This essay replies to critics since 1995 of my “biostatistical theory” (BST) of health. According to the BST, a pathological condition is a state of statistically species-subnormal biological part-functional ability, relative to sex and age. Theoretical health, the total absence of pathological conditions, is then a value-free scientific notion. Recent critics offer a mixture of old and new objections to this analysis. Some new ones relate to choice of reference class, situation-specificity of function, common diseases and healthy populations, improvements in population health, the practice of pathologists, “Cambridge changes” in health status, and comparative vs. absolute health concepts. I make no changes in doctrine, except to consider treating “normal aging” as pathological by taking young adults as the standard for all adults.

Keywords: biostatistical theory, disease, disorder, dysfunction, health, pathology, philosophy of medicine

I. INTRODUCTION

My unified naturalist analysis of concepts of health and function, first offered about forty years ago (Boorse, 1975, 1976a, 1976b, 1977) and slightly altered thereafter (Boorse, 1987, 1997), still evokes vigorous debate. My previous rebuttal on health (Boorse, 1997) answered nearly all critics through 1995. This paper replies to later ones through 2013, with, I hope, similar breadth. We can best begin with a summary of the analysis and some needed clarifications.1

II. SUMMARY AND CLARIFICATION OF THE BST

The target of my basic health paper (Boorse, 1977) was scientific medicine’s concept of theoretical health as normality, i.e., the total absence of
pathological conditions. Originally, I used “disease” in the broadest possible sense for departures from health, but eventually realized that “pathological” is a more natural choice. My analysis of pathology relied, in turn, on an account of the concept of biological function (Boorse, 1976a). The fundamental idea is that a pathological condition is a state of statistically species-subnormal biological part-function (Boorse, 1997, 4), relative to sex and age.

Here is a slightly corrected summary from 1977:

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 [or] reproduction.
3. 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.
4. A disease [later, pathological condition] is a type of internal state which impairs health, i.e., reduces one or more functional abilities below typical efficiency (Boorse, 1977, 562).

Three comments on this definition may be helpful. First is a structural point: the analyses of health and of function are separable (Boorse, 1997, 10–11). Other accounts of biological function could be substituted for my goal-contribution view if they gave better results—e.g., a selectionist one like Neander’s (1991a, 1991b) or Wakefield’s (1999b). Second, clause 3 presupposes an explication of “with at least typical efficiency” as “at efficiency levels within or above some chosen central region of their population distribution” (Boorse, 1977, 558–559)—that is, not far below the statistical mean. Third, I omit a 1977 clause aimed at typical environmental injuries, having decided (Boorse, 1997, 86) that it may not be worth the criticism it evoked.2 But in 1997, to meet the challenge of atypical environmental diseases like heat stroke or mountain sickness, I added the following clause:

[A] statistically species-subnormal function (in the usual sense of an arbitrarily chosen lower tail) is pathological if it results from an environmental factor outside an arbitrarily chosen central statistical range of that factor in the environments where the species lives (Boorse, 1997, 84).

Here “subnormal” means far below the mean of function in a typical environment (not the rare one), and the condition is on environmental factors like heat or air pressure, not whole environments.3

I claim that theoretical health, so analyzed, is value-free, since none of its component ideas requires value judgments. But besides theoretical health, I also recognized a great variety of value-laden “disease-plus” concepts: first illness (Boorse, 1975; later retracted) and then diagnostic normality (absence of a clinically detectable pathological condition; Boorse, 1987, 365) and
therapeutic normality (absence of a diagnostic abnormality worthy of treatment), as well as many social disease-value hybrids such as civil incompetence, criminal insanity (Boorse, 1997, 100), and perhaps disability (Boorse, 2010).

As to clarifications, first, two corrections are due independently to Schwartz and others. In 1987 I pictured the analysis with a bell-like curve, reprinted in 1997. Unfortunately, the axes got mislabeled. In a corrected diagram (Schwartz, 2007, 373), functional efficiency should be the x-axis, frequency (or, in the continuous case, probability density) the y-axis. The second correction is that my phrase “survival and reproduction” would better be “survival or reproduction.” Obviously, reproductive functions, such as pregnancy, need not help survival and may hurt it (Boorse, 1997, 92–94), and people can have diseases beyond their reproductive years. I wrote “and” in the same way as governments advertise rewards for contributions to the “arrest and conviction” of a criminal. Some people help the arrest; others help the conviction. But “or” would have been clearer, so I welcome the change and incorporate it above.4

Very important is that the biostatistical theory (BST) is, and always has been, a dynamic account of normal physiology (Boorse, 1997, 78–79). Some critics imagine it to say that a given function, such as blood circulation, must be performed all the time and at the same level. Rather, Kingma is right (2010, 249) that the BST uses dispositions to function, situation-specificity of function, and quantitative as well as qualitative normality. I was explicit about all three elements, though she adds useful terminology. As to the first two, I required “functional readiness,” not just current function, and noted that “biological functions are usually performed on appropriate occasions, not continuously” (Boorse, 1977, 561–562). As to normal quantitative variation, it is so obvious that I mentioned it only once: “the function of the thyroid is not merely to secrete hormones, but to secrete the right amount of them for current metabolic needs” (Boorse, 1977, 559; italics added). I admit never saying that normal heart rate varies with activity, being lower at rest and higher during exercise. But this is a fact known to everyone and exemplifies a typical feature of physiology. That a concrete process may have to vary to fulfill its function is required by my, or almost anyone’s, analysis of biological function. Blood circulation serves survival by carrying sufficient oxygen and nutrients to body cells and evacuating their wastes. Since oxygen needs rise during exercise, the heart, to fulfill its normal function, must increase its rate. So, except in critics’ imagination, there never was a static BST, only a dynamic one. In particular, normal function varies with both an organism’s activity and its environment.

Let me also clarify a rather confusing paragraph. In my “Rebuttal on functions,” discussing whether evolutionary accounts of function can define normality, I wrote:

Now the most obvious logical feature of medical normality is that most functions have a normal range of values. No one value of heart rate, blood pressure, blood
urea nitrogen, serum glutamic-oxaloacetic transaminase, forearm strength, height, IQ, and so on is uniquely normal. Rather, there is a range of normal variation around a mean, with either one or two pathological tails (Boorse, 2002, 101).

The list in my second sentence is very heterogeneous. Some items on it are true functional capacities (forearm strength, IQ). Others are clinically measurable concrete substances or processes that have functions (SGOT, heart rate, blood pressure), and one (BUN) is a clinical item that has no function but measures the function of something else (the kidneys). I did not mean to imply that all the items are functions. After all, I have repeatedly distinguished a concrete process from its function and stated that a true function can only have one tail (subnormality), not two (Boorse, 1977, 559; Boorse, 1987, 371). I only meant that these variables’ having a normal range implies that their associated functions have a normal range. However, if I had known in 2002 how misleading the term “normal range” is, I would have rewritten this argument for that reason, too.

Let us now consider recent critics, who offer a mixture of old and new objections. I begin with critics who misdescribe the BST in important ways.

III. MISDESCRIPTONS OF THE BST

Bolton

There are two possible interpretations of Bolton’s discussion of the BST in What is Mental Disorder? On the first, he distorts my view beyond recognition by saying that the BST equates medical normality to statistical normality. The main objection to this approach is that mere difference will not do for capturing concepts like disease or dysfunction. .... In my view this is the main problem with Boorse’s analysis and any other attempt to equate dysfunction in health contexts with mere statistical difference (Bolton, 2008, 112).

In summary, then, Boorse’s proposal to construe norms of mental functioning as a fact of nature, in terms of statistical normality, does not work for many reasons. First, because deviance from statistical normality in itself – independent of any problems that may result – does not warrant attribution of pathology, physical or mental (114).

The key point is ... the mistaken equation of difference with dysfunction. ... [Boorse’s analysis] is particularly problematic now in its proposal that mere statistical difference from some population norm constitutes disease or some mental equivalent. It invites the protest from individuals with such conditions – now that they have a voice – that difference is being pathologized and hence disqualified (115).

I have, of course, never said that “mere difference” or “statistical rarity” (Bolton, 2008, 114) is pathological. Just the opposite: in every summary of the BST, I have stated that statistical abnormality alone cannot define pathology. In my original paper, this point occupies a full page (Boorse, 1977, 546–547; see also Boorse, 1987, 369; Boorse, 1997, 7; Boorse, 2011, 21–22).
These statements by Bolton ignore two points. First, for pathology, the BST requires a specific kind of statistical abnormality: substantial subnormality. Subnormality is not “mere difference” or “statistical rarity.” Second, what is pathological by the BST is subnormality of function. Bolton seems to ignore the function element in my definition altogether. He never mentions my analysis of biological functions as species-typical causal contributions to individual survival or reproduction. Perhaps he is misled by the loose usage of “function” in mental-health professions to cover any sort of human activity or output. That is not, of course, biological or medical usage, in which “function” has some content. The now-vast philosophical literature on analyzing biological function statements starts precisely from the observation that “function” means more than “effect” or “activity.” For example, noise is an effect of the heart’s contraction, but not its function.

The worry that Bolton ignores my concept of function persists when he gives a longer and more favorable treatment of Wakefield’s account. He earlier explained the difference as follows:

Boorse’s approach has the natural fact underlying disorder attribution as being a matter of statistical abnormality; Wakefield’s approach invokes the evolutionary design of mental and behavioral functioning (Bolton, 2008).

This seems a strange contrast, given my many references to “species design” (Boorse, 1975, 59–60; Boorse, 1977, 556–557). In any case, how does evolution design traits with functions? By selecting those that causally contribute to survival and reproduction (S & R). Thus, the difference between my view of functional design and Wakefield’s is just this: on my view, a functional trait must serve S & R in the present, while on Wakefield’s, it must have served S & R in the past and been selected for that effect. If this difference bears at all on Bolton’s discussion, it is surely to the BST’s advantage, since on my view no evolutionary evidence is necessary to establish a trait’s function.

It is true that Bolton does sometimes specifically say “below-average” (Bolton, 2008, 112) or “below-normal” (113). So a second interpretation might be that his criticism of the BST is the same as Wakefield’s in this volume: that part-dysfunction, without harm, cannot suffice for medical disorder. Unfortunately, this interpretation violates Bolton’s text. As the above quotes and others show, his repeated complaint is that I equate mere difference in function to disease, pathology, and dysfunction (112–115). As far as I know, it is not medically controversial to call substantially below-average part-function pathological, i.e., to call it dysfunction. Wakefield (this volume) denies neither statement. So, whether or not people with, say, very low IQ (mental retardation) or unusually poor reality testing (psychotics) “have a voice” and “protest” their classification, they are not being “pathologiz[ed] on the grounds of mere difference” (115). There is nothing wrong with pathologizing the pathological. On the contrary, what is wrong is to refuse to pathologize the pathological on political grounds. In any event, if this
second interpretation fails, Bolton’s version of the BST remains a caricature—an absurd view, but not mine.

Murphy and Ananth

Dominic Murphy, in his book (2006) and Stanford Encyclopedia of Philosophy entry (2008), also misdescribes the BST in several ways. He does rightly categorize my view as “objectivist” (his term for naturalism) rather than “constructivist.” But it is not so easy to locate the BST on his other dimension, “conservative” vs. “revisionist” theories. First, his distinction runs together two questions: whether lay intuitions are authoritative about (1) the concept of disease, or (2) what actual conditions fit this concept (Murphy, 2008, 7; Murphy, 2006, 20–21). A theorist could be conservative about the intension of “disease” (1), about its extension (2), about both, or about neither. For the BST, the answer is “neither,” since it aims to analyze a theoretical medical concept of disease in the sense of “pathological condition” (Boorse, 1977, esp. 551; Boorse, 1997, 7), and for this task lay ideas of disease are of little interest (Boorse, 1997, 62–63). I do take medicine as authoritative about the concept’s intension, but only in the sense that we determine the meaning of “pathological” as the best explanation of the body of medical judgments of what is and is not pathological. It can still be true that the best explanation of some specific judgments is error or prejudice, as with masturbation (1997, 72–78). Murphy’s suggestion that I may believe that “commonsense intuitions are the ultimate court of appeal when we are trying to decide what counts as mental disorder” (Murphy, 2006, 45) is mistaken.

