

# Paracetamol, Poison, and Polio: Why Boorse's Account of Function Fails to Distinguish Health and Disease

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## ABSTRACT

Christopher Boorse's Bio Statistical Theory (BST) defines health as the absence of disease, and disease as the adverse departure from normal species functioning. This paper presents a two-pronged problem for this account. First I demonstrate that, in order to accurately account for dynamic physiological functions, Boorse's account of normal function needs to be modified to index functions against situations. I then demonstrate that *if* functions are indexed against situations, the BST can no longer account for diseases that result from specific environmental factors. The BST is impaled on either horn of this dilemma and therefore must be dismissed.

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The distinction between health and disease is considered foundational to distributive justice in health care and to problems in bio-ethics, such as the distinction between treatment and enhancement.<sup>1</sup> 'Disease' in this context has a much broader meaning than in ordinary or medical language. It is an umbrella-term that encompasses all conditions that impair health, including traumata, disabilities and minor or temporary pathology not normally considered unhealthy, such as bruises, short-sightedness, a slight cold or tooth-decay.

The literature on health and disease is divided into two main and opposing positions: *naturalism* and *normativism*. Naturalists claim that health and disease are not determined by our subjective evaluations of a state, but are purely a matter of biological fact.<sup>2</sup> Normativists reject this claim to objectivity and maintain that health and disease are essentially value-laden.<sup>3</sup> The most dominant theory of health and disease is Christopher Boorse's ([1975], [1977], [1997]) naturalistic *Bio Statistical Theory* (BST), which defines health as the absence of disease and disease as the adverse departure from normal species functioning. Normal species functioning, in this account, is defined statistically. Despite 30 years of criticism, the BST has remained relatively unscathed.<sup>4</sup>

In this paper I present a novel challenge to the BST. I contend that Boorse's account of normal species function fails to distinguish between physiological and pathological responses that are due to a specific environmental factor. For example, it cannot tell us that a tan in response to sunshine is healthy, but that sunburn is not. It thereby cannot account for some of our most basic and ordinary disease examples, such as infectious diseases, broken legs, scurvy and poisoning.<sup>5</sup> I conclude that the BST either needs to be dismissed altogether, or needs to be substantially modified.

The paper is divided into three sections. In Section 1 I present the first horn of a dilemma: unless the BST is modified to index normal function against

<sup>1</sup> See, for example, (Daniels [1985]; Anand *et al.* [2004]; Cooper and Megone [2007]).

<sup>2</sup> See (Boorse [1975], [1976b], [1977], [1987], [1997]; Kass [1975]; Kendell [1975]; Scadding [1990]; Schramme [2007]; Szasz [1960]).

<sup>3</sup> See, for example, (Agich [1983]; Cooper [2002]; Engelhardt [1975], [1976]; Goossens [1980]; Kopelman [1975]; Margolis [1976]; Nordenfelt [1987], [2001]; Reznick [1987]; Whitbeck [1978]).

<sup>4</sup> See (Khushf [2007]). Boorse ([1997]) provides an almost complete discussion and bibliography of the first 20 years of criticism. For subsequent objections, see (Amundson [2000]; Cooper [2002]; Ereshefsky [forthcoming]; Kingma [2007]).

<sup>5</sup> For rudimentary versions of a similar argument, see (Nordenfelt [1993], pp. 90–1; Van Der Steen and Thung [1988], p. 90).

situations, it is unable to account for many of our dynamic physiological functions. I present a more sophisticated version of the BST that has the resources to accommodate this problem. This version, however, is subject to the second horn of the dilemma, which I present in Section 2: if functions are indexed against situations, then the BST can no longer account for diseases, such as infectious diseases or poisoning, that result from specific environmental factors. The BST is impaled on either the first or second horn of this dilemma, which is a genuine dilemma; unlike many previous objections, it does not rely on examples of disease that are exceptional, vague, or borderline. Instead it relies on typical examples of large groups of straightforward diseases that any credible account of disease would have to account for. In Section 3 I discuss and reply to potential responses to my argument. I demonstrate that the only way out of the dilemma entails a sacrifice of naturalism. This not only undermines Boorse's naturalistic account of *disease*, but also demonstrates that his naturalistic account of *normal function* does not work. The latter claim is entirely novel, and has implications that reach beyond the question of health and disease.

## 1 A More Sophisticated Version of the BST

The BST is presented in the following final version:

- (1) The *reference class* is a natural class of organisms of uniform functional design; specifically, an age group of a sex of a species.
- (2) A *normal function* of a part or process within members of the reference class is a statistically typical contribution by it to their individual survival and reproduction.
- (3) A *disease* is a type of internal state that is either an impairment of normal functional ability, i.e., a reduction of one or more functional abilities below typical efficiency, or a limitation on functional ability caused by environmental agents.
- (4) *Health* is the absence of disease. (Boorse [1997], pp. 7–8; see also Boorse [1977], p. 562.)

In other words, health is normal function, where normal function is the statistically typical contribution to survival and reproduction in a reference class. The general idea is that when I am healthy, I perform all the functions that are normal for my species, such as seeing, hearing, breathing, and pumping blood. When I am not healthy, one or more of these functions are impaired.

For the purposes of this paper, in which our analysis of the BST will already become unpleasantly technical and detailed, I will somewhat simplify the

BST. First, I will ignore reference classes, and pretend that a species just presents one reference class, or functional type, only.<sup>6</sup> Second, I will ignore how one determines whether something is an *adverse* departure from normal function, as opposed to a *beneficial* departure from normal function: having superior vision, for example, is a departure from the normal, but does not constitute disease, whereas inferior vision does. I shall pretend that on the BST *any* departure from normal function is a disease. This does not affect my argument.

