessed June 4, 2011.
- Public Health Agency of Canada. *What is the population health approach?* <http://www.phac-aspc.gc.ca/ph-sp/approach-approche/index-eng.php#def_health> Accessed July 4, 2011.
- Purushotham, A. D., & R. Sullivan. (2010). "Darwin, medicine and cancer", *Annals of Oncology* 21: 199–203.
- Putnam, Hilary. (1973). "Meaning and reference", *The Journal of Philosophy* 70: 699-711.
- (1975). *Mind, language and reality: Philosophical papers, volume 2*. Cambridge: Cambridge University Press.
- (2002). *The collapse of the fact/value dichotomy and other essays*. Cambridge, MA: Harvard University Press.
- Quetelet, Adolphe. (1842). *A treatise on man and the development of his faculties*. New York: Burt Franklin, Reprinted in 1968.
- Quine, W. V., & J. S. Ullian. (1978). *The web of belief*. 2nd ed. New York: Random House.
- Raz, Joseph. (1980). *The concept of a legal system*. 2nd ed. Oxford: Clarendon Press.
- (2002). *Engaging reason: On the theory of value and action*. Oxford: Oxford University Press.
- Reid, Jasper (2002), "Natural kind essentialism", *Australasian Journal of Philosophy* 80(1): 62-74.
- Reznek, Lawrie. (1987). *The Nature of disease*. London: Routledge and Kegan Paul.
- (1995). "Dis-ease about kinds: A reply to D'Amico ", *The Journal of Medicine and Philosophy* 20: 571-584
- Richman, Kenneth A. (2004). *Ethics and the metaphysics of medicine*. Cambridge, MA: MIT Press.
- Robertson, Teresa. (2008). "Essential vs. accidental properties", in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy* <<http://plato.stanford.edu/archives/fall2008/entries/essential-accidental/>> Accessed July 4, 2011.

- Rodríguez-Trelles, Francisco, Rosa Tarío, & Francisco J. Ayala. (2005). "Is ectopic expression caused by deregulatory mutations or due to gene-regulation leaks with evolutionary potential?", *BioEssays* 27: 592–601.
- Rupinder, Sodhi K., Aulakh K. Gurpreet, & Singh Manjeet. (2007). "Cell suicide and caspases", *Vascular Pharmacology* 46: 383–393.
- Scadding, J. G. (1967). "Diagnosis: The clinician and the computer", *The Lancet* 2: 877-882.
- Scadding, S. R. (1981). "Do 'vestigial organs' provide evidence for evolution?", *Evolutionary Theory* 5: 173-176.
- Schiefsky, Mark J. (2005). *Hippocrates on ancient medicine, translated with introduction and commentary by Mark J Schiefsky*. Leiden: Brill Academic Publishers.
- Schramme, Thomas. (2007), "A qualified defence of a naturalist theory of health", *Medicine, Health Care and Philosophy* 10: 11-17.
- (2010). "Can we define mental disorder by using the criterion of mental dysfunction?", *Theoretical Medicine and Bioethics* 31: 35-47.
- Schwartz, Peter H. (1999). "Proper function and recent selection", *Philosophy of Science* 66: S210-S222.
- (2007). "Decision and discovery in defining 'disease'", in Harold Kincaid & Jennifer McKittrick (Eds.), *Establishing medical reality*. Dordrecht: Springer, 47-63.
- (2008). "Risk and disease", *Perspectives in Biology and Medicine* 51(3): 320-334.
- Searle, John R. (1995). *The construction of social reality*. New York: The Free Press.
- (2010). *Making the social world: The structure of human civilization*. New York: Oxford University Press.
- Senate of Canada. (2008a). *Population health policy: Issues and options, Fourth report of the Senate Subcommittee on Population Health*. Ottawa: Author.
- (2008b). *Population health policy: Federal, provincial and territorial perspectives*. Ottawa: Author.
- (2009). *A healthy, productive Canada: A determinant of health approach. The standing senate committee on social affairs, science and technology: Final report of subcommittee on population health*. Ottawa: Author.