Two other errors are as follows. First, Murphy repeatedly states that I have a selectionist analysis of function, like Wakefield (or Neander or Millikan or, for biological functions, Wright). He writes: “Boorse and his followers have tied disease conceptually to an evolutionary concept of function as a naturally selected capacity” (Murphy 2008, 15; see also Murphy, 2006, 35, 37). This is the opposite of the truth. I am one of the earliest and most vocal critics of “etiological” accounts of function in general, and selectionist accounts of biological function in particular (Boorse, 1976a, 2002). Naturally, I believe that the majority, probably the vast majority, of physiological functions arose by natural selection. That merely means that I am not a creationist. This statement is true not just on my analysis of biological function, but on any sensible one. How else could the body’s incredibly intricate functional organization arise? But I have never said that selection is part of the meaning of biological function. As I (Boorse, 1976a, 76) and others have explained, an item can currently make a species-typical contribution to S & R without ever having been selected for that purpose—for example, if it acquires a new function after its initial design. Perhaps Murphy’s mistake is to assume that an analysis of function in terms of current contributions to S & R is an analysis “in evolutionary terms” (Murphy, 2006, 35). But that
is wrong, as the function literature shows. Finally, his distinction between evolutionary and “causal-mechanical” explanations is nearly identical to my distinction between functional and operational explanations (Boorse, 1976a, 75). To a great extent, when Murphy sets out to critique my view, he instead expounds it.

Strangely, even Ananth is led astray by this topic. He writes:

Boorse is caught in a contradiction as a result of his concept of function. ... [H]e wants his concept of function to respect the fact that functions (at least some) are a product of a certain Darwinian history. This point is also clear ... when Boorse claims that he defends a stabilizing selection interpretation of natural selection. Yet, it is just this sort of Wrightian etiological account that he was at pains to reject in his reply to Engelhardt. So, sometimes Boorse agrees that his concept of function includes an etiological/evolutionary dimension, but at other times he is adamant that his concept of function does not include an etiological/evolutionary dimension (Ananth, 2008, 163).

Here Ananth confuses the meaning of a term with what is true of some, or most, or even all of its extension. “At least some” cars are Toyotas, but being a Toyota is no part of the meaning of “car,” and the car concept includes no Toyota dimension. Even if the vast majority of forest fires are caused by lightning, that does not mean that causation by lightning is part of the meaning of “forest fire” or that the forest-fire concept includes a lightning dimension. In fact, this would be true even if human beings had not yet made their first fire in the wilderness, so that all forest fires were due to lightning. The first human-caused forest fire would still be a forest fire. Similarly, even if nearly all physiological functions are the result of evolution and are preserved over long periods by stabilizing selection, that does not imply that anything about selection is part of the concept of function. So I remain adamant.  

Murphy’s third error is less important, but still worth a mention. He describes me, as well as Wakefield, as representatives of the “two-stage picture of the foundations of psychiatry,” which is supposedly a thesis about the concept of “mental disorder” or “mental illness” (Murphy, 2006, 19). The thesis is that these concepts consist of a descriptive component (dysfunction) and a socially constructed normative component (e.g., harm). While this description fits Wakefield, it does not fit me. My basic concept, defined via dysfunction and serving as the foundation of theoretical medicine, is “pathological condition,” which is value-free. In an early paper I said that “illness” was value-laden, meaning a disease serious enough to be incapacitating and therefore having three evaluative features (Boorse, 1975, 61). But I never said that illness could serve as “the foundation of” theoretical medicine; many pathological conditions never cause illness. Soon thereafter, I abandoned this analysis of illness anyway in favor of a descriptive one, systemic disease. I still recognize various “disease-plus” concepts consisting of disease with assorted evaluative features (Boorse, 1997, 100–101). But
none of these is a suitable foundation for medical theory, either. Of course, I do regard medical theory as the scientific foundation of medical practice. But it is hard to see how anyone could fail to be a normativist about concepts of practice; there is no dispute about that. Finally, I do not treat value judgments as socially constructed, since I do not accept a cultural-relativist metaethics.

Miscellaneous Writers

A related misstatement by several writers is that the BST is a theory of the limits of proper medical practice. Venkatapuram writes:

[Boorse] assumed, like so many others still do, that clinical medicine/healthcare and human health are mutually encompassing ideas. If the concept of health is defined, then the scope and purpose of medicine becomes defined; if the scope of medicine is defined, then health becomes defined, a supposedly perfect mutuality (Venkatapuram, 2011, 45).

Again, this is the opposite of my view. Obviously, health can be promoted by nonmedical means, such as sanitation, purification of air and water, and other public-health measures. Conversely, I have always stressed that many accepted medical treatments, such as contraception and cosmetic surgery, do not aim at improving health. Such treatments are one reason Richman and Budson (2000, 350), too, are wrong to assume my view to be that only disease is properly treated. They also seem to think I hold that diseases which should be treated are always illnesses. I am not sure that I thought that even in 1975, and, if I did, I was confused. A skin injury, dental cavity, and dislocated shoulder are treatable pathological conditions, but not illnesses by my later account, since they are not systemic.

Some other miscellaneous mistakes about the BST are as follows. Venkatapuram (2011, 46–47), perhaps misled by my confusing 2002 paragraph, charges me with “conflating” a clinical variable such as heart rate with the heart’s functional efficiency. He seems to believe that if we have no specific “efficiency test,” my bell-curve–like figure (Boorse, 1987, 370; Boorse, 1997, 8) makes no sense. I do assume functional efficiency to be measurable. For many homeostatic functions, we already know how to measure it and could easily construct a histogram. For example, if the function of the temperature-regulating system is to keep core body temperature near 98.6°F, then inefficiency is just the size of the swings away from that value. As for the bell-like curve, there is no reason why such a distribution could not be roughly normal. I do not see why it is any less plausible to assume that efficiency of a process, properly measured, would be Gaussian than to assume that the process itself would. In any case, my analysis, contrary to Giroux, does not assume anything to be Gaussian. My 1977 paper did not even contain the bell-curve picture, which I added later for ease of
comprehension. Its description of normality of a part’s function was merely this: “at efficiency levels within or above some chosen central region of [its] population distribution” (Boorse, 1977, 559).

Two final points about Cooper: first, I have never been inconsistent (Cooper, 2005, 13) about whether the BST covers mental disorders. My 1977 essay explained why it ignored them (Boorse, 1977, 543). To settle the controversy over their existence, one must first know what a disorder is, and so must analyze the disease concept in somatic medicine. As a footnote says (543 n. 1), my (1975) and (1976b) apply the resulting analysis to this issue. Although I have yet to return to the topic, I do not doubt the existence of mental disorders in exactly the BST’s sense. Second, I did not “claim” (Cooper, 2005, 18) that homosexuality is pathological. I merely suggested it might well be (Boorse, 1975, 63), and that this judgment need not be disturbing. But if, for example, any of the kin-selection hypotheses were correct, by the BST homosexuals would be as normal as worker bees. They would merely have a variant method of reproduction. I fully accept inclusive fitness (Garson and Piccinini, 2014) as a biological goal.

From now on, I shall group replies under the same headings as in my previous rebuttal on health (Boorse, 1997), but adding one at the end.

IV. TECHNICAL OBJECTIONS

DeVito, Ereshefsky, Kingma

Several writers continue to charge that the BST is a value-laden analysis of health and disease because, to begin with, the choice of some of its elements rests on value judgments. Reviving a criticism by Engellhardt (1976, 263–66), DeVito (2000) and Ereshefsky (2009) argue that the BST rests on an evaluative choice of S & R as human goals. Similarly, Kingma argues that my choice of reference class is “an evaluative choice” (Kingma, 2007, 132). After quoting some of these arguments, I will explain once again why they seem to be formally defective and so cannot establish their conclusion (Boorse, 1997, 25–26).

As to goals, Ereshefsky writes:

There is a more fundamental problem with Boorse’s claim that biological fitness is the biological goal of humans and all organisms. Biologists describe many types of states that organisms have, and many of those states have nothing to do with fitness. There is eating for eating’s sake. There is non-reproductive sex. There is the release of endorphins. Biology describes various states organisms can be in, and one type of state happens to concern fitness. Biology does not tell us that surviving and reproducing, versus achieving other kinds of states, are the goals of organisms. That choice comes from outside of biology. By choosing fitness as the goal of organisms, Boorse violates a main tenet of naturalism – that biology and biology alone should tell us what is ‘health.’ (Ereshefsky, 2009, 223)
DeVito says that the BST is value-laden in two distinct ways, the first being the same as Ereshefsky's.

[W]hen there are multiple sets of criteria that adequately divide the world into things to which the concept applies and things to which it does not (in essence, when there are multiple conceptions of the same thing), we can choose one of these concepts as the “correct” or accepted concept. Values can enter at this choice-level. The second place where values can gain entry is at the level of the criteria themselves. Boorse’s concepts of health and disease are value-laden at both levels (DeVito, 2000, 541–2).

The reason the BST is, for DeVito, value-laden at the choice level is, as with Ereshefsky, its choice of the goals of physiology. First, physiologists, like all scientists, choose what to study for evaluative reasons. Second, medicine could use physiological knowledge, yet have different goals and interests from physiologists, and so define health and disease differently (DeVito, 2000, 542). DeVito also claims that the BST’s criterion of health is itself value-laden—his second level. That is because it refers to life and reproduction, because life and reproduction are goods, and because “any concept that is based on a criterion that refers to goods is a value-laden concept” (544). But either this second point reduces to the first, or it is false. Life and reproduction are (sometimes) good for human beings, but that does not mean that they are evaluative concepts. Even DeVito concedes that “there are value-free facts about life-span and reproductive ability” (544), as there could not be if life and reproduction were good by definition. The definition of many generally good things is value-free. Usually wealth, social power, and physical strength and endurance are good for people. But that does not mean that “Warren Buffett is a billionaire” or “Allyson Felix can run 100 meters in under 11 seconds” is a value judgment. So I will ignore the supposed second level of value-ladenness. Either it is the same as the first, or it is mistaken.

Finally, Kingma (2007) rightly says that the BST requires reference classes because normal human functioning varies among groups. But it cannot, she says, justify its actual choice of an age group of a sex of a species. Since it seeks to define disease, it cannot, without circularity, exclude groups like blind people or people with pneumonia on the grounds that such conditions are diseases. It also cannot, she argues, explain how its choice is “a natural class of organisms of uniform functional design” (Boorse, 1977, 562). That is because none of three interpretations of “natural”—“occurring in nature,” “normal,” or “natural kinds”—nor two interpretations of “design”—innateness and natural selection—yield the stated result, nor does “uniformity” (Kingma, 2007, 129–130).

To show that the BST cannot answer the question “Is homosexuality a disease?” at all, let alone in a value-free way, Kingma says:

Imagine that there are two candidate concepts for health. One is the BST, and one is the XST. The XST is exactly like the BST, but has one more reference class: sexual
orientation. Thus on the XST homosexuality is a normal, therefore healthy, function in the reference class of homosexual people. On the BST however, homosexuality interferes with statistically typical reproductive function in the reference class of all men, and is therefore a disease. The question ‘is homosexuality a disease?’ then reduces to the question ‘is the BST or the XST the right account of health?’ or, ‘is sexual orientation an appropriate reference class or not?’ Since, as I have argued, there are no facts that determine which reference classes are appropriate, there is no empirical fact that determines whether homosexuality is an appropriate reference class. Therefore there is no empirical fact that tells us whether the BST or the XST is correct (Kingma, 2007, 132).

I believe that all these arguments, of similar structure, are formally incapable of showing their conclusion: that the BST is a value-laden analysis. A correct definition of concept H in terms of concepts C₁, C₂, ... Cₙ is value-laden precisely if one of the Cᵢ is value-laden: that is, if a judgment of the form “x is Cᵢ” is a value judgment. It does not matter at all how concept H was “chosen,” only what it is (Boorse, 1997, 23–26). The BST analyzes health using the concepts of statistical normality, survival, reproduction, organism, part, process, species, sex, age, and causation. Ereshefsky, DeVito, and Kingma do not allege any of these concepts to be value-laden, except for DeVito’s confusing remarks on life and reproduction as “values.” Nor, of course, are these writers here claiming that the BST ill fits medical judgments of disease.