I will now systematically run through the BST to explain it in detail.

### 1.1 Normal function

Boorse ([1976a], [2002]) defines a function as the causal contribution of a system to an overall goal. The goals of biological organisms are survival and reproduction.<sup>7</sup> To illustrate this account, take the example of a squirrel tail. A squirrel tail helps to balance a squirrel when it jumps across trees. This prevents the squirrel from dropping to the ground and thus makes a causal contribution to the squirrel's overall goals of survival and reproduction. The function of the tail is therefore *balancing*.

Function (F) = causal contribution to survival and reproduction.

Though *any* causal contribution to survival and reproduction is a function according to Boorse, for the purposes of accurately describing health not all of these functions are relevant; only *normal* or *species-typical* functions are. The normal or species-typical function of a subsystem is a causal contribution to survival and reproduction that a subsystem *usually* or *statistically typically* makes in the species ([1976a], [2002]). For example, kidneys typically filter blood, but they only occasionally contribute to the survival of their owner by generating large sums of money on the illegal organ market. Therefore, *filtering blood* is a *normal* or *species-typical* function of the kidney, which is relevant for health, whereas *generating revenue* is only an *accidental* function of the kidney, which is *not* relevant for health.

Health = *Normal* or *species-typical* function (NF)  
= *statistically typical* causal contribution  
to survival and reproduction.

<sup>6</sup> For an objection to the BST that is based on reference classes, see (Kingma [2007]).

<sup>7</sup> Schaffner ([1993]) has objected that these goals are not objective, thus violating one of the premises of Boorse's claim to naturalism. See also (Engelhardt [1986], pp. 167–71; Brown [1985], pp. 315–6). For a response, see (Boorse [1997], pp. 25–7, [2002], pp. 77–8).

## 1.2 Health as a quantitative normal function

Though the BST states that health is a normal function, merely performing a normal or species-typical function is not enough for health. In order to equate to health, normal or species-typical function must be performed *at the right level*: the heart, for example, must not merely pump blood, but it must pump blood at the right speed, pressure, etc. I call the former function ascription—pumping blood—the *qualitative* normal function of the heart, and the latter function ascription—pumping blood at the right level, speed, pressure, etc.—the *quantitative* normal function of the heart. *Quantitative* normal function is normal or species-typical function at *the right efficiency level*, which, in keeping with naturalism, must be defined statistically.

Health = Quantitative normal function (QNF)  
           = statistically typical causal contribution  
           to survival and reproduction with a  
           *statistically typical level of efficiency.*

There are thus two notions of normal, which are captured by Boorse's *normal function*. One refers to the *causal contribution* of a system that is statistically typical for the species (*normal species* function or *qualitative* normal function), and the other refers to the *rate or level* of that function that is statistically normal for the species (*quantitative* normal function). In Boorse's own definition of the BST, these two notions of normal are conflated, but I prefer to distinguish them. It is evident that the BST aims to capture both.<sup>8</sup>

So, to give an example, if we were to determine whether a token squirrel is healthy, we have to consider all its subsystems, and for each subsystem we should determine (i) which causal contributions this system statistically typically makes in most squirrel tokens (NF), (ii) whether this subsystem in this particular squirrel makes this contribution, and (iii) if it does, whether this subsystem performs this function with statistically typical efficiency (QNF). If every single subsystem of the squirrel does this, then, and only then, is the squirrel healthy.

Take, again, a squirrel tail, the function of which is balancing (NF). We must now check whether (i) this tail performs the function *balancing*, and (ii) whether this tail balances the squirrel within the statistically normal range of balancing efficiency for squirrels (QNF). If we find this tail is rather wonky and does not balance within the statistically normal range of balancing efficiency, then this tail is dysfunctional and this squirrel has a disease.

<sup>8</sup> See, for example, 'a disease [...] reduces one or more functional abilities below *typical efficiency*' (Boorse [1977], p. 562, [1997], pp. 7–8). (italics my emphasis.)

### 1.3 Dispositional function

Our detailed run-through of the BST has already become quite complicated. But the definition ‘Health is quantitative normal function’ is still far from the full definition of the BST. Two more aspects have to be added, the first of which is *dispositional function*.

As Boorse points out, ‘most biological functions are performed only on specific occasions’ ([2002], p. 93). At the moment, for example, my immune system is not warding off an infection; my legs are not transporting me; my mouth, tongue and throat are neither eating nor talking; and my esophagus is not transporting food to my stomach. What *is* happening is that I am breathing, my heart is pumping blood, my fingers are typing and some of my blood is forming a clot because I just cut my finger. It is clear that many of these functions are and should be performed on specific occasions only: swallowing and propelling food to the stomach happens when eating, blood clotting when there is a damaged blood vessel, immune activity when there is an infection, etc. Some of these occasions are currently present, others are not, and the corresponding functions are performed or not performed accordingly.

Now, for me to be healthy, to have quantitative normal species function, it is not merely enough that I perform my *currently active* normal species functions (breathing, blood pumping, typing and digesting) with normal efficiency. It is also necessary that, when the appropriate occasion arises, I have the ability to perform, with normal efficiency, all those normal species functions that are currently not performed. For example, my immune system and tongue are currently inactive. But if I am faced with an infection or with an opportunity to talk, these systems will (and should) become active and perform their appropriate quantitative normal species function, i.e., an immune response and speech. If these systems lack the ability to do this, I have a disease now.

