This essay provides an account of how to distinguish between health and pathology of trait tokens in medical theory. It (1) proposes to distinguish between two health/pathology concepts—health/pathology pertaining to survival and health/pathology pertaining to reproduction. It (2) defines measures for survival-efficiency and reproduction-efficiency of performances of physiological functions. It (3) provides an account of how, using the efficiency measures, to draw the line between health and pathology. The account draws, but seeks to improve, on Christopher Boorse’s biostatistical theory. In relation to that theory, the suggested account has the advantages (1) that it defines efficiency and (2) that it harmonizes with judgments in medical theory in cases of common diseases and “normal aging.” Furthermore, the essay argues against a competing idea of how to improve on the biostatistical theory, advocated by Peter Schwartz and Daniel Hausman.

**Keywords:** biostatistical theory, efficiency, health, pathology, philosophy of medicine

I. INTRODUCTION

How well tokens of a certain trait type work vary among individuals. We clearly consider some trait tokens to be working so poorly that they are pathological. Now, what determines the line between health and pathology of trait tokens? The purpose of this essay is to provide an account of this.\(^1\) The idea is roughly that a trait token is pathological if and only if at least one of its performances of a physiological function has a particularly low efficiency.

The proposed account deals with two problems of Christopher Boorse’s biostatistical theory (the BST), which has been argued to be the most worked
out and promising account for answering how to draw the line between health and pathology (Schwartz, 2007). The BST says that, for every trait type in a reference class, the trait tokens performing with untypically low efficiency are pathological. However, the BST is not without problems. First, there are issues about how to account for efficiency. Second, there are problems concerning where, among the possible levels of efficiency, to draw the line distinguishing health from pathology.

What is the point of defining “health” and “pathology” in medical theory? My overall motivation for pursuing this project is about theoretical consistency and clarity. Intuitions about health and pathology seem to involve both some descriptive biological component and some normative component. However, as for practical concerns, for example, for answering questions such as which states should and which states should not be treated by public health care, the biological component does not always seem relevant. As for more scientific concerns, the normative component seems not always relevant. Hence, we have a tension between intuitions about descriptive biological features and intuitions about normative features. What I intend here is to focus only on the biological, descriptive part: how may we consistently define a purely theoretical distinction between health and pathology? For the philosopher of biology, this is interesting in its own right. For researchers in medicine, which are interested in practical implications, this is not directly relevant, though. Yet, my hope is that, in the long run, the spelled-out contrast between biological and normative features may also help to enlighten and clarify the reasoning about what does and what does not matter when dealing with practical questions.

The essay is structured as follows. Section II presents how the BST distinguishes between health and pathology and presents two problems with this account. Section III develops a different account of how to distinguish between health and pathology and explains this account’s advantages over the BST. Section IV argues against a competing idea of how to improve the BST, as advocated by Peter Schwartz and Daniel Hausman. Section V adds some concluding remarks.

II. THE BIOSTATISTICAL THEORY

By the BST (e.g., Boorse, 1977, 1997, 2014), Christopher Boorse aims to define “health” and “pathology” in medical theory. First, let us look at this theory. Then, let us consider two problems, or challenges, that it meets.

The Theory

First, it should be clarified what Boorse sets out to do by the BST. By the BST, Boorse aims to account for health and pathology as biological phenomena referred to in theoretical medicine, that is, physiology and pathology. The
concepts analyzed are not special for human beings; they apply also to animals and plants. Given the aim of the BST, facts about how “pathology” and “health” are used by ordinary people are uninformative. However, while physiologists and pathologists’ usage of the concepts plays an important role in evaluating the account, Boorse’s project is not purely descriptive. Rather, it is explicative—it seeks to improve on extant usage (Boorse, 1997, 53; 2014, 713). The BST is value free; it has no normative component parts and no normative implications by itself. Hence, it cannot be used (in isolation) to answer practical questions, for example, whether a certain physiological state is good or bad or whether it should be treated. The theoretical concepts of health and pathology are thus to be clearly distinguished from other possible concepts of health and pathology, for example, clinical, social, and political concepts (see, e.g., Boorse, 1997, 45–6).

Let us now turn to the content of the theory. The BST is built on three major components: (i) reference classes, (ii) physiological functions, and (iii) efficiency levels. Let us start with reference classes. A basic idea of the BST is that health and pathology are relative to reference classes. For example, when determining the health or pathology in a newborn human male baby, it is relevant to take into account the functioning of other newborn human male babies, but not the functioning of 40-year-old human females or of newborn giraffes. Boorse describes reference classes as “natural class[es] of organisms of uniform functional design”. He takes them to be individuated by species, sex, and age (Boorse, 1977, 558, 562).