Rather, their form of argument is as follows. There are several “candidate concepts” of health (Kingma, 2007, 132), or “multiple conceptions of the same thing” (DeVito, 2000, 541–542). Allegedly, my analysis chooses one of them for evaluative reasons. In the first place, that is not so: I try to choose that analysis which best fits medical usage (Boorse, 1977, 551). The medical concept of health that I seek to analyze already exists as a target. “Candidate concepts,” by contrast, exist only in the minds of philosophers. So the only way to run an argument of this type is to claim that medicine—not the BST—has chosen one of many possible health concepts. The basic problem remains: none of these writers gives sense to the idea of a value-based “choice of a health concept” by medicine. The obvious way to do so fails. That is to assume from the outset that “disease” is an evaluative concept, meaning something like “undesirable condition” or “condition deserving medical treatment.” Then medicine will choose what conditions fall under this description—i.e., make value judgments about what conditions are undesirable or need treatment. But, so clarified, the argument has two fatal defects. First, it is circular, since it assumes its conclusion, that “health” is value-laden. Second, it ignores one of the most basic features of medical usage of “disease”: that disease and medically treatable condition do not coincide. As noted, medicine does not call everything it treats a disease, or pathological. Unwanted fertility, unwanted pregnancy, male foreskins, sagging jowls, and small breasts are treated by medicine, yet never counted as pathological.
To see the issue clearly, consider a parallel to Kingma’s BST/XST argument. I offer an analysis of horse-breeders’ concept “mare” as “adult female horse.” I call this analysis the “horse theory” (HT). Suppose Kingma now proposes an alternative candidate mare concept, the MT, as “adult female mammal.” These two concepts differ only in their basic reference class: horses vs. mammals. Now let Lucy be some sow, i.e., an adult female pig. Suppose Kingma argues that horse-breeders’ judgment “Lucy is not a mare” is not an “empirical fact” (Kingma, 2007, 132). After all, to decide whether it is true we need to decide “whether the [HT] or the [MT] is correct” as an analysis of marehood. In fact, Kingma may note, horse-breeders’ choice of the HT “may reflect some deep underlying normative commitments” (132), namely, their great preference for horses over pigs. For example, they choose to raise and race horses rather than killing and eating them, as they would do to pigs. So marehood is a nonempirical, probably value-laden, concept. All this would be nonsense. The HT is correct by the usage of horse-breeders. Other “candidate mare concepts,” including the MT and the PT, are wrong. And, since none of the HT’s component ideas requires a value-judgment for its application, “mare” is a value-free empirical concept. That this or some other group of speakers could have chosen to mean something else by “mare,” such as “giant igneous rock,” is irrelevant.

Thus, I continue to maintain that value-laden choice of a health concept by anyone, me or medicine, is a mythical process. Certainly, it is an evaluative choice by physicians to promote health rather than to destroy it, to cure diseases rather than to cause them. But that is a value-laden choice of what to do about health and disease, not what health and disease are—exactly as with the corresponding choice about life and death. So I hope that we can lay arguments of this type to their final rest.

V. THE BST AS BAD BIOLOGY

While I think the preceding answer (which I gave in Boorse, 1997, 25–26) suffices, I will add some thoughts on the biological naturalness of medicine’s idea of health. Ereshefsky says that for a naturalist, “biology and biology alone should tell us what is ‘health’” (Ereshefsky, 2009, 223). But that is true of the BST, which defines health as normal physiological functional ability. Biology and biology alone does tell us what are biological functions. (If, as some writers argue, there is more than one type of biological function, then the BST seeks to pick whatever type is characteristic of physiologists’ function statements.) As mentioned before, there is a huge literature on analyzing biological function statements. But most views, like mine, take a function to be some sort of contribution to fitness, past or present. Ereshefsky could attack my specific analysis of function, but he does not do so. Rather, he advocates that biologists should eliminate function talk entirely from their
“technical discussions” (226), just as physicians should avoid talk of health and disease. In other words, he seeks not to analyze all these biomedical concepts, but to eliminate them. My aim has always been more modest: to explain key concepts of biology and medicine, not to revolutionize both fields.

As for Ereshefsky’s remarks on goals, there is no reason, on any view of health or biological function, why organisms cannot pursue goals subsidiary to S & R. On the contrary, I have always said they do.

The structure of organisms shows a means-end hierarchy with goal-directedness at every level. Individual cells are goal-directed to manufacturing certain compounds; by doing so they contribute to higher-level goals like muscle contraction; these goals contribute to overt behavior like web-spinning, nest-building, or prey-catching; overt behavior contributes to such goals as individual and species survival and reproduction. What I suggest is that the function of any part or process, for the biologist, is its ultimate contribution to certain goals at the apex of the hierarchy (Boorse, 1977, 556).

But it is only the lower goals’ contribution to fitness by which evolutionary theory can explain the existence of the traits that serve them. Does Ereshefsky think that human hunger, sexual desire, or endorphins have no biological function? Presumably, he believes that they, like virtually the whole human functional design, are the product of evolution. If so, nothing he says about lower-level goals contradicts the BST.

As for choice of reference class, the one that I suggested medicine uses—an age group of a sex of a species or subspecies—could hardly be a more biologically natural choice. Apart from one detail, the BST’s reference class is just one morphological type in the smallest taxon to which an organism belongs. When biologists describe and classify organisms, they sort them into species or subspecies, separate them by sex in sexual species, and distinguish immature from adult forms. The BST departs from this only in making old age as well as youth count for classification, and I suggest below the possibility of no longer doing so. Kingma is free to ask biologists why they classify organisms as they do. Why aren’t blind pigs, or diabetic pigs, or Luton pigs their own taxon? I would imagine that the answer is that while male pigs and female pigs—or pig embryos, piglets, and adult pigs—have distinctive, effective functional designs, that is not true of blind or diabetic vs. normal pigs. Blindness or diabetes is not a special, equally efficient way of functioning. It is a loss of function that other species members have, with no compensating advantage. But since the BST need not answer this question, I will ignore it. It is irrelevant unless there are actual examples of medical disputes about whether something is pathological which turn on choice of reference class. But, to use Kingma’s example, in the 1973 American debate over homosexuality, APA members did not divide into those who opposed and those who defended sexual preference as a basic factor in the
reference class. After all, in medical thought, saying “diabetes is normal for diabetics” does nothing to change its disease status. So a dispute over reference class would go nowhere.

Hamilton and Sober

Let us now consider some other charges that the BST rests on bad biology. Hamilton collects several such criticisms, saying that “Boorse ... fails to comprehend ... just how devastating the ‘bad biology’ arguments are to his case” (Hamilton, 2010, 325). Though I have already explained at length why I find them harmless (Boorse, 1997, 28–41), I will expand on my answer. Hamilton first endorses an argument by Lewontin and Sober that normality is not a biological notion. As this is the most important one, I will treat it fully. The argument is based on the idea of a “norm of reaction.” Sober writes:

All possible phenotypes of a genotype are “natural,” since all are possible. This radically non-Aristotelian idea is codified in the genotype’s norm of reaction.... The norm of reaction shows the different phenotypes a given genotype will produce in different environments. So, for example, we might graph the height that a corn plant with a particular genotype will attain as a function of how much water or nutrition or sunlight it receives. There is no such thing as its “natural” height. We may prefer a taller plant to a shorter one, and natural selection may, too. But the norm of reaction marks no such distinction. ... If each individual had a natural phenotype, these normative judgments could perhaps be grounded in biology. But we must look elsewhere to find a basis for these discriminations ... (Sober, 1984, 160, 161).

Sober’s term is “natural” and Hamilton’s “normal” or “healthy,” but there is no reason to think that Sober would resist the change. On the contrary, as Hamilton notes, in Sober’s original article on this topic, he wrote:

[O]ur current conceptions of function and dysfunction, of disease and health, seem to be based upon the kinds of distinctions recommended by the Natural State Model. And both of these distinctions resist characterization in terms of maximum fitness. For virtually any trait you please, there can be environments in which that trait is selected for, or selected against. Diseases can be rendered advantageous, and health can be made to represent a reproductive cost. (Hamilton, 1980, 325, quoting Sober, 1980, 377; italics added).

Both these last points, of course, I and others have made many times. They are basic features of the disease concept—not somehow, as Sober imagines, objections to it. At any rate, it is easy to see, first, that Sober’s argument20 is fallacious, and, second, that its conclusion is false, refuted every day by both the implicit and explicit standard practice of biologists.

To see that Sober’s argument is fallacious, note its form:

Both states called health and states called disease are possible, since they are within the norm of reaction of a genotype to its environment. Therefore, the distinction between health and disease is not a biological fact.
Now simply substitute “life” and “death” for “health” and “disease.”

Both states called life and states called death are possible, since they are within the norm of reaction of a genotype to its environment. Therefore, the distinction between life and death is not a biological fact.

This substitution is in no way unfair. Sober himself mentions lethal environments, where “a corn plant of a particular genotype withers and dies” of malnutrition (Sober, 1980, 375; Sober, 1994, 223). But the distinction between life and nonlife is not just a biological distinction: it is the distinction on which the entire science of life is based. No one claims that disease or death is impossible. Thus, the only significant content of each premise is that whether each occurs may depend on the environment. But that could not possibly prove that biology cannot draw distinctions among points on the norm of reaction, objectively calling some disease or death. Indeed, Sober concedes in the quoted passage that natural selection draws such distinctions—which are then not “normative,” but as biological as can be.

Moreover, beyond life and death, it is obvious that biology implicitly uses a normality concept in its most basic operation: species description. Here is a quotation from an Oxford encyclopedia of insects:

One of the most generally known and oft-repeated facts about insects is that they possess three pairs of legs.... Indeed, this condition is in the fundamental ground plan of insects... (Resh and Cardé, 2009, 555).

To avoid trivial objections, I will narrow my focus to ants, since a small minority of apodous insect species are legless, as an adaptation to stationary or parasitic life. Certainly, it is a truism to say, with Keller and Gordon (2009, 13), that ants “invariably” have six legs. (There are no apodous ant species.) Hamilton himself quotes Buller—author of several “devastating” objections to the BST—saying something similar: “[All]l primates have two hands, all mammals have lungs, and all vertebrates have two eyes, a heart, a liver, and a stomach” (Buller, 2005, 426). Without an implicit restriction to normal organisms, such statements would be false. One does not have to watch an ant colony very long to see ants lacking one or more legs. If impatient, one can simply create ants to taste, with 5, 4, 3, 2, 1, or 0 legs. These are still ants, indeed live ants. They are merely not intact: they are abnormal, injured ants, ants with increasingly serious leg pathology. All basic biological description of animal and plant species ignores diseased or damaged specimens as irrelevant to the type. The fact that some environments contain little boys and other pathogens is irrelevant.

Furthermore, biologists, as well as philosophers of biology including Sober himself, often use the terminology of health and disease (Boorse, 1997, n 25). Indeed, biologists apply these concepts across the whole spectrum of life. Summaries of evolution or ecology often include generalizations about disease, using that term or related ones like “pathology” or “pathogen.” Here is Maynard Smith:
[Darwin’s] theory of natural selection starts from the observation that in optimal conditions, with unlimited supply of food and space, and in the absence of predators and disease, all animal and plant species are capable of increasing in numbers in each generation (Smith, [1958] 1993, 43; italics added).\textsuperscript{24}

J.B.S. Haldane states:

A study of the causes of death in man, animals, and plants leaves no doubt that one of the principal characters possessing survival value is immunity to disease (Haldane, [1932] 1966, 141; italics added).

George Simpson says:

Each normal individual organism has a life pattern which is characteristic of its species and which it follows without strong deviations, barring accident or pathology (Simpson, 1967, 186; italics added).