- Shackleton, Mark, Elsa Quintana, Eric R. Fearon, & Sean J. Morrison. (2009). "Heterogeneity in cancer: Cancer stem cells versus clonal evolution", *Cell* 138(5): 822-829.
- Sharma, Arya M. (1998). "The thrifty-genotype hypothesis and its implications for the study of complex genetic disorders in man", *Journal of Molecular Medicine* 76(8): 568-571.
- Sherwood, Lauralee. (2010). *Human physiology: From cells to systems, 7th Edition*. Belmont, CA: Brooks/Cole.
- Singh, Ilina. (2008). "Beyond polemics: The science and ethics of ADHD", *Nature Reviews Neuroscience* 9: 957-964.
- Smylie, Janet, & Paul Adomako, (Eds.), (2009). *Indigenous children's health report: Health assessment in action*. Toronto: The Centre for Research on Inner City Health.
- Sober, Elliott. (1980). "Evolution, population thinking and essentialism", *Philosophy of Science* 47: 350-383.
- (2010). "Natural selection, causality, and laws: What Fodor and Piatelli-Palmarini got wrong", *Philosophy of Science* 77: 594-607.
- Sommerhoff, Gerd. (1950). *Analytical biology*. London: Oxford University Press.
- Stark, Andrew. (2006). *The limits of medicine*. New York, NY: Cambridge University Press.
- Statistics Canada. (2008). *Earnings and incomes of Canadians over the past quarter century, 2006 Census*. Ottawa: Statistics Canada.
- Stempsey, William E (1999). *Disease and diagnosis: Value-dependent realism*. Boston: Kluwer Academic.
- (2000). "A pathological view of disease", *Theoretical Medicine* 21: 321-330.
- Stone, Irwin. (1966). "On the genetic etiology of scurvy", *Acta Geneticae Medicae et Gemellologiae*, 345-350.
- Sulmasy, Daniel P. (2005). "Disease and natural kinds", *Theoretical Medicine and Bioethics* 26: 487-513
- Sutherland, Jessica E., & Max Costa. (2003). "Epigenetics and the environment", *Annals of the New York Academy of Sciences* 983: 151-160.

- Szasz, Thomas S. (1960). "The myth of mental illness", in A. L. Caplan, J. J. McCartney. & D. A. Sisti. (Eds.), (2004). *Health, disease and illness*. Washington, D.C.: Georgetown University Press, 43-50.
- (1973). *The second sin*. Garden City, New York: Anchor Press.
- (1997). "Mental illness is still a myth", *Review of Existential Psychology and Psychiatry* 23(1/2/3): 70-80.
- (2009). "The shame of medicine: The case of Alan Turing", *The Freeman* 59(4) <<http://www.thefreemanonline.org/columns/thetherapeutic-state/the-shame-of-medicine-the-case-of-alan-turing/>> Accessed July 4, 2011.
- Thagard, Paul. (1998). "The concept of disease: Structure and change", in Philip Van Looke (Ed.), *Nature of concepts: Evolution, structure and representation*. London: Routledge, 215-242.
- U.S. Census Bureau. *International Data Base, December 2010 Update*. <<http://www.census.gov/ipc/www/idb/worldpopgraph.php>> Accessed July 4, 2011.
- Vácha, Jirí. (1978). "Biology and the problem of normality", *Scientia* 72: 823-846.
- (1985). "German Constitutional Doctrine in the 1920s and 1930s and pitfalls of the contemporary conception of normality in biology and medicine", *Journal of Medicine and Philosophy* 10(4): 339-367.
- Vendantam, Shankar. (2001). "Drug ads hyping anxiety make some uneasy", *Washington Post*, July 16, 2001, A01.
- Wachbroit, Robert. (1994). "Normality as a biological concept", *Philosophy of Science* 61: 579-591.
- Wakefield, Jerome C. (1992). "The concept of mental disorder: On the boundary between biological facts and social values", *American Psychologist* 47(3): 373-388.
- (1999). "Mental disorder as a black box essentialist concept", *Journal of Abnormal Psychology* 108(3): 465-472.
- (2005). "Biological function and dysfunction", in David M. Buss (Ed.), *The handbook of evolutionary psychology*. Hoboken, New Jersey: John Wiley & Sons, Inc., 878-902.
- (2007). "The concept of mental disorder: diagnostic implications of the harmful dysfunction analysis", *World Psychiatry* 6: 149-156.