Health therefore does not merely equal *actual* quantitative normal species functioning, i.e., the performing of all normal species functions appropriate for the *present* occasion with statistically typical efficiency, but, in addition, being healthy implies having *the disposition* to perform quantitative normal species function in a variety of non-actual situations, i.e., the disposition to perform in a range of situations all the normal species functions that are appropriate for those situations, with statistically typical efficiency. Boorse calls this *normal functional ability*, which he has ‘defined dispositionally, as the readiness of an internal part to perform all its normal functions on typical occasions with at least typical efficiency’ ([1997], p. 8). I prefer to label it *dispositional quantitative normal species function*.

The BST states that ‘Health is normal function(al ability)’  
 which means ‘Health is *actual* quantitative normal species function  
 + *dispositional* quantitative normal species function’  
 (H = AQNF + DQNF)

which equals

'An organism is healthy if and only if all of its subsystems perform in the current situation with a statistically normal level of efficiency all the causal contributions to survival and reproduction that are statistically typical for those subsystems in the species *and* all of its subsystems are disposed to perform in other situations with a statistically normal level of efficiency all the causal contributions to survival and reproduction that are statistically typical for those subsystems in the species'.

### 1.4 Situation-specific function

Having clarified the BST this far, by distinguishing normal species (qualitative) and normal efficient (quantitative) function, and by making explicit the dispositional nature of health, there is one further aspect of the BST that I need to elucidate: the *situation-specificity* of normal function. This is the aspect that Boorse is least clear about in his work, and this part of my analysis therefore may or may not be a modification of his theory.<sup>9</sup>

As established in the previous section, it is a given that 'most biological functions are performed only on specific occasions' ([2002], p. 93. See also Boorse [1977], pp. 561–2; Nordenfelt [1987], pp. 24–9). Note that some of these occasions, and their corresponding functions, may be relatively rare. Infections and wounds are relatively uncommon, for example, and so my immune system is neither mounting an immune response most of the time, nor are my platelets initiating blood clots most of the time. Whether and how these immune and blood clotting systems should perform their normal species function therefore varies from occasion to occasion: in most cases they should not perform their normal function at all. Platelets, for example, do, and ought to, spend most of their time floating around in an inactive state. Platelets that are not floating around in an inactive state cause diseases, such as deep vein thrombosis, lung embolisms, strokes and heart attacks.

This, however, creates a problem for the BST. Because Boorse has defined normal species function *statistically*—as the function that is statistically typical in the species—the BST is unable to account for some functions, such as blood-clotting, which are de facto *not* statistically typical in the species. Because broken veins are relatively uncommon, platelets statistically typically do *not* form blood clots. Therefore, Boorse's account of normal function as the *statistically typical* contribution to survival and reproduction cannot bear out

<sup>9</sup> See also footnote 11.

The BST therefore needs to be modified: normal species function, which equals health, is not something that is *generally* statistically typical. Instead it is the function that is statistically typical *in a certain situation or occasion*. For example, in order to arrive at the normal species function of platelets by statistical abstraction, one must abstract over situations where there are damaged blood vessels only; for only when we take the normal function of platelets to be the causal contribution to survival and reproduction that is statistically typical *in the situation of a damaged blood vessel* can the BST tell us that the normal function of platelets (in the situation of a damaged blood vessel) is to clot blood. If the BST abstracts over all situations, however, the normal species function of platelets would be inactivity. Normal, i.e., statistically typical, species function, which equals health, must therefore be indexed against specific situations.

This observation, that what is healthy (or normal) function varies from situation to situation, applies even more strongly to *quantitative*, or normal efficient, function than to qualitative function. For example, the normal ranges of heart rate and cardiac output are very different on the occasions of sleeping (when both are low), and the less common occasion of strenuous exercise (when both are very high). Normal ranges of insulin production, glucose absorption and glycogen synthesis are very different depending on whether and what a person is eating and/or doing. Therefore, what the normal, healthy, correct or appropriate *quantitatively* normal level for a specific function is depends on the situation or occasion too. This means that, as we just established for qualitative function, the BST can only give an accurate *statistical* account of *quantitative* normal function, or health, if it does the statistics for different situations separately.

The BST must take situation-specific function on board. Thus, if we take the examples of squirrels one last time, then if we are to establish whether a

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particular squirrel is healthy, we must consider for each subsystem whether it (i) makes all contributions to survival and reproduction that that subsystem statistically typically makes in squirrels in the particular situation that the squirrel is in, and (ii) whether it does so with a level of efficiency that falls within the statistically typical range of efficiency levels for the situation of the squirrel. Then we must establish for each subsystem, for a variety of situations, whether it is (iii) disposed to make all the contributions to survival and reproduction that that subsystem statistically typically makes in each of those situations, and (iv) whether it is disposed to do so in each situation at a level of efficiency that falls within the statistically typical range of efficiency levels for that situation. If the squirrel ticks all these boxes, then, and only then, is the squirrel healthy.

### 1.5 Summary and justification

Here is a summary of the improvements I have made to the BST:<sup>11</sup>

The BST needs to

- (i) distinguish between *qualitative* normal function and *quantitative* normal function. The qualitative normal function of the heart is to pump blood, and the quantitative normal function of the heart is to pump blood at a certain rate, with a certain output, with regularity, etc.
- (ii) employ *situation-specificity of functions*: a function is only normal or abnormal relevant to a specific environment or situation. For example, a heart rate of 60 bpm is normal during sleep but abnormal during exercise.
- (iii) employ *dispositional function*: to be healthy includes not only functioning normally with respect to the current environment, but also the *ability* to function normally with respect to other environments. For example, a heart with a resting heart rate of 60 bpm that is not *able* to beat faster during exercise is not healthy, irrespective of which situation it is presently in.