And Dawkins writes:

Wild animals almost never die of old age: starvation, disease, or predators catch up with them long before they become really senile (Dawkins, [1976] 1989, 112; italics added).\textsuperscript{25}

Not surprisingly, then, it is easy to find biological talk of normality and pathology for organisms of any kingdom, indeed almost any animal phylum. A single volume (41, 2005) of \textit{Journal of Wildlife Diseases} has articles about bears, boars, coyotes, cheetahs, seals, bats, frogs, turtles, alligators, salamanders, bass, bluejays, magpies, vultures, and many other animals. Study of insect diseases, which goes back at least to Pasteur’s work on silkworms, is chronicled in the \textit{Journal of Insect Pathology}—later renamed \textit{Journal of Invertebrate Pathology}, and now in its 116th volume. Two years of it (90–91, 2005–2006) discuss numerous insects: besides many kinds of moths, flies, and ants, there are articles on diseases of bumblebees, wasps, beetles, mosquitoes, planthoppers, burrower bugs, aphids, and thrips. The same volumes explore diseases of such other invertebrates as crabs, shrimp, snails, cockles, clams, and earthworms. For diseases of trees (elm, poplar, alder, horse chestnut) or other plants (orchid, gladilolus, grass), one can look in any recent volume of \textit{Plant Pathology} or other journals of this field.\textsuperscript{26}

Thus, it is clear beyond dispute\textsuperscript{27} that biology constantly uses a normality, or health, concept implicitly, and often explicitly as well. How could Lewontin and others have come to say otherwise? Lewontin’s political motivation is clear in his text (Lewontin, 1977), and Sober’s too (1984, 161): to discredit, \textit{e.g.}, the idea of natural intelligence. Stephen Jay Gould resorted to statistical deception and shameless fabrication in this noble crusade (Lewis et al., 2011). Lewontin and Sober merely deny the obvious. But untruths convenient to one political goal can prove inconvenient to another. Combine Sober’s position that health is not a biological concept with Kitcher’s view that the alternative to objectivism is social constructivism, and we seem to
reach the position that a clitoris is a genuine disease today in some North African countries. In any case, many other African children, in regions of famine or endemic disease, get to experience the extremes of their norm of reaction. So do children of abusive parents in America. Even here, some 14-year-old girls weigh 110 pounds; others weigh 42 pounds; others are dead. By the lights of Lewontin, Sober, and Hamilton, biology cannot call any of these states more natural, normal, or healthy than any other. That is false. Nothing in biology—evolutionary, populationist, or otherwise—shows that Danieal Kelly was not objectively malnourished, or Khalil Wimes objectively injured, long before they were both objectively dead at their parents’ hands. Least of all does a norm of reaction, the bare fact that environment affects phenotype, do so.

For another of Hamilton’s criticisms—the nonexistence of species design—he relies on Buller (2005). Since I have covered this issue before (Boorse, 1997, 33–38), I will be brief. Hamilton says that biology “undermine[s] [Boorse’s] central notion of a species design as anything other than a theoretical abstraction” (Hamilton, 2010, 325). This criticism is puzzling, since I explicitly called species design an “ideal type” that “abstracts” from individual differences and “may not exactly resemble any species member.” (Boorse, 1977, 557) It is silly to say that “there are numerous differences between individual human beings” (Hamilton, 2010, 326). No one has ever denied that. The point is that biology and medicine consider some such differences normal, others pathological. Hamilton then quotes and summarizes Buller:

As a result, ‘strictly speaking, there is no single human anatomy and physiology possessed by all humans around the world’. In support of this, [Buller] lists conditions such as situs inversus, children born with only one kidney, or with ambiguous genitalia and less dramatically the variation in human blood type. He urges therefore that we abandon ‘the idea that Gray’s Anatomy provides a single “detailed” and “precise” picture of the anatomy and physiology of every human on earth [since this] is plausible only if one ignores known facts about human anatomical and physiological variation’. The same holds, mutatis mutandis, for Boorse’s appeal to uniform design in support of this theoretical definition of health (Hamilton, 2010, 326).

First, Buller’s four specific examples are no threat to the BST. My analysis recognizes normal variants, i.e., variants of equal functional capacity. I myself cited blood type as a normal polymorphism (Boorse, 1977, 558). Situs inversus, without cardiac pathology, is also a normal variant for the same reason. But congenital absence of a kidney and ambiguous genitalia are pathological, both for medicine and for me. So, presumably, are Hamilton’s male bass feminized by Potomac River pollution into producing eggs (Hamilton, 2010, 325). Why would one think that an unusual environment cannot cause disease? In other fish species, however, either sequential or simultaneous hermaphroditism is normal. It all depends on the species.
In the second place, species design is used “for pedagogic purposes” (325) precisely because of its cognitive value. If textbooks of anatomy, physiology, biochemistry, immunology, and pathology were mostly inaccurate guides to real patients, how could they be the foundation of all medical education? Third, Hamilton misses the most basic point. What the BST implies is simply that wherever no definite species design exists, we cannot speak of health and disease. Thus, no examples of design indeterminacy threaten the BST unless medicine does, or should, classify them as healthy or diseased. Neither Hamilton nor Buller offers such an example. Given a real example of design indeterminacy with no clear medical judgment, I would say, as I suggested about heterosis (Boorse, 1997, 90), that the BST, far from failing, is valuable precisely in showing the limits of the health concept—the borders beyond which it needs replacement by some other concept, such as adaptation.

Gammelgaard

Apart from her choice-of-goals argument, I am in fairly close agreement with nearly everything in Gammelgaard’s essay (2000). For example, I hold none of the following views that she attacks:

1. The functioning of a healthy human organism is deducible from evolutionary theory (113).
2. Physiological functions should be defined in evolutionary terms (113).
3. Evolution has S & R as goals (114).
4. S & R are intrinsically valuable to organisms (114).

No one, I think, believes (1); available evidence about human evolution is hugely inadequate to identify all current physiological functions. That is why “Darwinian medicine” (which came to prominence after my original papers) is only a “suggestive heuristic” for physiological research (Gammelgaard, 2000, 114–115). As to (2), I hold a goal-contribution analysis of function, which is equivalent, I believe, for biological functions to an S & R analysis, which is itself equivalent to a causal-role account with S & R as the outputs of interest. Since I am not a selected-effects theorist, the unit-of-selection controversy (113) does not touch the BST. As to (3), I do not attribute goals to evolution, but only—in the Nagel-Sommerhoff sense—to organisms and some of their parts. And I have never said that (4) S & R are intrinsically valuable for organisms. On the contrary, nothing has value to a nonsentient organism, while for sentient ones like us, life or reproduction may be worse than the alternatives, and diseases can be advantageous. What remains of Gammelgaard’s criticisms, I think, is only the incontinence example, discussed below.
Kovács

Finally, Kovács (1998) is, strictly speaking, not a critic of the BST. That is because he concedes that “the position of Boorse is a nearly correct description of the actual medical usage of the term ‘disease’” (Kovács, 1998, 35). His objection is rather that “the species-design view of health, which is the paradigm of contemporary medical thinking, is also at odds with modern evolutionary biology” (35; italics added). Because Kovács agrees that my account hit its target, I will not reply to all his ideas on health. Still, I do not think he succeeds in showing that medical thought conflicts with evolutionary thought—only, as with Sober, that their categories differ. Take his example of human immunodeficiency virus (HIV) resistance:

[The HIV virus has only recently been introduced into the human population, to which humans are not adapted, so it is highly lethal. There are however some—today highly species-atypical—individuals who happen to be somehow resistant to the HIV virus. Can we say, that they are diseased because they are species-atypical? ... This—today highly atypical—trait will become the typical one, after many generations. This is the basic principle of evolution. What is species-atypical today, can be advantageous in a new environment, and will become species-typical in the future (32).

The answer to Kovács’ question is “No.” HIV resistance is not a disease, since it is an extra function, not a missing one. If it became species-typical, then lack of it would be pathological; but its lack is not so today, any more than any other typical human weakness. Today, lack of HIV resistance helps cause a disease but is not itself a disease. This failure to distinguish the pathological from the pathogenic, health from adaptedness, recurs throughout Kovács’ essay, as in the preceding example of native American and Polynesian populations. Indians’ and Hawaiians’ lack of immunity to European pathogens that killed them was not itself pathological. Europeans were better adapted to environments containing these pathogens, but not thereby intrinsically healthier. I believe that Kovács’ attempts to identify adaptation and health, like Sober’s and others’, are misguided. As I have often argued (Boorse, 1977, 548–549, 553; Boorse, 1997, 41; Boorse, 2011, 51), these are simply different concepts, the first relative to environment, the second not. So if medicine focuses on health and evolutionary biology on adaptation, that does not mean medicine is in error. In general, Kovács seems to have little respect for standard medical concepts, and he sometimes frankly abuses them, as in speaking of “diseased” environments (Kovács, 1998, 34–35). Only living organisms can be healthy or diseased, though environments can be healthful or unhealthful for them. Finally, I reject any “cultural notion of health” (35). The fact that “the conscious goals of individuals and cultures” include abortion and contraception (35) does not show pregnancy to be a disease and infertility to be health, any more than clitoridectomy and human sacrifice show a clitoris to be a disease and death to be
health. Neither individuals nor societies have any power to decide what is a theoretically healthy human being.

VI. THE BST AS BAD MEDICINE

Let us now turn to critics who allege that the BST fails to fit medicine, in some general feature or as to specific conditions.

Cooper and Giroux

Rachel Cooper offers two criticisms. One is that the BST’s reference class is too narrow for medical judgments.

Boorse claims that the reference class for an organism consists of individuals of the same species, sex, and age. However, reference classes are going to need to be far more fine-grained than this. What is normal depends on a host of additional factors. Masai are naturally sensitive to growth hormone, pygmies are not. Athletes normally have a lower heart rate than other people. People who live at high altitude, or in hot climates, adapt in various ways. Thus the organisms in a reference class must not only be of the same species, sex, and age, but must also be of the same race, and must have undergone similar training, and have lived in the same kind of environment. This means that some reference classes are going to turn out very small. Elderly female Masai mountain-bikers, Asian male teenagers who have been brought up in Wales, and half-Chinese half-Eskimo boy toddlers will all need their own reference classes (Cooper, 2005, 14).

Here Cooper names three more factors: race, training, and environment. I have always said (Boorse, 1977, 558; Boorse, 1987, 370) that medicine may need to add race, and I have noted (Boorse, 1997, 8) Reznik’s Masai-pygmy example (Reznik, 1987, 85–86). Still, I am ambivalent about it. Supposedly, the height of each race is adaptive in its environment, which is what makes people want to call pygmies normal rather than sharing a disease. But if adaptation is our test of health, then we must equally call the condition of an HGH-insensitive Masai or an HGH-sensitive pygmy a disease. The latter, at least, is an implausible view for which I know no medical authority. Cooper’s point about mixed race is another good reason to resist adding race as a factor. As for environment, the BST already takes account of it in the ways noted below regarding Kingma. Also, I count typical irreversible adaptations to distinctive nonextreme environments, like hot or elevated ones, as permanently normal (Boorse, 2010, 67). I doubt that Wales is sufficiently unique for “Welsh childhood” to be a separate factor. But, in any case, we need not determine normality in an environment by people actually in it. Even if there were only one Masai child in Wales, what thousands of other Masai children’s physiology would be like in the same environment is still a fact.29

As to training, Cooper offers no reason to think it relevant in defining disease. The question is what is medically normal. Even if athletes have lower heart rates, that does not imply that a heart rate normal for nonathletes (say, 70) is pathological for them. It would be if caused by some sort of neural,
hormonal, or cardiac disease that overwhelmed the training effect. But such a condition is a disease by comparison with the usual reference class, not athletes alone. No condition is a disease only in athletes (or in nonathletes), and the idea of a condition that counts as a disease only in mountain bikers is a joke. So Cooper offers us only two factors—not “a host” of them—and the relevance of one of them, race, to medical disease judgments is disputable. Thus I have little fear that, as Cooper suggests (2005, 15), the BST will end up with single-member reference classes, or classes too small to distinguish functions from accidental effects.

Giroux (2009) has a different reference-class criticism. Increasingly important in modern medicine is epidemiology, with its study of risk factors. But epidemiologists study particular, concrete populations, not the whole human race. Giroux suggests that the BST would better fit contemporary medicine by replacing its single reference class with multiple ones, thus embracing the population-relativity of normality. She suggests that this means giving epidemiology equal status with physiology as a basic science serving to define health. But, first, epidemiology seems to presuppose a concept of disease, not to define one. Risk-factor epidemiology, as Giroux says, is about factors that increase morbidity and mortality from a given disease. Second, I am not sure that epidemiologists ever claim that the same condition (e.g., diastolic blood pressure over 90) is a disease in one group but not in another group. Although they work with specific populations, that does not mean that they are trying to define, say, “disease of a Framingham male.” Finally, I currently believe, with Schwartz (2008) and, I think, Giroux herself, that risk factor and disease are two separate categories badly confused in contemporary risk-based medicine.30

Cooper’s second criticism of the BST is that on it, homosexuality might be pathological, yet not harmful. But the anger of gay-rights protesters against psychiatry “implies that it is part of our concept of disease that diseases are bad.” And “if Boorse’s account is to be an account of disease, as opposed to an account of some quite distinct concept, it cannot stray far from our normal concept” (Cooper, 2005, 18). First, as I have consistently said, the BST’s target is the medical concept of disease (or, better, pathological condition), not any lay concept. If lay people misunderstand medical concepts, that is not my or medicine’s fault. Any science, including medicine, needs its own technical vocabulary. Second, I cannot tell whether Cooper also means to suggest that homosexuality cannot be pathological because to say so makes homosexuals angry. I hope not. That argument would also show that deafness, pedophilia, and even schizophrenia are normal, since some people who have them also protest their classification. If, as many believe, the American Psychiatric Association normalized homosexuality in 1973 because of a fear of further public protests, it was the end of psychiatry’s claims to be a science.
Kingma

Kingma (2010) argues that the BST “cannot account for some of our most basic and ordinary disease examples, such as infectious diseases, broken legs, scurvy and poisoning” (Kingma, 2010, 242). She presents her main argument via three examples of poisoning.