- Walsh, Denis. (2008). "A commentary on Blute's 'updated definition'", *Spontaneous Generations: A Journal for the History and Philosophy of Science* 2(1): 6-10.
- Walsh, Dennis M. & André Ariew. (1996). "A taxonomy of functions", *Canadian Journal of Philosophy*, 26(4): 493–514.
- Watson, James D., Tania A. Baker, Stephen P. Bell, et al. (2008). *Molecular biology of the gene*. 6th ed. San Francisco: Pearson/Benjamin Cummings.
- Watters, Ethan. (2010). *Crazy like us: The globalization of the American psyche*. New York: Free Press.
- Weatherson, Brian. (2008). "Intrinsic vs. extrinsic Properties", in Edward N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy* <<http://plato.stanford.edu/archives/fall2008/entries/intrinsic-extrinsic/>> Accessed July 4, 2011.
- Wilkerson, T. E. (1988). "Natural kinds", *Philosophy* 63: 29-42.
- (1998). "Recent work on natural kinds", *Philosophical Books* 39(4): 225-233.
- Wilson, Robert A., Mathew J. Barker, & Ingo Brigandt. (2007). "When traditional essentialism fails: Biological natural kinds", *Philosophical Topics* 35(1-2): 189-215.
- World Health Organization. (1946). *Constitution of the World Health Organization*. Geneva: Author.
- (1986). *Ottawa Charter for Health Promotion*. Geneva: Author.
<www.who.int/hpr/NPH/docs/ottawa_charter_hp.pdf> Accessed July 4, 2011.
- (2004). *ICD-10: International statistical classification of diseases and related health problems: tenth revision*. (2nd Ed.), Geneva: Author.
- (2008a). *The world health report 2008: Primary care now more than ever*. Geneva: Author.
- (2008b). *Closing the gap in a generation: health equity through action on the social determinants of health. Final report of the commission on social determinants of health*. Geneva: Author.
- (2009). *Official ICD-10 WHO updates combined 1996-2008, Volume 3*. Geneva: Author. <<http://www.who.int/classifications/icd/icd10updates/en/index.html>> Accessed July 4, 2011.
- . "Cancer" <<http://www.who.int/tobacco/research/cancer/en/>> Accessed July 4, 2011.

- Wouters, Arno. (2005). "The function debate in philosophy", *Acta Biotheoretica* 53(2): 123-151.
- Wright, H. G. (2007). *Means, ends and medical care*. Vol. 92, *Philosophy and Medicine*. Dordrecht, The Netherlands: Springer.
- Wright, Larry .(1973). "Functions", *The Philosophical Review* 82(2): 139-168.
- Yu, Catherine H.Y., & Bernard Zinman. (2007). "Type 2 diabetes and impaired glucose tolerance in aboriginal populations: A global perspective", *Diabetes Research and Clinical Practice* 78: 159–170.
- Zahid, Alyia. (2004). "The vermiform appendix: Not a useless organ", *Journal of the College of Physicians and Surgeons* 14: 256-258.

Appendix A

Proposed *DSM-V* revision for “Oppositional Defiant Disorder”

- A. A persistent pattern of angry and irritable mood along with defiant and vindictive behavior as evidenced by four (or more) of the following symptoms being displayed with one or more persons other than siblings.

Angry/Irritable Mood

1. Loses temper
2. Is touchy or easily annoyed by others.
3. Is angry and resentful

Defiant/Headstrong Behavior

4. Argues with adults
5. Actively defies or refuses to comply with adults’ request or rules
6. Deliberately annoys people
7. Blames others for his or her mistakes or misbehavior

Vindictiveness

8. Has been spiteful or vindictive at least twice within the past six months
- B. (NOTE: UNDER CONSIDERATION) The persistence and frequency of these behaviors should be used to distinguish a behavior that is within normal limits from a behavior that is symptomatic to determine if they should be considered a symptom of the disorder. For children under 5 years of age, the behavior must occur on most days for a period of at least six months unless otherwise noted (see symptom #8). For individuals 5 years or older, the behavior must occur at least once per week for at least six months, unless otherwise noted (see symptom #8). While these frequency criteria provide a minimal level of frequency to define symptoms, other factors should also be considered such as whether the frequency and intensity of the behaviors are non-normative given the person’s developmental level, gender, and culture.
- C. The disturbance in behavior causes clinically significant impairment in social, educational, or vocational activities.
- D. The behaviors may be confined to only one setting or in more severe cases present in multiple settings.¹

¹ American Psychiatric Association. “Q 00 Oppositional Defiant Disorder” <<http://www.dsm5.org/ProposedRevision/Pages/proposedrevision.aspx?rid=106#>> Accessed July 2, 2011.