<sup>11</sup> I consider these improvements but they are not all modifications; some of the improvements is merely in the presentation: Boorse is explicit that health equals normal *functional ability*—dispositional function—in his final definition of the BST ([1977], p. 562, [1997], pp. 7–8), but fails to see the consequences of this for the situation-specificity of function. (See also his discussion in [1977], pp. 561–2). And though the situation-specificity of function is my contribution—and only implicitly derives from Boorse's work as a direct result of dispositional function—Boorse either does or would have to agree with it. In response to objections he writes: '[t]hough I did not stress the dynamism of normal physiology in presenting the BST, I always assumed it' ([1997], p. 79). Boorse therefore acknowledges that normal function is relative to situations, but simply fails to work out the consequences of this for the BST. Finally, the distinction between qualitative and quantitative function is a clarification only; Boorse explicitly mentions *typical efficiency* in his final definition ([1977], p. 562, [1997], pp. 7–8. For a discussion, see [1977], p. 559). All of this has been so consistently overlooked in the literature, however, that I deemed it necessary to expound in detail.

Though these improvements may seem to make the BST unnecessarily complicated, they are necessary if the theory is to give an accurate account of dynamic physiological function. In particular, Boorse cannot afford to take my counter-examples on board as anomalies or exceptions, for they are not: organisms are dynamic and responsive, and so are their internal systems. Blood pressure, cardiac output, breathing and sweating are all very responsive to physiological demand, and maladjustments constitute well-known and typical diseases, such as high blood pressure, heart failure and dyspnea. Hormonal systems are tightly regulated, with the actual level of hormonal production being highly variable depending on the time of day (melatonin, ACTH), time of the month or duration of a pregnancy (female reproductive hormones), physical levels of activity and stress and/or external threats (cortisol, adrenaline), and internal metabolic requirements (insulin). Maladjustments can have fairly dramatic consequences and they nearly always constitute a departure from health. Examples of these are Cushing's syndrome, Addison's disease, infertility and diabetes. All of these are archetypical cases of disease.

It is a constraint on any credible account of disease that it can capture the archetypical cases. An account of disease that cannot meet this constraint, i.e., the unimproved BST, must be rejected. The improved BST must therefore be adopted instead.

## 2 An Inescapable Problem

In the previous section I presented a problem for the BST: without determining normal functions separately for each situation, the BST cannot account for many of our dynamic physiological functions and hence cannot accurately distinguish health and diseases. To avoid this problem, I presented an improved version of the BST, and of Boorse's account of function, which is committed to situation-specific functions. Situation-specific normal functions are the quantitative causal contributions that are statistically normal in particular situations.

In this section I present a problem for the improved version of the BST: if functions are situation-specific, i.e., determined separately for separate occasions, then the BST cannot account for diseases that are the direct result of specific situations or occasions. Together these two problems form the horns of an inescapable dilemma. If the BST does *not* adopt situation-specific statistics, it is subject to the first problem, described in the previous section. If the BST *does* adopt situation-specific functions, it falls prey to the second problem, described in this section. Whichever way it goes, the BST cannot give an accurate account of health and disease. Moreover, the BST is systematically unable to answer this objection, because of the following central tension: the *disposition* to become ill when poisoned is compatible with health, but *actually*

being poisoned constitutes a disease. Since the BST uses the same formula for actual and dispositional function, it is not able to tease out this difference.

## 2.1 Harmful environments and situation-specific diseases

There is a set of diseases that are directly caused by an external or situational factor, such as a *harmful environment*, a specific action, occasion or situation. I will call these diseases *situation-specific diseases*. What makes these diseases interesting to us is that they are *statistically normal* in their respective specific environments or situations; every organism or nearly every organism in this environment or situation would end up with the disease.

These diseases pose a problem for our improved, situation-specific BST: an organism that has such a situation-specific disease, i.e., an organism that is in such a *harmful situation*, neither has a statistically *abnormal* function for that situation, nor a set of dispositions or functional abilities that is *abnormal* for that situation. Therefore, although we want to call these conditions *diseases*, it seems that the BST has no way of doing so.

Take, for example, cases of taking poison or drugs. Anybody who overdoses on paracetamol 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 the situation of overdosing on paracetamol. According to the situation-specific BST it therefore must be healthy, but according to our intuitions it is clearly a disease. Another example is carbon monoxide poisoning, in which there is a reduction in hemoglobin's capacity to bind oxygen. This reduced function is, again, statistically typical in the situation of exposure to high levels of carbon monoxide. It is therefore healthy according to the situation-specific BST, but not according to our intuitions.

## 2.2 A detailed example

I will now give a more detailed example to illustrate this problem. Consider an individual on four specific occasions, and focus on her digestive subsystem, the function of which, broadly, is to digest food. The *first* occasion is whilst relaxing after a meal. In this situation all digestive systems contribute to survival at maximum capacity. The *second* occasion is after a period of fasting. In this situation not much food resides in the digestive system and little blood is flowing there. The digestive system is fairly inactive. The *third* occasion is in the middle of a lengthy exercise session. In this situation little blood flows to the digestive system because most blood flow is directed to the skeletal muscles. The digestive system is virtually dormant. The *fourth* occasion is after ingesting a specific poison that immobilizes the digestive system (but has no other effects).

In this situation little blood flows to the digestive system, which is virtually dormant.

Although the normal species function, i.e., the typical causal contribution to survival and reproduction, of the digestive system is *digesting food*, we must understand this function to be relative to specific situations: on occasion two, where there is no food to digest, the digestive tract is not ill or dysfunctional because it is *not* performing the function of food digestion, or because it is not performing this function at the *usual* quantitative rate. Instead the digestive tract is healthy, because it makes the normal contribution to survival and reproduction (doing very little) for its present situation (there being no food in the digestive tract).