Anybody who overdoses on paracetamol [= acetaminophen] will induce a severe and irreversible case of liver failure. Liver failure, which is a reduced level of liver function, is the situation-specific quantitative normal species function in [this] situation .... According to the situation-specific BST it therefore must be healthy ... (251).

After a second example, carbon-monoxide poisoning, she offers a third, digestive example. She names four situations in which a person’s digestive system (following Hausman, let us call her Carol) has a typical level of function: (1) relaxing after a meal, (2) fasting, (3) after hard exercise, (4) after ingesting a poison that immobilizes the digestive system. After describing normal digestive function in situations 1–3, Kingma writes:

By the same analysis ... the digestive tract on occasion four (poison) should also be healthy: although the digestive tract does not perform its function (digesting food) on the occasion of having taken the poison or only performs this function to a very small degree, that is the normal contribution to survival and reproduction of digestive tracts on the occasion of poison taking. Moreover, the digestive tract is still disposed to behave normally on all other occasions (such as resting after eating a meal which did not include the poison, or resting after fasting). We should thus be forced, following the BST, to conclude that the poisoned digestive tract is healthy (252).

Kingma’s mistake here is her second-to-last sentence, which, as Hausman (2011) explains,\(^{31}\) is false. It is easy to see why if, like Hausman, we consider lethal environments, which, of course, include those causing acute liver failure, as in the acetaminophen case. If, as he imagines, Carol’s landmine environment has blown her to bits, she is not still disposed to behave normally on all other occasions. Rather, she is dead, and has no functional abilities at all. Health by the BST, as Kingma realizes (Kingma, 2010, 246), requires “normal functional ability,” which is “the readiness of an internal part to perform all its normal functions on typical occasions with at least typical efficiency” (Boorse, 1997, 8; cf., Boorse, 1977, 561–562). The fact that if Carol had not stepped on a land mine, her physiological functions would be intact is irrelevant. She did, and has now lost all functional readiness. Also irrelevant is that if other human beings had stepped on the same land mine, they would have suffered the same injury. They didn’t. The BST requires, for Carol to be healthy, that she have all functional abilities in any situation that typical human beings would have if placed there, at not atypically low levels. Typical human beings have not exploded. So they have innumerable functional abilities in mine-free environments that Carol now lacks. In particular, in Kingma’s own example, nonpoisoned human beings are fully ready to digest a meal.
Thus, Kingma fails to show that the BST cannot handle infectious diseases and injuries from poisoning or trauma, like broken bones. The only kind of disease to which her argument in the quotation is relevant is diseases maintained by an environmental factor, with no lasting change in the organism. This is a relatively tiny class, of conditions such as heat exhaustion, mountain sickness, or nitrous-oxide intoxication, which are almost instantly cured by change of environment. Even CO poisoning and heatstroke do not fall into this category. To deal with this tiny class of diseases, I proposed in 1997 to add to the BST a clause about rare environments:

\[ \text{[A] statistically species-subnormal function (in the usual sense of an arbitrarily chosen lower tail) is pathological if it results from an environmental factor outside an arbitrarily chosen central statistical range of that factor in the environments where the species lives (Boorse, 1997, 84).} \]

As Hausman says, I meant that typical functioning “in unusual environments that would be subnormal in usual environments is pathological” (Hausman, 2011, 661)—not, as Kingma supposed, that “all normal functions in rare environments are diseases” (Kingma, 2010, 255).\(^{32}\) The reason for the italicized condition, which Kingma misses, is that supernormal function produced by a rare environment is not pathological. On this understanding, the rarity of an environment with digestive poison is part of what makes digestive paralysis (scenario 4) pathological, but slow digestion after the common phenomenon of exercise (scenario 3) normal.\(^{33}\) And this difference remains though both scenarios, exercise and poison, produce a temporary change in the organism itself.

But there is another reason why (3) is normal: exercise is not just a typical human activity, but a typical function. The same holds of Kingma’s new examples in a later paper (Kingma, forthcoming) of normal losses of functional readiness. A man with an erection cannot urinate; a pregnant woman is less able to run, lift, bend over, or get up directly from a supine position, not to mention having normal morning sickness; a sleeping person suffers paralysis, loss of consciousness, and lower sensitivity to various sensory stimuli. As Kingma notes, one possible view of such examples is to call them “temporary pathologies,” disputing their classification as normal. I reject this answer, and not only because it departs from medical usage. It is also because all the examples illustrate a ubiquitous physiological phenomenon: the normal inhibition of one function by another. Efficient management of any goal-directed system—industrial plant, hotel, or organism—needs different functions active or ready at different times, since many tasks compete for resources, and some are incompatible. In vertebrates, an entire nervous system, the autonomic, governs the distribution of effort among organs. Sympathetic nerves stimulate one group and inhibit another; parasympathetic nerves, the reverse. So important to physiological goals is such regulation that it is duplicated by several hormones and fine-tuned for
specific organs by others (Boorse, 1997, 78–79). So the clearest difference between digestion inhibited by exercise and by poisoning is that exercise is a typical function in human beings, while eating poison is not. But if eating the poison were a species-typical activity—e.g., if the poison were in a typical foodstuff—then digestive inhibition following such a meal would be normal by the BST and, I submit, for medicine, too. In short, all of Kingma’s examples of functional losses that biomedicine counts as normal are part of the typical life pattern by which our species survives and reproduces. Only atypical activities or environments that degrade function can cause pathology.

To this, let me add a reminder that the BST requires all parts of a healthy organism, including cells and organelles, to have species-typical functional readiness. Now physiology tells us the typical lifespan of cells, which varies with tissue. In some tissues, notably the epithelium of skin and of the lining of the respiratory and gastrointestinal tracts, cells live only briefly before being replaced by new ones. But in most tissues, cell life is far longer, with some cell types living as long as the organism itself. The BST says that any premature cell death is pathological—including even clinically undetectable liver necrosis, as in mild poisoning. The same is true for cuts and bruises. If typical human beings always had dead cells in a particular location, then I would have to call the phenomenon normal, and so perhaps diverge from pediatricians’ and pathologists’ opinion of little boys’ knees. But I see no obstacle to calling pathological the skin injuries of adults like Kingma (2010, 256).

Several of her other examples of “harmful non-rare environments” also pose no threat to the BST, or at least no new one. One is sunshine. Kingma, who is light-skinned, says her “normal response to a couple of hours of summer sunshine” is a bad burn (Kingma, 2010, 256). Light skin is, as she says, one normal trait in a polymorphism, thought to be an adaptation to aid vitamin-D production, especially for pregnant women. To enhance this ability in low-light northern latitudes, some protection against ultraviolet radiation has been sacrificed. Now a light-skinned person can get burned in either of two ways. First, she could travel to a region of more intense sun. This is Engelhardt’s example of the Norwegian in Africa, which I have discussed before (Boorse, 1997, 80). Or, as in Kingma’s case, she could simply stay out in the sun at home. Then, atypically for light-skinned people, she is exposing herself too long to a constant environmental factor. Here the polymorphism is irrelevant. Much of our species lives in regions where, in winter cold, all naked people will freeze to death outside. But we do not do so, since we guard our bodies with clothes, shelter, and fire. Similarly, all humans have access to water, yet almost none die of water intoxication. So, although none of these environments can be called atypical, still, given typical behavior, the injury—sunburn or death—is atypical of our species and even of Kingma’s normal-variant subgroup. As for her examples of
endemic disease (Kingma, 2010, 256), while it may be that most humans live in malarial regions, most humans do not have malaria,\(^3\) nor are most children malnourished. But if such pathology were typical, then the BST would have to declare it normal (Boorse, 1977, 566–567). To the problem of typical disease, I see no solution but to retreat to a concept of ideal design which, so far, I am unable to define.

DeVito

DeVito (2000) begins (539–540) with an example to which he returns at length at the end. Only values, he says, distinguish the effect of *Helicobacter pylori* on one’s duodenum—a peptic ulcer, which is disease—from the effect on one’s colon of *Escherichia coli*, which is part of the normal biota of the large intestine.

DeVito describes duodenal ulceration as “perforation of the epithelium.” He concedes that whether this occurs, its likely effects on “other biological systems,” and how to cure the ulcer are all value-free facts.

But notice, knowing these facts about the body does not tell us whether or not the body is diseased. The development of duodenal ulcerations due to the presence of *H. pylori* is a perfectly normal (although unpleasant) way for the duodenal epithelium to respond to the presence of *b. pylori* or high acid secretion. Furthermore, the only difference in kind between the interaction of *b. pylori* and the duodenum, and *e. coli* and the intestines, is that we do not like the results of those interactions. Each species of bacterium takes nutrients from the body, seeks to avoid being destroyed by the body’s immune system, releases wastes and toxins into the body, etc. The only difference is an evaluative one. We do not like the effects of *H. pylori* infestation, while, for the most part, we do not mind the effects of *e. coli* infestation (DeVito, 2000, 561–562).

It should by now be clear how to answer this argument. To see quickly that it is fallacious, substitute for *H. pylori* a lethal bacterium like *Yersinia pestis*, or a lethal virus like Ebola. Then the conclusion is that only values distinguish life from death, which is absurd. There is an objective difference in kind between life and death, basic to biological science, and it does not lie in which we prefer. In peptic ulcer, the patient, of course, need not die; but mucosal cells do, in large numbers. An ulcer is a straightforward case of bodily injury, like a knife wound. A better example for DeVito’s purpose would have been *Vibrio cholerae*, which does not destroy colon cells. But biology not only distinguishes life from death; it also describes normal physiological function. Cholera is a disease because the vibrios block the colonic mucosa from its normal function of absorbing water. The contrast between bacteria that disrupt normal physiological functions and those that do not is reflected in the biological terms “pathogen” and “commensal.” (If both kinds of bacteria are, from their own point of view, doing similar things, that does not mean that their effects on the host are the same.) But DeVito would
perhaps claim that this biological distinction is value-laden, as he seemed to do earlier about life and death.

DeVito also criticizes the BST for its use of statistical normality. Different choices of reference class, he says, yield different health judgments. In his first example, if one chose “a population of 70-71-year-old women all of whom have osteoporosis” (DeVito, 2000, 546), then, he says, not only their osteoporosis, but also the resulting fractures would be healthy. This is, of course, neither medicine’s nor the BST’s view, for two reasons. First, the reference class could be women aged about 70, but not women with an atypical bone condition. Second, even if osteoporosis were typical in 70-year-old women, that would not make the resulting fractures normal, since it is false that most 70-year-old women break bones. Alternatively, DeVito says, if we choose all adult women as the reference class, osteoporosis becomes abnormal, and likewise abnormal over the lifetime of each. But the latter viewpoint is obviously not medicine’s: if normality were relative to the individual, there could be no such thing as congenital disease. In any case, the variation of health judgments with reference class cannot threaten the BST’s fit to medicine, any more than its value-neutrality (§III), unless its actual choice yields judgments with which medicine disagrees. DeVito offers no such example.

DeVito’s last objection is that the BST’s use of statistical normality also makes health and disease “contingent.” His examples involve changes in human lifespan and fertility. However, both misapply the BST.

Over the last 50 years life-span has increased by approximately 25 years and (in the West) reproductive output has decreased. So, a person who was 60 years old 50 years ago, an age beyond the average life-span of the time, and who had coronary artery disease that shortened her life-span from 70 years to 61 years was not diseased. But if that same person were to be examined today, she would be diseased. Similarly, if a person was only able to have 2 children 50 years ago, considerably below average for the time, she was diseased. But if that same person was only able to have 2 children today (quite close to the average) she would be well (DeVito, 2000, 546).

As to lifespan, DeVito seems to assume that, by the BST, no one older than the average age at death can have a disease. This is not so: the reference class includes only live individuals of a given age, so 60-year-olds at any time are compared with their living peers, not their dead ones. Or, perhaps DeVito is suggesting that, given the rise in life expectancy, Westerners 50 years ago had more badly clogged arteries than Westerners today. Certainly, the Western decline in fertility (546) is not due to a decline in women’s reproductive organs, so there is no reason to think medicine would judge a woman of limited fertility healthier today. But, in any case, I do not change the reference class every 50 years or relativize it to Westerners, since that would violate the idea of a human species design. Again, DeVito
offers no evidence that actual medical judgments differ from the BST’s in these cases.