Next, consider the second component—physiological functions. What the distinction between health and pathology concerns, according to the BST, is the physiological functioning of organisms. Boorse defends a goal-analysis of the concept of function, according to which the function of a part is determined by its contribution to the goals of the system of which it is part (Boorse, 1976). What the relevant goals are and what the relevant system is depends on the context. In biology, the relevant goals and system are determined by the interests of the biological subdiscipline. Boorse says that “[i]n physiology the goal-directed system […] is the individual organism and the relevant goals its own survival and reproduction” (Boorse, 1976, 84). It may be clarified that a physiological function need only contribute to survival or reproduction (Boorse, 2014, 685). However, even if a trait token’s performance of a function contributes to one, or both, of these goals, it need not be a performance of a physiological function. On Boorse’s view, physiological functions are primarily assigned to types, rather than tokens. Boorse says: “[w]hatever contributes to these goals [individual survival and reproduction] reliably, throughout a […] reference class, is assigned a physiological function” (Boorse, 1976, 84, my emphasis). For example, since hearts typically pump blood, and, by doing so, typically contribute to their bearer’s survival, a physiological function for hearts is to pump blood. A heart token might perform some further function that also contributes to the bearer’s
survival or reproduction. However, if this function is unique for this individual, it does not count as a physiological function. Also, although Boorse does not explicitly say so, it seems to follow from Boorse’s type-relative view of physiological functions that a trait token which does not carry out any physiological function may still have a physiological function given to it by its membership of a trait type. For example, if the blood pumping of hearts contributes to individual survival and reproduction reliably throughout a reference class, then even a heart token in the reference class that does not pump any blood has the physiological function of pumping blood.

Now, let us turn to the third component of the BST—efficiency levels. Physiological functions can be performed more or less efficiently. It is by comparing the efficiency levels of the performances by different trait tokens that the BST draws the line between healthy and pathological trait tokens. By looking at the performances of the tokens of a trait type in a reference class, we may describe normal, or typical, efficiency levels. For example, in a reference class of human 7- to 9-year-old females, there is a (range of) typical efficiency level(s) for their hearts’ blood pumping. For a trait token to be healthy, it must have a disposition to perform each of its physiological functions on typical occasions with at least typical efficiency.3 Otherwise, the trait token is pathological (Boorse, 1977, 558–9, 562). Exactly where, along the range of efficiency levels, to locate the line distinguishing health from pathology is a matter of convention (Boorse, 1977, 559). However, Boorse (1977, 559; 1997, 8) says that the distinguishing line must be drawn a certain distance below the average efficiency level, and Boorse (2014, 684) says that it must be drawn far below the average efficiency level.

What does efficiency mean? This is not clearly stated by Boorse. But there are at least two different rough ideas that one could discern here. One natural interpretation of Boorse (1977, 559) is that the efficiency level of a trait token’s performance measures how close the performance comes to some “optimal performance” (e.g., for the thyroid, to secrete the optimal amount of hormones for current metabolic needs). In contrast, a natural interpretation of Boorse (1977, 562) is that the efficiency level of a trait token’s performance equals that performance’s level of contribution to the bearer’s chances of succeeding in survival or reproduction.

We may now summarize how the BST defines health and pathology of trait tokens.

Health\textsubscript{BST}: A trait token is healthy if and only if, for each of its physiological functions $F$, it has a disposition to perform $F$ on typical occasions with at least typical efficiency, relative to the organism’s reference class.

A trait token is pathological if and only if it is not healthy, that is:

Pathology\textsubscript{BST}: A trait token is pathological if and only if, for at least one of its physiological functions $F$, it does not have a disposition to perform $F$ on
typical occasions with at least typical efficiency, relative to the organism’s reference class.

In order to be more exact about what the BST says about the line distinguishing health from pathology, we should, however, look at two possible interpretations of the theory. Let us call the first interpretation the BST$_1$. On this interpretation, the line is “statistically determined.” I take “statistically determined” to require that there is a function

\[ d: \text{distributions of efficiency levels} \rightarrow \text{efficiency levels} \]

such that, for each population distribution $E$ of efficiency levels of trait token performances of a physiological function in a reference class, $d(E)$ is the efficiency level where healthy trait tokens are distinguished from pathological trait tokens. Importantly, one and the same function $d$ is used to draw each distinguishing line. The existence of such a function $d$ is probably not a sufficient condition for the distinguishing line to be “statistically determined.” I do not have any concrete suggestions for further sufficient criteria but I think the idea is that $d$ should be a “nonexotic” function defined in terms of statistical measures such as averages and standard deviations. In particular, a definition of $d$ on a case-by-case basis should not qualify as “statistical.”