According to the same analysis, the digestive tract on occasion three—prolonged heavy exercise—is also healthy. Although this digestive system does not perform its normal function—digesting—currently, this is the normal contribution to survival and reproduction of the digestive tract on the occasion of heavy exercise. Moreover, the digestive tract in the person doing heavy exercise is still (after some rest) disposed to make its normal contributions to survival and reproduction on the occasion of resting after a meal (digesting very actively) or on the occasion of resting after fasting (not doing much at all). The digestive tract is thus healthy.

By the same analysis, however, 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. That, however, is a straight contradiction of both our intuitions and our expectations of an account of health and disease.

### 2.3 Two possible replies refuted

There are two quick responses to the above example, and neither works. (I will consider other possible responses in the next section). The first, intuitive, reply is that the poisoned digestive tract *must* be diseased because it is not performing its normal function, which is digestion. But if that is the answer, then by the same analysis the digestive tract of the person who is performing heavy exercise, and even that of the person who is fasting, is also diseased: on these occasions the digestive tract is equally not performing its function (digestion) as well as it could. If the BST must give up on allowing subsystems to sometimes perform no function, then any subsystem is ill *whenever* it does not perform its

function as well, or as actively, as it could. If this is the answer then the BST is impaled on the first horn of the dilemma, described in the previous section: only if the BST employs an account of function that takes note of what is normal in a particular situation—a situation-specific account of function—can it be an accurate account of health and disease in dynamic biological systems.

A second response might be that there *is* an important difference between the runner and the poisoned person, which makes the lack of digestion healthy in one and unhealthy in the other. This is that the lack of digestion in the *runner* is easily reversible, whereas the inability to digest in the case of being *poisoned* is *not* so easily reversible; if the runner sits down for a meal she will start digesting pretty quickly. But if the poisoned person does, she might, depending on the poison, take a very long time to clear the poison and start digesting—or she may never be able to. Reversibility is the crucial difference between health and disease.

Not all slowly reversible or irreversible situation-specific normal functions are diseases, however. Good or neutral effects of exposure to a specific environment may also have a lasting effect. For example, calcium deposits in bones, a tan, and the effect of cardiovascular exercise take much longer to clear than the effects of carbon monoxide poisoning, a bruise, a cut, or a small infection. At the same time some environmental diseases may be lifted almost instantaneously if the environment is changed, as is the case in suffocation because of a lack of oxygen. Therefore this is not a compelling response.

## 2.4 Conclusion of Section 2

The BST faces an inescapable dilemma: *without* situation-specific function it fails as an account of most of our dynamic physiological functions and cannot account for the diseases mentioned in Section 1. Therefore, in order to make sense of dynamic physiological functions, the BST must adopt situation-specific statistics. But *with* situation-specific function it has trouble accounting for diseases that are the normal (i.e., species-typical) reaction to a certain environment, occasion or external factor: the situation-specific diseases discussed in this section.

It would be too high a price to pay for Boorse to concede that the BST is unable to account for situation-specific diseases. The examples of situation-specific diseases that I discussed, i.e., infectious diseases, traumata, vitamin deficiencies and poisoning, are not borderline or exceptional cases of disease. Instead they are typical examples of straightforward somatic diseases. It must be a constraint on any credible account of disease that it can accommodate these, as an account of disease that cannot is simply too far removed from medical theory and practice. But since a concession on the first horn of the dilemma, presented in the previous section, was also not an option—as this

would therefore render the BST unable to account for dynamic physiological functions and disturbances —the BST is truly stuck. Unless we can come up with a way out of this dilemma, the BST must be rejected.

### 3 Potential Ways out of the Dilemma

In this section I examine whether the BST could somehow find a way out of the dilemma. I then address some potential objections to my argument and conclude that there is no way out of the dilemma that is compatible with naturalism.

#### 3.1 Distinguishing between harmful and normal situations

The dilemma for the BST is as follows: *without* situation-specific function it cannot account for dynamic physiological functions and disturbances thereof, but *with* situation-specific functions it cannot account for diseases that are statistically normal in, or the direct result of, a specific environment. There might, however, be a way for the BST to avoid the second horn of this dilemma. Let's label environments that normally cause disease *harmful environments*. Now if the BST had a way of distinguishing between *harmful* and *normal* situations and environments, then it could adopt a clause stating that health is a statistically normal situation-specific function *unless* the present situation is a harmful situation; in a *harmful situation* the statistically normal function is a disease.

For this modification to work we need an adequate account of what it is to be a harmful environment, situation or external factor. We have an intuitive grasp of this: harmful environments and situations are the situations and environments where diseases are statistically normal, such as environments/situations high in microbes, carbon monoxide, or paracetamol, or low in essential nutrients. This is a circular definition of harmful environments/situations: we need to define a harmful environment to give an account of disease, so we cannot appeal to disease to give an account of what a harmful environment is. I will now consider several non-circular ways in which one might propose to distinguish harmful situations from normal situations, and demonstrate that each of them does not work. One important criterion here is that, given that the BST is a naturalistic account of health, any acceptable definition of a harmful situation must be naturalistic. As I will demonstrate, such a naturalistic solution is not possible.

#### 3.2 First solution: Statistically abnormal environments

Boorse both acknowledges a version of the problem I present, and treats it as fairly harmless:

if the BST admits that a 150/min heart rate, though statistically atypical, is normal on the occasion of exercise, or sweating on the occasion of hot

weather, as I always supposed, then every reaction of the organism will be normal on the occasion of whatever provokes it [. . .].