Finally, both DeVito and Gammelgaard complain that the BST fails to fit medicine because medical treatment often aims at goals other than survival and reproduction.

[Life and reproduction ... do not specify every type of body-state that interests people or that falls under the category of diseases. People are also interested in maintaining quality of life. For example, patients with multiple sclerosis do not simply want to live longer. They want the spasticity of their bladders to be reduced so that they are not confined to a life of locating the nearest toilet. The eighty-seven-year-old bed-ridden man does not necessarily want the heart operation so that he will live to be eighty-eight. He wants to have the operation so that he can get out of bed. Finally, the patient blinded by glaucoma does not simply want to live for a long time; she wants to be able to see (DeVito, 2000, 545).]

Examples like these are no threat to the BST unless they are diseases without biological dysfunction. Is DeVito suggesting that, in the human species, vision is not a biological function of the eye? Does he think that pumping adequate blood for locomotion is not a biological function of the heart, and waste retention is not a biological function of the human cystic and anal sphincters? To a biologist, such claims, even the last, are ridiculous. How does DeVito imagine the two dual sphincters arose? It is just conceivable, as he says, that we might eventually eliminate any effect of these diseases on survival—though it is harder to imagine eliminating the reproductive disadvantage of someone who cannot “walk, hold a cup, speak, or maintain bladder control” (DeVito, 2000, 545). But if we did both, then, on my analysis of function and most others, eyes and sphincters would have lost all biological function in the human species. So the human design would have changed in such a way that the conditions were no longer diseases. Then medicine, presumably, would just add them to the current list of nondiseases, like monthly ovulation or small breasts, that patients want changed. But I cannot see how DeVito’s futuristic fantasy is an objection to the BST as an account of the medical concept of disease.

Venkatapuram

In two ways, Venkatapuram (2011) is not clearly a critic of the BST. First, he says from the outset that his goal is to overturn the “dominant” medical view of health as absence of disease, whereas the BST aimed only to analyze it. Second, much of his critique of the BST ignores my actual analysis. Besides conflating concrete processes with their functions (Venkatapuram, 2011, 46), he spends a whole page on a BST-like theory that “conceives of human beings as functioning at one constant level” (51): the BST, he says, “holds constant both the activity and the environment” (52). There never was such a theory (§II). He also usually treats my reference class as a “population” of
human beings, like Chernobyl-area residents (49), rather than as the whole species divided only by sex, age, and perhaps race. I will answer only his criticisms of the actual BST, as amended in 1997 to include the clause on rare environments.

Except for two points, Venkatapuram’s criticisms of the actual BST center on the issue of improving human health by changing behavior and environment. That this is both possible and desirable I agree. First, he seems to think that the BST makes most health comparisons impossible, since it allows only a binary classification, diseased or disease-free (Venkatapuram, 2011, 56). Such a division is, indeed, the BST’s target. Nevertheless, its biological-function framework allows many types of health comparisons. A single person may have more or fewer diseases; likewise for a population, which can also have more or fewer cases of the same disease. Also, any individual’s disease can be more or less severe, depending on how many functions it depresses, and by what degree. Finally, I endorsed a limited concept of positive health, beyond the absence of disease: “improvements of function that do not sacrifice any possible improvements of others,” something most Westerners can achieve by an exercise program (Boorse, 1977, 572).

Venkatapuram’s main concern is that to relativize normality to (non-rare) environments works against his goal of improving population health by changing environment and behavior. Presumably, massive nuclear radiation (Chernobyl) or chemical pollution (Union Carbide) (Venkatapuram, 2011, 49) is an atypical environment, as are environments with various endemic local pathogens. But, even among typical environments, suppose $E_2$ allows overall higher species-typical biological function than $E_1$. The BST does stop us from saying that changing $E_1$ to $E_2$ is eliminating disease. But it lets us call the change an improvement in the positive health of the population so affected. For all these reasons, I would not deny that “it is biologically possible for an entire population to achieve better functionings, and ... the differences are likely to be in the social differences between the two populations” (50), nor even that this change improves health. And nothing for me hangs on calling either situation “natural.” Contrary to Venkatapuram (53), many biological species improve their environments by building webs, nests, dams, etc., and nothing is more natural to human beings than to do so intentionally. I do not agree that any of this introduces values into the concept of health, even of positive health. What makes one state healthier than another is still level of biological function, not anyone’s values. But just for that reason, I doubt that the BST impedes Venkatapuram’s arguments for health justice, any more than it blocks disability-rights advocacy (Boorse, 2010, 77). Rather, it allows the full spectrum of positions on either topic to be defended.

Nordenfelt and Stempsey

The BST’s claims to fit medical usage, especially in pathology, are disputed by Nordenfelt and Stempsey. Consulting the standard world disease classification,
ICD-10 (World Health Organization, 1994), Nordenfelt finds some entries which he thinks involve no physiological dysfunction (Nordenfelt, 2001, 22). I agree as to many items in section R, “Symptoms, signs, and abnormal clinical and laboratory findings.” But there is no reason to think that ICD-10 is calling all these conditions diseases. The term “disorder” is not used in the section-R heading, and R includes items like “false positive Wassermann test” (World Health Organization, 2008, 859), which are, by definition, not pathological. As to his other examples, “delayed sexual development” is a reproductive dysfunction, and “obesity” is viewed by many as a metabolic disorder. Mental-health categories (Nordenfelt, 2001, 23–24) are covered by my remarks on Cooper in §III. I am not surprised if the writers of a psychopathology textbook, like so many people in mental-health fields, assume psychiatric classification to be a matter of social values, not biological dysfunction (24). If so, so much the worse for psychiatry.

Nordenfelt (2001, 21–22) also quotes from a standard medical textbook its description of one disease, asthma. He notes that the description includes much more besides the “basic pathophysiological element” of narrowed airways, such as clinical features, prognosis, and treatment. I have noted this from the first (Boorse, 1977, 552–553; Boorse, 1987, 364–365) in discussing “disease entities.” My assumption has always been that to decide what conditions are pathological (the demarcation problem) is independent of their classification (nosology). With asthma and other entities, one can debate whether the disease is truly the airway constriction, or its underlying cause (Nordenfelt, 2001, 18–19). Otherwise, I see no issue here until critics produce a pathological condition lacking physiological dysfunction—perhaps, an early stage of disease.

Most recently, I said that the BST aims at a pathologist’s concept of disease, not a clinician’s (Boorse, 1997, 45–46, 49–50), so I am willing for it “to live or die by the considered usage of pathologists” (Boorse, 1997, 53). Nordenfelt and Stempsey agree that this test kills it. For one thing, they think pathology textbooks define the field otherwise than does the BST. They quote several such definitions by textbook writers:

Pathology has been defined as the medical science that deals with all aspects of disease, but with special reference to the essential nature, the causes, and the development of abnormal conditions (Stempsey, 2000, 325).

Pathology is the scientific study of disease; it follows the process from its inception to its termination and it investigates the changes produced (Stempsey, 2000, 325).

Any departure from the normal structure and function of an organism is a disease (Nordenfelt, 2001, 19).

Translated literally, pathology is the study (logos) of suffering (pathos). As a science, pathology focuses on the structural and functional consequences of injurious stimuli on cells, tissues, and organs and ultimately the consequences on the entire organism (Stempsey, 2000, 325).
Nothing in these quotations conflicts with the BST except the inclusion of abnormal structure, which I have discussed before (Boorse, 1977, 565–566). Otherwise, these definitions describe pathology exactly as I did (Boorse, 1997, 52–53), as the basic general science of disease in all its aspects. That answers Stempsey’s point about the prominence of “the morphological characteristics” of disease in pathology practice (Stempsey, 2000, 323). It is true that to reach the modern scientific view of disease, one must join Virchow’s cellular pathology (323) to Bernard’s pathophysiology. But by all four definitions above, pathophysiology, which the BST takes to define disease, is part of the science of pathology. What these definitions do not do, but the BST does, is to break out of the circle (Boorse, 1977, 542) of interdefinable medical concepts via a substantive analysis of disease in biological and other scientific terms.

In any event, in saying that the BST’s test should be the considered usage of pathologists, I did not mean their general definitions. I meant their considered judgments of individual conditions as normal or pathological. There is no reason to expect pathologists, or any other scientists, to offer accurate analyses of their concepts, a task more suited to analytic philosophy. For example, two definitions Stempsey quotes cite etymology, and he actually endorses it: “Pathology is the study of suffering” (Stempsey, 2000, 326). But that is the etymological fallacy. The current meaning of English words is not determined by their foreign roots, else an apology would be a self-justification and an entrée an appetizer, or even by their meaning in earlier English, as readers of Shakespeare know. Taken as a definition, Stempsey’s Grecian formula is wildly overbroad: pathology ignores rejection in love, loneliness, career failure, poverty, political oppression, torture, and so on through countless other kinds of nonmedical afflictions. It is also too narrow. A pathological condition may never cause suffering, as with early prostate cancer in an old man who will soon die of something else. A pathological condition can consist precisely in lack of suffering, as with the loss of pain sensation in syringomyelia or leprosy, or in total unconsciousness, as in coma, an example that Nordenfelt later uses against Fulford (Nordenfelt, 2001, 83). Remarkably, Nordenfelt (20) still cites two plant pathologists claiming that “plant pathology is the study of the suffering plant,” a usage they see as “not stretched too much.” Unless they believe plants to be conscious, suffering in the usual sense is stretched to impossibility, as it is for nonsentient animals like the cockles and clams of the Journal of Invertebrate Pathology.

A good example of what I mean by “considered” judgments is pathology’s view of defense mechanisms like inflammation, fever, and the immune response. Nordenfelt suggests that these are generally seen as part of a disease (Nordenfelt, 1987, 30–31; Nordenfelt, 2001, 17). I agree that inflammation and fever are often called pathological in medical books. However, I think this is an error, due to two causes. One is a traditional failure to recognize the nature of at least the first two of these processes as defense
mechanisms, \textit{i.e.}, normal, usually beneficial biological functions. For fever, the importance of this mistake has been emphasized by Wakefield (1999a, 392–393) and apostles of Darwinian medicine. The second cause is terminological: the lack of a standard medical term for “indicating disease.” I have proposed “pathodictic” for this purpose (Boorse, 1987, 387). If “pathodictic” joined “pathologic” and “pathogenic” in medical usage, I feel sure that pathologists would agree with me that in infection, inflammation, fever, and the immune response are pathodictic but normal (nonpathologic). By contrast, the immune response in autoimmune disease, such as systemic lupus erythematosus or Graves’ disease, is pathologic. But, as this point illustrates, Nordenfelt is right (2001, 26) to describe my project as “philosophical explication,” or rational reconstruction, in the logical-empiricist tradition of Carnap, Hempel, and Quine. That is already clear from my rejection of medical descriptions of purely structural abnormalities and of typical diseases as pathological (Boorse, 1977, 565–567). I still think my effort is in some sense an attempt at a lexical definition of scientific terms (Boorse, 1977, 551), but with the proviso that scientists are sometimes confused, inconsistent, or (as with fever) empirically wrong about their subject.\textsuperscript{12}

Finally, Stempsey has a section arguing that the BST is not value-free because “[o]ur very descriptions of anatomy and physiology are themselves value-laden” (Stempsey, 2000, 327). His reason for this is that science and knowledge in general are value-laden. But, as I have said, the only important issue is whether medical theory is value-laden in a way that the rest of science is not (Boorse, 1997, 56). Stempsey offers a specific argument, inspired by Putnam on mereology and using my own example of normal structure, the cranial nerves. We could, he says, consider the trigeminal nerve as two nerves, motor and sensory, just as we could consider the pancreas as two organs, exocrine and endocrine.

But to choose a particular conceptual schema that tells us which are the wholes and which are the parts ... must reflect the conceptual values one has .... Such choice has important implications for the concept of disease in the BST because the specification of proper functioning of organs must necessarily rely on what we count as organs (Stempsey, 2000, 328).

Waiving all objections to Putnam or “conceptual values,” I do not see how such indeterminacy poses a problem for the BST. For either nerve or pancreas, if the organ is $A + B$, then it has two (types of) functions, while if $A$ and $B$ are organs, each has one. But, either way, impairment of $A$ is pathological by the BST, since an organ fails in one of its (one or two) functions.

Schwartz

Peter Schwartz has a fine criticism (2007). He notes that, for many functions, the proportion of individuals that we intuitively view as dysfunctional varies with reference class. He divides this point into two problems
for the BST: “common diseases” and “healthy populations.” As to common disease, if it turned out that 20% of 70-year-old men had cardiac ejection fractions of 20%, we would still want to call this congestive heart failure. Already, 17%–28% of such men have urinary dysfunction from benign prostatic hypertrophy (Schwartz, 2007, 375). On the other hand, if all 20-year-old men had ejection fractions ranging from 50%–70%, we would not wish to call even the lowest 1%–2% pathological. Although Schwartz does not put it this way, we see here a fundamental objection: that on the BST, for any biological function, it looks like a necessary truth that disease exists in the reference class. Intuitively, we must allow the possibility of a totally healthy population, especially for at least one function.

Schwartz’s solution to both problems is to add a dimension to the BST. Besides functional level and prevalence, the axes of my bell-like curve, he suggests adding a third axis, “negative consequences.” At first sight, adding a dimension just seems to replace a line-drawing problem (Schwartz, 2007, 366) with a curve-drawing problem, as he may realize. At any rate, rather than discussing his arguments for the superiority of his approach, I will try to defend the BST from his criticism.

First, Schwartz is one of several writers to think that statistics itself requires some specific choice of normal range, such as $\mu \pm 2\sigma$. He says that, for a normal (Gaussian) distribution, “the line must be drawn near two standard deviations below the mean. The exact location can be chosen arbitrarily as long as it falls in this area” (Schwartz, 2007, 375). But, for a Gaussian or most other distributions, the limits $\mu \pm 2\sigma$ are merely conventional; they have no theoretical significance in mathematics or statistics, only practical convenience. I specifically said that this choice is no way required by the BST (Boorse, 1987, 371; cf., Boorse, 1977, 559). Hence, the BST is consistent with disease prevalence of 35%, 20%, 5%, 1%, or, I suppose, even 0%, and with prevalence varying from disease to disease. What it is inconsistent with is prevalences $\geq 50\%$.

Still, most examples of such “typical disease” are diseases of old age. So a second point is that almost all these problems, and more, would disappear if the BST counted age as irrelevant after adulthood. That is, we could make young adults the standard for all adults. To do so would make the BST look even more biological, since biologists, though they catalogue immature stages, do not usually catalogue stages of senescence. This view, of course, greatly limits the category of “normal aging.” All functional declines with age to far below the young-adult mean would be pathological. But Schwartz is sympathetic to this result, citing some people’s view of aging as a universal disease. So I would wish to explore this slightly revised BST before adopting Schwartz’s more radical three-dimensional picture.
VII. FORMAL FEATURES OF THE HEALTH CONCEPT

Two recent writers question whether the BST has the right formal features for a concept of health.

Guerrero

J. David Guerrero (2010) argues that on my analysis, an individual’s health status could change, not because of any internal physiological change in him, but merely because of a change in the rest of the reference class. The possibility of such a “Cambridge change,” Guerrero says, both creates internal conflict within the BST and is “theoretically dubious” (Guerrero, 2010, 272, 276), by which he seems to mean medically implausible.

My answer is threefold: (1) the theoretical possibility of a Cambridge change in someone’s health status seems to be entailed by any view of health as normality, an idea basic to scientific medicine. But (2) it is far more difficult than Guerrero thinks for such a change to occur, and (3) the only realistic ways in which one could occur are of no importance to medical practice.

A Cambridge change does seem theoretically possible by the BST. To use Guerrero’s first example, if Alvin’s heart had average blood-pumping ability, but the rest of current humanity instantly acquired Diana Nyad’s cardiac fitness, then Alvin would, it seems, suddenly become diseased. Actually, as Guerrero notes, I count a “reasonable time-slice of the species” into the reference class (Boorse, 1997, 66). So the paradox is possible only if the current human population outnumbers the rest of the population in our time-slice, who had Alvin-like hearts during their lives. Also, one must presumably wait a while for the new, improved hearts to beat long enough to overwhelm the whole human cardiac past, so the change would not be immediate. All the same is true for the reverse possibility: that Alvin, with current heart disease, instantly becomes healthy because everyone else’s heart suddenly suffers major damage. It seems to be a myth that most human beings are now alive. But, even if this claim is false, it could have been true. So I will just embrace the theoretical possibility of an individual’s changing from health to disease, or the reverse, without any internal change in him.

Contrary to Guerrero (2010, 276–277), this creates no conflict within the BST. What is judged health or disease is still an internal functional capacity of the individual. What has changed is only species-typical functional capacity—the benchmark for whether the individual capacity is healthy or not. Because the healthy or diseased state is internal in either case, my answer to Hare on fleas and lice is irrelevant (Boorse, 1997, 68). And there is no conflict with my account of function. Alvin’s heart’s capacity to contribute to his survival and reproduction is unchanged. It has merely gone from species-normal to species-subnormal, or the reverse.
Guerrero tries hard to come up with a Cambridge-change scenario with some real chance to occur. He discusses at length my eye example: “If the whole earth went pitch black for two days, I would not say eyes had lost all function in the human species” (Boorse, 1997, 66). But, with one exception, I do not see how any of his variations on it create a Cambridge change, unless by the same scenario as Alvin’s heart. For example, the distinction between qualitative and quantitative functional normality (Guerrero, 2010, 275–276) seems irrelevant. The exception is this. We imagine that the earth is pitch-black for a very long time, long enough that we reach a day when we are ready to count human eyes as having, like the appendix, finally lost their function. On the day when this occurs, blind people’s vision changes from unhealthy to healthy. Note, however, that there is no reason for an organ to become vestigial on a specific day, or perhaps in anyone’s lifetime. The vestigiality concept is not that precise. And, even if it were, the change would have no effect on medical practice, since, in the millennia-old darkness, no one with eyes of any kind could now see anyway. Thus, unless some Alvin-type scenario is plausible, I conclude that the theoretical possibility of a Cambridge change is no reason to find medicine wrong, as Guerrero suggests (277), to view health as species-typical functioning. In reality, it is no more implausible to view “healthy” as relative to a reference class than to view “tall,” “strong,” or “smart” in the same way—all of which terms, on some standard semantic analyses (Schroeder, 2013, 3), also allow Cambridge changes in extremely unlikely situations.

Schroeder

Andrew Schroeder writes a remarkably interesting essay (2013) on the logic of health concepts. He distinguishes *comparativist* from *noncomparativist* accounts of health. The former take “x is healthier than y” to be the basic concept, while the latter use “x is healthy.” The BST, he says, like all other influential accounts, is noncomparativist. I agree. Schroeder argues that this not only gives the BST (well-known) problems with common diseases, but also makes its treatment of “intergenerational” health comparisons implausible. As to the latter, he poses a paradox for traditional accounts of health. He imagines Alys, a medieval woman with a diet rich in iron for her time, and Allie, a contemporary woman of the same age with a diet poor in iron for our time. Because modern diets have much more iron than medieval ones, Allie still has more iron than Alys. On a traditional health theory, Schroeder thinks, we are drawn to assert each member of an inconsistent triad.

1. Alys was healthy.
2. Allie was unhealthy.
3. Allie was healthier than Alys. (Schroeder, 2013, 139)

He admits that I am not caught in this paradox, since my reference class does not vary with recent historical time. For me, (1) and (2) cannot both
be true. But he objects that such a reference class is implausible, since then “whether or not my heart is healthy depends on the heart function of people who have yet to be born” (Schroeder, 2013, 139 n. 13).

Actually, this conclusion is not so wild. If we are willing to conclude that most medieval women suffered from iron-deficiency anemia, we might, as Schroeder says (2013, 140), admit the possibility of discovering that most people even in 2014 suffer some as-yet unidentified deficiency disease, which will be avoided in the future. But, if we wish to block this possibility, we can just appeal again to standard practice in biology. When biologists describe a species or subspecies, they report how it is and has been—not how it will be, which is usually unknowable. If ornithologists say that the red-breasted nuthatch is confined to North America, they are not claiming that this will always be so. It is natural in biology (even if it upsets metaphysical eternalists) to view the past and present as facts, but not yet the future. Since I suppose medicine to take a similar attitude, I exclude future organisms from my reference class. This leaves me, I admit, unable to recognize diseases that have always been species-typical.

Schroeder sketches a comparativist version of the BST (Schroeder, 2013, 144–146) and argues that it is superior, save for one potential problem: confusing fitness with health.

Extreme weakness or fatigue is plausibly a health problem, but at the other end of the spectrum we wouldn’t ordinarily say that the difference between a gold medal-winning endurance athlete and a mere olympic qualifier was one of health.

Everyday discourse and medical practice, then, seem to recognize a difference between disease or disability, on the one hand, and something like fitness or talent or superior ability on the other. ... For any trait where we call a severe deficit pathological or disabling, a comparativist theory ... will be pressed to make all other (non-deficient) levels potentially differences in health, too (148).

I find Schroeder’s answer to this objection unconvincing. His examples of making healthy blood pressure or weight even “healthier” involve the currently fashionable confusion of “healthy” and “healthful,” cure and prevention. And the thesis he simply embraces, that “[t]he gold medalist is, in a certain respect, healthier than those she regularly defeats” (149), is implausible.

Schroeder’s comparativist view has other drawbacks that he does not mention. First, it makes talk of “perfect health” not just utopian, but nonsensical. Although we can still define “disease” on Schroeder’s comparativist BST, we cannot call total absence of disease perfect health, since any functional ability could be improved. Second, not only can we now do intergenerational health comparisons, as he wishes. We can also do sex and age comparisons—at least, like Schroeder, with respect to single abilities. On average, men’s biceps muscles will be much healthier than women’s. Women, though, will be far healthier in their ability to give birth. In that
respect, all men will be as unhealthy as can be. In countless ways, adults will be healthier than children. On the bright side, children get healthier and healthier as they grow. Sadly, though, babies are an utter health disaster. Since Schroeder eliminates species-relativity from health judgments, we will also have to accept health comparisons between species. Thus human color vision will be healthier than dogs’, who see the same colors as people with red-green color-blindness. But eagles’ vision is healthier than humans’ in that their long-distance acuity is far greater. Worse yet: even diseased members of one species will be healthier than nondiseased members of another. A horse or cheetah with muscle disease that can outrun an average human 21-year-old will be healthier. I find each of these theses a far greater departure from medical, indeed from ordinary, usage than any awkward aspect of the BST. Still, like Schroeder, I look forward to future development of comparativist health theories—especially those based on the BST’s central ideas of biological function and species-relativity.52

NOTES

1. For a previous overview of my position, see Boorse, 1997, 6–15.
2. Since it was in my final 1977 analysis, it was not, as Venkatapuram states (2011, 48), added in 1997, but more nearly subtracted then.
3. For more discussion, see §VI below on Kingma and Venkatapuram.
4. Schwartz notes a possible objection: that a variant trait could have the function of extending survival by interfering with reproduction (Schwartz, 2007, 372 n. 8). On my view, this could be true of an individual. I am not sure it can be part of a normal species polymorphism.
5. See my “Clinical normality,” §I.
7. Murphy claims that “Boorse ... adduces everyday linguistic usage and commonsense intuitions as evidence, even though he claims to be discussing the clinical concepts of health and disease” (Murphy, 2008, 16, quoting 2006, 52). But he cites no examples.
8. This empirical fact is all that is assumed in Murphy’s only two citations where I supposedly take an evolutionary view: Boorse, 1977, 550 and Boorse, 1997, 32. The former paragraph calls the connections it describes “empirical” (as opposed to conceptual). The latter quotes a later paragraph from that 1977 essay, which only rebuts the charge that my concept of species design is inconsistent with evolution.
9. A common example, often called by Gould and Vrba’s term “exaptation,” is sea-turtles’ use of their flippers to dig egg-holes. There is, of course, a debate among evolutionists between “adaptationism” and other points of view. But what is at stake is how much of organisms’ structure is accounted for by adaptation. All sides would agree that most structures agreed to have a function were designed for it by selection.
10. The same mistake is apparently made by Bolton (2008, 116).
11. For a full-length discussion, see my “Goals of medicine” (Boorse, forthcoming).
12. The confusion between concrete process and function ultimately leads Venkatapuram to a misconception about the BST much like Bolton’s.
frequently in mental functioning among a group of human beings should be viewed as what is healthy (Venkatapuram, 2011, 48).