*The most* this line of argument could establish is that Daniels ([1981], p. 156n) was right that the BST must introduce a concept of normal environment. [. . .] But it can easily define one in its usual value-free statistical way. (Boorse [1997], pp. 83–4, italics my emphasis).

Boorse goes on to propose that a function ‘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], p. 84).<sup>12</sup> This solution equates *harmful* environments to *rare* environments and entails that all normal functions in rare environments are diseases. I will examine two sets of examples to show that this solution does not work and that *rare* environments do not equate to *harmful* environments (i.e., environments in which statistically normal function is a disease). The first group of examples are rare environments, situations or external factors in which the statistically normal function of a subsystem is *not* a disease. The second group of examples consists of *non-rare* environments, where statistically normal function *is* a disease.

### 3.2.1 Rare non-harmful environments

My first example is that of a spermatozoon that finds itself in the oviduct in the proximity of an ovum. This is a *very* rare environment for a spermatozoon to be in; most spermatozoa never leave the male body or make it past a cervix. The statistically normal causal contribution to reproduction of a spermatozoon in this *extremely* rare environment, however, is to fertilize the egg. Now according to Boorse’s proposal, which equates rare environments to harmful environments, fertilization ought to be a disease because it is the statistically normal function of sperm in a rare, and therefore harmful, environment. This cannot be the right result.

A second example of a rare-but-not-harmful environment is complete and utter pitch-black darkness (we are nearly always exposed to some light at least). The normal function of the eyes in this environment is to do nothing at all.

<sup>12</sup> In actual fact, Boorse writes that ‘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 [. . .]’ ([1997], p. 84, italics my emphasis). But since a ‘statistically species-subnormal function’ was *already* pathological according to Boorse’s original definition, this rephrasing of his definition adds nothing new. In particular it does not solve the problem of statistically *normal* functions that are nevertheless diseases because they occur in harmful environments. I therefore interpret Boorse to have meant the following: ‘the BST needs to add a rule that statistically species-normal function is pathological if it directly results from an environmental factor outside an arbitrarily chosen central statistical range of that factor in the environments where the species lives.’ This interpretation, I think, both does justice to Boorse’s intent and is the most promising possible reply to my argument.

This means that on Boorse's proposal eyes are diseased in pitch-black darkness, which does not agree with our intuitions.

What these examples, contra Boorse, demonstrate is that being rare is not *sufficient* for being harmful. I shall now give a second set of examples, which demonstrate that being rare is also not *necessary* for being harmful.

### 3.2.2 Harmful non-rare environments

A first example of an external factor that is harmful but not rare is UV radiation—or sunlight. My normal response to a couple of hours of summer sunshine is to burn quite badly. I share this normal response with a sizeable part of my (light-skinned) reference class—yet burns are undoubtedly a disease. Boorse, I suppose, does not want to say that having a light skin is a disease; it is a prime example of a normal *polymorphism*.<sup>13</sup> But he also does not want to say that a few hours of sunshine is a very rare environmental factor; not only are some normal external factors more rare, but also if a few hours of sunlight is a rare environmental factor, then the other normal response to this factor—which in some people more lucky than me is tanning—must be a disease too. This cannot be right. This example does not just demonstrate that an environment can be harmful without being rare; it also shows that whether an environment is harmful depends on the organism.

A second example is formed by skin traumata. I bruise and cut myself many, many, many times a year, so things knocking into me are not exactly rare external factors. But my normal response to these incidents, i.e., bruises and cuts, are diseases.

A third and rather unfortunate example is that a substantial part of the world population is exposed to harmful environments on a regular basis: those transmitting malaria or causing diarrhea, and those deficient in essential nutrients. Children, especially, are exposed to malnutrition on a large scale. Although these are not *omnipresent* environmental factors, they are, unfortunately, not extremely rare either. According to Boorse's proposal they should therefore not cause disease, but it is clear that they do, and that we want to be *able* to say that they do.

We can conclude that it simply is not the case that the distinction between *harmful* and *non-harmful* environments aligns with the distinction between *rare* and *non-rare* environments. Both distinctions are obviously somewhat vague, but even granted vagueness it is not clear that their vague boundaries are related in some way. Boorse's proposal that harmful environments equate with rare environments must therefore be rejected.

<sup>13</sup> See (Boorse [1977], p. 558). *Polymorphisms* are normal variations in, for example, hair color, skin color or blood type.



### 3.3 Second solution: Adverse environments

A better, and more intuitive, solution to the problem of distinguishing harmful and non-harmful environments might appeal to the *consequences* of a situation or environment for the organism: harmful environments and situations are environments and situations that affect us adversely, that are *bad* for us. Such a proposal deals with most of the above examples, such as, for example, the fertilization example. Although fertilization is rare, it is not, generally, a bad thing. Therefore, being around a fertile ovum, though rare, was not a harmful situation for the spermatozoon to be in, and hence not a disease.

In order for this solution to save the BST, however, we must show (i) that we can determine in a non-circular and *naturalistic* way whether an environment or situation is bad for us, and (ii) that every environment that is *harmful*, i.e. disease-causing, is also bad for us (and vice versa). The first criterion immediately disposes of two potential ways of defining *bad situations*, the first of which is *disease-causing*. Since we aim to define disease, defining *environments that are bad for us* as *disease-causing environments* would beg the question. The second is to give a non-naturalistic definition of bad situations; for example, situations that interfere with well-being or flourishing, or situations that we disvalue. These non-naturalistic definitions may or may not define harmful situations accurately, but since our attempt is to give a *naturalistic* account of disease, I do not consider them here.