Any such appearance is due, I expect, either to a vacuous concept of function or to a confusion of mere abnormality with subnormality.

13. “the centrality of the normal-distribution curve in the BST” (Giroux, 2009, 51, my translation). A more amusing error by Giroux is to read me as saying that adulthood begins around the age of 7–9 years (49). Pas du tout! Otherwise, her presentation of the BST seems very careful.

14. I do not discuss Varga’s essay (Varga, 2011) in this paper because its three main criticisms of me come from other writers. His charge of circularity, which would fit into §IV, is the same as Bechtel’s (1985), to which I have already replied (Boorse, 1997, 17–18). Kingma and Cooper I answer below.

15. DeVito also attacks the BST on its choice of reference class, but does not claim, as Kingma does and as he himself did about goals, that this choice is evaluative. He merely notes that if one varies the BST’s reference class, the health status of conditions will change. See §VI below.

16. DeVito purports to contrast a scientific concept like “electron” with goods like life and reproduction, but I do not understand his contrast. He says that while we may value electrons, “the criteria for being an electron have nothing to do with our values or whether we value an object that carries a particular charge” (DeVito, 2000, 543). But he does not explain how life and reproduction differ from this—unless he believes that they are actually defined in value terms, which, again, contradicts the statement quoted in my text.

17. Actually, “mare” can be used to cover any adult female equine animal, including asses, mules, and zebras, and “adult” here means “sexually mature.” I oversimplify for simplicity’s sake.

18. For more vivid examples, consider the vertebrate theory (VT) of marehood and Sheila the salmon; the animal theory (AT) of marehood and Sheila the octopus; etc.

19. I omit Ereshefsky’s remarks on “inherent” or “intrinsic” nature vs. genealogical species, since I have fully defended the BST against charges of essentialism (Boorse, 1997, 32–40).

20. In 1980, Sober stopped short of the conclusion that normality and health are not biological concepts. He wrote:

What we should conclude is that these functional notions of normality are not to be characterized in terms of a historical notion of fitness. Perhaps they can be understood in some other way; that remains to be seen. (Sober, 1980, 378)

But, in 1984, he flatly denied that mental retardation from malnutrition is objective pathology:

The perspective on development offered by the norm of reaction cuts very deep. ... Malnutrition while in the womb may reduce the child’s intelligence; a well-nourished child may have its intelligence boosted by various enriching experiences. But through the complexity of these thousands of variables, we may think we discern the child’s “natural” level of intelligence ...... The norm of reaction, on the other hand, makes this whole system of thinking look like a delusion (Sober, 1984, 160–61).

Given this shocking dismissal of the whole idea of normal vs. pathological child development as unscientific, I think it is fair to attribute to Sober the argument in my text.

21. In death, all biological functions of all parts fail; in disease, on the BST, at least one biological function of one part fails. Where such failure is total, as in almost all disease states, the disease judgment doesn’t even suffer from vagueness.

22. In his 1980 summary, seeking to draw a political moral from his analysis, Sober writes: “It is no more a part of human nature to be healthy than to be diseased” (Sober, 1980, 379). Again, this is no more true, profound, or morally relevant than its life-death counterpart: “It is no more a part of human nature to be alive than to be dead.” Both are either false or trivially true, depending on what “nature” means. Interestingly, Sober’s (1994) version of this essay dropped all five 1980 paragraphs on the scientific status of health and its moral and political implications.

23. And normality cannot be historically defined as “having all the parts it ever had,” since any animal species can have congenital defects.

24. Indeed, what Darwin identifies as “the first recognition” of “the principle of natural selection” is W. C. Wells’s 1818 hypothesis that one of the races of early man was selected by being “better fitted than the others to bear the diseases of the country” (Darwin, [1859] 1882, xv, quoting Wells, italics added).

25. For still other leading evolutionists’ broad use of disease language, see Huxley (1954) 1958, 115) (“pathogen,” for plants generally); Gould (1987, 361) (“disease,” for plants generally); Mayr (2001,
199) (‘pathogen,’ for organisms generally); Williams (1996, 243) and Sober (1984, 89 ff.) (‘disease,’ for organisms generally).

26. E.g., Phyttopathology or Plant Disease. The examples are from Plant Pathology (2004) and (2005). Since plant pathology, like human or veterinary medicine, is a clinical discipline, most research time is spent on diseases of plants important to human beings. But, as with animals, that does not restrict its concept of pathology, and there are general treatises on plant disease not limited to cultivated plants, such as Scheffer, 1997. An example of a paper on diseases of wild grass is Newsham, Fitter, and Watkinson, 1995.

27. See also the arguments in Wachbroit (1994), though I do not agree with all he says about biological normality.


29. See also note 39 below.

30. An extended discussion is in part III of my “Clinical normality” (2012).

31. One of Hausman’s points about counterfactuals was also made independently by Garson and Piccinini (2014).

32. Because of this misinterpretation, Kingma’s examples of rare nonharmful environments are irrelevant. As to her total-darkness example, I do not see why Hausman (2011, 661) finds it problematic for me. The function of the eyes is to transmit to the brain images caused by available light. In darkness, there is no occasion for the eyes to function, just as there is no occasion for the stomach to digest food during a fast.

33. Hausman does not, however, embrace this answer (Hausman, 2011, 661 n. 6, 665). See his last section for a different suggestion about digestion after exercise.

34. World Health Organization (2012) recently estimated that 3.3 billion people are at risk of malaria, but only 219 million have it.

35. For more on this issue, see the discussions of Venkatapuram and Schroeder below.

36. Gammelgaard (2000, 110) repeats the incontinence example.

37. I guess he could propose, implausibly, that the sphincters evolved in our animal ancestors but have no function today. Then, as with any useless structure, they are being selected against. In that case, today’s incontinents are the wave of the evolutionary future, when human adults will dump their wastes wherever they go or else wear bulky, smelly diapers.

38. DeVito writes: “It is absurd to deny that health care providers, physiologists and patients are interested in maintaining or improving the quality of life of patients. ... Boorse is wrong to attribute solely the function statements in biology.

39. One is Nordenfelt’s defense-mechanism example (Nordenfelt, 1987, 30; for my reply, see Boorse, 1997, 84–85). The other is Venkatapuram’s complaint that the BST treats dying prematurely from childbirth as “normal functioning” (Venkatapuram, 2011, 56). This is a fair criticism, since, in discussing normal reproductive costs, I referred to “the high morbidity and mortality” from pregnancy and birth “without medical treatment” (Boorse, 1997, 94). On the BST, large atypical functional limitations due to reproduction are pathological. Unlike the species mentioned in my 1997 paragraph, in which father or mother invariably dies, most human deliveries do not end in the mother’s death, and most human pregnancies do not cause serious illness. Nor is it true that most human females eventually die in childbirth. The fact is that I got carried away in that passage and misapplied the BST.

40. A crucial point that Venkatapuram misses is that my 1997 environment clause relativizes normal functions to individual environmental factors, such as temperature or elevation—not to unique total environments. After all, gardeners distinguish multiple “microenvironments” within a single backyard, where only one organism of a species might live. So it is unlikely that the BST’s “environments” will ever match political boundaries (Venkatapuram, 2011, 54). Also, even if most of a local population is exposed to and infected with, e.g., Schistosoma, it is an atypical, extreme environmental factor for the species as a whole and so can still be a pathogen by the BST. Only species-typical pathogens would be a problem.

41. I am, however, gratified to see Nordenfelt’s other plant writers offering something very like the BST, describing plant disease as a process “harmful to the physiological ... vital functions” of plants (Nordenfelt, 2001, 20).

42. As a parallel, my aim seems similar to Neander’s description (1991a) of her goal in analyzing function statements in biology.

43. For other examples of diseases of high prevalence, see Boorse (2010, 66) and section III of “Clinical normality” (Boorse, 2012).
Schwartz also mentions a 30% prevalence of hip dysplasia in some dog breeds. I would throw out this example, since a breed is not a species or subspecies. Human beings often create pathology, directly or indirectly, by redesigning an individual organism or group of them. Unlike Cooper (2005, 37), I am inclined to call both the size and the resulting obstetrical problems of dwarf rabbits pathological. Still more natural is to call total sterility in human-bred plant varieties, like seedless grapes (ibid.), pathological. The fact that "they are as plant breeders want them to be" is irrelevant. Animal models of human diseases are bred by medical researchers specifically to have a certain disease; it does not cease to be a disease because they are bred to have it. Likewise, neutered farm animals and pets have a pathological condition, however pleased their owners are that they do.

44. He concedes the vagueness of "significant" negative consequences (Schwartz, 2007, 383).
45. For some discussion, see my "Clinical normality" (2012, §1).
46. This proposal differs slightly from one made to me independently by two people (Jeff McMahan and Mark Greene): to deal with aging by the rule "once a disease, always a disease." If completely general, that rule would make some normal conditions that are pathological in childhood pathological in adulthood too, such as closure of the fontanelles or cessation of bone growth. My idea here is to start the McMahan-Greene rule at the onset of adulthood.

Another idea would be just to lump adults into one age group, making all adults of the same sex a single reference class. But then older adults might swamp younger ones. For example, if most adult women were past menopause, youthful infertility would cease to be a disorder.

N.B.: just as the age of full mental maturity can be long after the age of full physical maturity, there can be different ages of maturity for different physical traits.
47. Would such a revision fit my methodology of analyzing existing medical concepts? I am not sure that medicine has ever had a consistent view of diseases of aging. If it did, there were signs it may be changing, and in the direction of better fit to biology, as just noted. For example, current views of osteoporosis precisely judge older women by young ones, defining it as a bone mineral density (BMD) more than 2.5 standard deviations below the mean for young, healthy adults.

However, medicine does not seem ready to call menopause pathological, as the revision makes it. And, in general, it seems to be a live scientific issue whether senescence is a selected, genetically controlled life stage in the human design. Some, therefore, might argue that whether "normal aging" is pathological is an empirical question. So I am not endorsing this revision of the BST. I thank Barry Smith for the ideas in this paragraph.
48. Curtin (2007) says: "the number of people alive today is ... dwarfed by the number of people who have ever lived". She cites an estimate by demographer Carl Haub that about 100 billion human beings have been born since early Homo, about 50,000 years ago, which would put Earth's current population at about 6% of the total.
49. To establish this, he quotes (2013, 150 n. 26) my original paper: We have supposed that the basic notion is 'X is a healthy Y' ... As long as the efficiency of all functions exceeds a minimum, any value of these traits is as healthy as any other. In this way, our definition [...] recognizes a wide range of individual differences of equal intrinsic health (Boorse, 1977, 562–3).

The second part of this quotation is flatly noncomparativist, but, interestingly, the first sentence is not. One could instead take the basic notion to be "x is a healthier Y than z is," perhaps leading to a plausible comparativist version of the BST.
50. This possibility was raised by Margolis (1976, 247) and Reznik (1987, 97).
51. Some of Schroeder's reasons, however, are debatable. He complains that the BST's boundary of normality for any function is vague—e.g., 20/40 vs. 20/50 vision (2013, 145)—whereas his comparative health judgments are precise. But, within the realm of pathology, the BST yields exactly the same comparative judgments. To use his analogy: wherever the boundary for "tall" is, two people's tallness rank relative to it is the same.

I also doubt that the comparativism-absolutism contrast has the implications for medical ethics that Schroeder claims. For example, I see no difference as to treatments vs. enhancements; health is equally "extrinsic" (Schroeder, 2013, 151) on both theories. And his examples of the moral importance of "mid-range health differences" (154) are questionable. He is right that the traditional approach may make it impossible to prefer treatment B (raising someone from 20th to 75th percentile) to treatment A (raising someone else from 19th to 30th percentile) on health grounds. But there is no reason why policymakers must prefer small health gains to large gains in other sources of utility, such as IQ (155), wealth, education, and so on. On Schroeder's view, his examples are choices within health policy; on mine, they are tradeoffs between health and other goods. But why does that matter? Exactly as he says just before, in rebutting a Rawlsian defense of noncomparativism, health is only 'one component of well-being' (154).
52. I am grateful to Jefferson Medical College in general, and to Dr. Gonzalo Aponte and Dr. Steven Herrine in particular, for allowing me to attend classes in pathology and clinical medicine, first in the 1980’s and then again in 2012. Most of what I know about medicine I learned beneath Jefferson’s winged ox.

REFERENCES

———. Forthcoming. Goals of medicine.