The second criterion should be kept in mind when we consider a possible naturalistic account of adversity: an adverse environment or situation is an environment or situation that reduces the organism's overall chances of survival and reproduction. Now, consider the following counter-example: UV radiation may normally cause sunburn in me and most of my reference class, but I also need sunlight to synthesize vitamin D; vitamin D deficiency causes rickets and rickets is a much worse disease than sunburn. Therefore, although sunlight might be a harmful, i.e. disease-causing environment (for some, not others), it is not one that reduces survival and reproduction, i.e. it is not a bad or adverse environment. Now consider certain environments full of microbes; some infectious children's diseases have a positive overall effect on survival and reproduction because they sensitize the child's immune system. Hence the environments in which these diseases arise do not reduce the child's overall chances of survival or reproduction. They are therefore not bad environments, but they do cause disease, so they are harmful. Finally, many minor diseases simply do not reduce one's chances of survival and reproduction at all; examples are minor traumata or the common cold. Therefore, the proposal that harmful environments are environments that are bad for us does not work: the distinction between *harmful* and *non-harmful* environments and situations

is not the same as the distinction between environments that reduce and that do not reduce an organism's chances of survival and reproduction.<sup>14</sup>

### 3.4 Third solution: Non-natural environments

A third possible solution consists of Daniels' ([1981], p. 156n) original proposal: harmful environments are non-natural environments. But as in any appeal to *natural* it is not quite clear what this means—and none of the possible options is likely to work. Natural environments are clearly not environments that occur in nature, because both harmful and non-harmful environments occur in nature. Since Daniels himself acknowledges that natural environments include social environments, 'natural environments' can also not designate 'environments that are not interfered with by humans'. An evolutionary approach to natural environments, finally, does not generate the desired distinctions either: most of our current non-harmful environments were not part of our evolutionary history, and our evolutionary history includes environments that are harmful. So Daniels' response also fails to solve the problem I presented.

### 3.5 Interim conclusion and diagnosis

I presented a dilemma: *without* situation-specific function, the BST cannot account for dynamic physiological function, but *with* situation-specific function, the BST cannot account for situation-specific diseases, i.e., diseases that are statistically typical in a certain situation or environment. The BST might be able to avoid the second horn if it could give a non-circular, naturalistic, account of harmful environments, i.e., environments that typically cause disease, but the three possible ways of defining harmful environments that I examined did not work; neither being *statistically uncommon* nor being *unnatural* is either necessary or sufficient for being harmful, and a naturalistic account of an *adverse* environment cannot be given. Therefore this solution fails.

There are two further, independent, reasons why an appeal to harmful environments will not generate an adequate account of disease. The first reason is that it is not specific; according to such a solution *any* bodily function in a harmful environment would be a disease. Consider, for example, our basic example of ingesting a poison that immobilizes your digestive system. According to the harmful environments proposal, the only means by which the BST could designate the poisoning a disease and the disabled digestive system dysfunctional is stating that ingesting poison is a *harmful external factor*. But that entails that *all* situation-specific statistically normal contributions to survival and reproduction in the situation of taking this poison would be diseases. This entails not only that the *digestive system* is dysfunctional—which is our desired

<sup>14</sup> See also (Schwartz [2007]).

result—but also that the eyes, brain, limbs, kidneys, and heart, which are all completely unaffected, are. All of these would count as having a dysfunction because they perform situation-specific statistically normal function in a harmful environment. Therefore, even if we found an adequate way of distinguishing harmful and non-harmful environments, the BST would not give an adequate account of health and disease, or of dysfunction.

The second reason that this proposal cannot provide an accurate description of our medical concepts is that within a harmful environment or (pathological) situation, medicine distinguishes between the normal and the (further) pathological. For example: an infection, such as polio, can be an environmentally caused, i.e., situation-specific, disease. But medicine distinguishes between a normal immune response and a pathological or absent immune response to an infection. This important distinction within medicine would not be possible on the harmful environments solution.<sup>15</sup> Distinguishing between harmful and non-harmful environments can therefore not save the BST from the second horn of our dilemma.

### 3.6 Abusing the function concept?

Before I conclude this paper I would like to consider two objections one might raise against the argument I have presented.<sup>16</sup> The first of these is that I abuse the notion of function; many of the examples I give seem to constitute statistically normal *responses* and not statistically normal *contributions to survival and reproduction* in a situation. For example, the inability of hemoglobin to bind oxygen, which occurs in carbon monoxide poisoning, may be considered a *response*, rather than a function. Equally, sunburn may be considered a *response* of the skin to UV radiation, rather than a function.

This objection is not compelling. Although it is true that I have sometimes used the shorthand ‘response’ instead of ‘causal contribution to survival and reproduction’, it remains the case that in all of these examples there is still *some* contribution to survival and reproduction. For example, a *reduced* level of liver function and a *reduced* contribution to survival by the digestive system are still examples of *some* contribution to survival and reproduction. They are thus examples of *functions*. Since an accurate account of biological function must allow for systems’ often performing very little function, and even on occasion performing no contribution to survival and reproduction whatsoever, I have not been inconsistent with Boorse’s analysis of function in calling these contributions to survival and reproduction, however minor they are, functions.

<sup>15</sup> For an early version of the objection that the BST has little to tell us about healthy and pathological immune responses, see (Nordenfelt [1987]). For a reply see (Boorse [1997], pp. 84–6).

<sup>16</sup> I am grateful to Christopher Boorse for suggesting these to me.

Second, goal-oriented systems, such as a squirrel, show several levels of functional organization that form ‘a very complex hierarchy of systems and subsystems, ascending from the level of genes through organelles, cells, tissues, organs and organ systems to gross behavior. Each level’s output is directed to goals that serve as input to the next’ (Boorse [2002], p. 70). One might argue that the problem I present in this section only arises because I fail to consider the hierarchical organization of functions.

This argument would go like this: in the case of situation-specific diseases I discussed, i.e., diseases that are statistically normal functions in harmful environments, some *higher-order* function is impaired, which is not impaired in the case of statistically normal functions that are *not* diseases, i.e. statistically normal functions in non-harmful environments. For example, if we consider the case of insulin production, the normal range of insulin production varies depending on external factors such as glucose intake. But the higher-order function of maintaining blood glucose, to which insulin production contributes, is kept constant in these normal situations. In the case of disease or harmful situations, however—if we had a poison that would induce diabetes, for example—the higher-order function of blood glucose level maintenance is also impaired. Thus, higher-order function marks the difference between health and disease.

The reason that this objection does not succeed is that even higher-order functions are relative to situations: most of the time my body makes no current contribution to reproduction, for example, which is the highest level of higher-order function. Nevertheless, this is not a disease. Also, there is not always an impairment of higher-order function in diseases that are directly caused by an external factor. In minor skin traumata, for example, and small infections, nearly all the higher-order functions go on as they do normally but we nevertheless consider these conditions to fall under the umbrella term ‘disease’. Therefore, I do not abuse or misunderstand the function concept.

#### 4 The BST Refuted

In this paper I have presented a two-horned problem for the BST. The theory cannot deal with this dilemma unless it gives up naturalism, but this is not an option for its main proponent. First, I demonstrated that, in order to accurately describe health and disease, the BST must index the statistically normal function against specific situations. I then presented a dilemma: *without* situation-specific function, the BST cannot account for dynamic physiological functions and diseases that are disturbances thereof, but *with* situation-specific functions the BST cannot account for diseases that are statistically normal in certain situations and environments (so-called situation-specific diseases). The BST is impaled on either horn of this genuine dilemma; a concession on either horn would be too high a price to pay for any account of disease, as both horns

relied on examples of typical diseases, the accurate designation of which is a constraint on any credible account of disease.

#### 4.1 A central tension

There is a particular feature of the BST that both prompts the dilemma I presented, and reveals it to be inescapable.<sup>17</sup> We established that my being healthy is not merely a fact about my present state of functioning, but also depends on facts about my functional dispositions in a range of non-actual situations. This is both intuitively plausible and, as I demonstrated, necessary to make sense of a whole range of diseases. It is also the case that a set of normal dispositions, i.e., dispositions that constitute health, always includes *some* dispositions to become ill in certain situations. For example, the fact that I *would* become ill if poisoned does not reveal that I am ill *now*. Together these facts constitute a central tension in the BST: the *disposition* to become ill when poisoned is compatible with health, but *actually* being poisoned constitutes a disease. Because the BST uses the same, statistical, formula to define both *actual* and *dispositional* function, it is never able to tease out this difference: the BST in its current form is committed to either diagnosing *both* actual and dispositional function in response to poison as a *disease*, or to diagnosing both as *health*. Neither outcome is compatible with an adequate account of health and disease: in the former case we would all have a disease permanently and in the latter case the BST cannot account for situation-specific diseases. Therefore, unless the BST is radically adjusted to define *dispositional* function (or functional ability) in a different manner from *actual* function—which seems to entail giving up on Boorse's statistically normal account of function—the BST is systematically unable to give us an adequate account of health.

#### 4.2 Differences with previous arguments

There are three elements that distinguish this argument from most previous objections to the BST. First, the argument is general. It does not rely on a few obscure and specific counter-examples, on vague, borderline cases, or on normativist intuitions. Instead it relies on entire classes of medically straight-forward, somatic diseases that the BST in one form or the other cannot account for. It is a constraint on any account of disease that it accurately captures these examples.

Second, my argument does not rely on a superficial, simplified interpretation of the BST. Instead I have given the most thorough analysis and charitable interpretation of the BST to date. If this version of the BST cannot give an accurate account of biological (dys)function, no naturalistic version of the theory can.

<sup>17</sup> I am grateful to Tim Lewens for helping me clarify this point.

Third, the argument presents a novel set of desiderata for disease-as-dysfunction accounts and accounts of biological function in general. I demonstrated (i) that normal biological functions are situation-specific, and (ii) that an account of (dys)function able to support an account of health and disease must specify *quantitative* rather than qualitative function; it must specify the normal function of the heart as quantitative ranges of pumping capacity, rather than as pumping blood simpliciter. These desiderata are a novel contribution to the philosophy of biology, and could have important implications for the literature on functions especially.

### 4.3 Conclusion

The BST is and has been the dominant example of a *naturalistic* account of disease, i.e., a definition of disease in value-free terms, for the past 30 years. Its failure to give a naturalistic definition of dysfunction that is compatible with medical function ascriptions is therefore bad news for naturalists. Whilst we might save the BST and hence disease-as-dysfunction accounts by adopting non-naturalistic elements, that won't save the naturalist's *claim*. This claim—that health and disease are value-free—can only be saved if we find an alternative naturalistic definition of medical function. Such definition must be compatible with the desiderata presented in this paper, i.e., it must provide an adequate account of the situation-specificity of function, of dispositional function, and of quantitative function. The challenge for naturalists now is to provide us with such an alternative account. Meanwhile I hope to have demonstrated a decisive reason, finally, to conclude that the best and only presently existing naturalistic account, the BST, is inadequate, both as a naturalistic account of dysfunction *and* as a naturalistic account of disease.

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