Let us call the second interpretation the BST$_2$. On this interpretation, we do not have a complete account of how health is distinguished from pathology. Rather, Boorse merely provides some necessary conditions that such an account must adhere to, for example, that the distinguishing line must end up below, or far below, the average efficiency level. In particular, this interpretation allows for “nonstatistical” ways of drawing the line.

First Problem: What is Efficiency?

One problem with the BST regards the concept of efficiency. In order to systematically ascribe health and pathology to trait tokens in accordance with the BST, one needs an account of efficiency from which it is clear, at least in principle, how to determine the efficiency levels of different trait token performances. Above I provided two rough ideas of efficiency that one can read into Boorse’s writings. However, even if we assume one of these ideas, it is still not clear what “optimal performance” or “level of goal-contribution” means. In any case, Boorse is silent about how to systematically determine and compare levels of efficiency.

Second Problem: What Determines the Distinguishing Line?

Another issue with the BST concerns how it draws the line distinguishing health from pathology (among the various efficiency levels). As mentioned, the BST is aimed to capture the concepts of health and pathology in medical theory. Hence, the soundness of the BST cannot be charged with failing
to harmonize with everyday intuitions. However, implications of the theory should harmonize quite well with physiologists and pathologists’ intuitions.\(^5\) Although the verdicts of the BST might often be said to conform rather well to physiologists and pathologists’ classification of states into healthy or pathological, there are exceptions where the theory gives controversial results. This happens both in cases where diseases are (very) common (section “Common diseases”) and in cases of “normal aging” (section “Normal aging”).

**Common diseases**

Diseases that are (very) common cause problems for both interpretations of the BST, although more seriously for the BST\(_1\). The problem is that although pathologists classify states that these diseases cause as pathological, the BST cannot say (or only very doubtfully) that all of these states are pathological, because they are quite common. Peter Schwartz (2007, 375) brings up a couple of examples of common diseases. One is a certain dysfunction of the hip joint in dogs (canine hip dysplasia), estimated to be present in 30 percent of the population in some breeds. Another is urinary dysfunction in human beings due to benign prostatic hypertrophy, estimated to occur in more than 17 percent of men older than 70. Still another example is senility of the Alzheimer’s type, which affects 16 percent of people older than 85. Boorse himself (1977, 566) brings up dental caries as an example of a common state classified as pathological by pathologists.

Let us first consider the BST\(_1\). Here the exact position of the distinguishing line is conventional, but it is at least drawn “statistically” and below, or far below, the average efficiency level. However, these requirements allow for several definitions of the line. Let us be more precise. In accordance with the BST, trait token performances of physiological functions are ascribed efficiency levels. Let us denote the function making these ascriptions \(e_{\text{BST}}:\)

\[
\begin{align*}
\text{\(e_{\text{BST}}: \) performances of physiological functions} & \rightarrow \text{efficiency levels} \\
\text{\(e_{\text{BST}}(p): \) = the efficiency level of the performance} & \text{\(p\)}
\end{align*}
\]

Although Boorse does not say exactly what efficiency levels are, he at least assumes that they are linearly ordered and are such that we can define statistical measures such as averages and standard deviations. This means that efficiency levels, e.g., could be real numbers, non-negative real numbers, or real numbers in the interval 0–1. Let us, for the purpose of some generality, assume that efficiency levels are real numbers.

As explained above, for the distinguishing line to count as “statistically determined”, there must be some, not too “exotic”, function

\(d: \) distributions of efficiency levels \(\rightarrow\) efficiency levels

such that a trait token is pathological, relative to a reference class \(R\), if and only if \(e_{\text{BST}}(p) < d(E)\), or alternatively \(e_{\text{BST}}(p) \leq d(E)\), for at least one of its
performances $p$ of a physiological function, where $E$ is the distribution of efficiency levels of the trait token performances in $R$ of that physiological function. Some reasonable definitions of $d$, which can reasonably be claimed to draw the line “statistically” are the following (where $\text{avg}(E)$ and $\text{std}(E)$ denote the average and the standard deviation of the distribution $E$, respectively):

\[
d(E) := \text{avg}(E) - c \quad (c \text{ a constant real number, } c > 0)
\]
\[
d(E) := c \cdot \text{avg}(E) \quad (c \text{ a constant real number, } 0 < c < 1, E \geq 0)
\]
\[
d(E) := \text{avg}(E) - c \cdot \text{std}(E) \quad (c \text{ a constant real number, } c > 0)
\]

Importantly, the BST$_1$ cannot allow for different definitions of $d$ in different cases, and particularly not different values of $c$ in different cases, since that would mean that the distinguishing line is not “statistically determined” but set by something else. However, none of the three possible interpretations of $d$ above will provide the “right” results both for tokens of trait types affected by common diseases and for tokens of trait types where pathology is rare. And, it seems very unlikely that one could come up with some other definition of $d$ that does so and can reasonably be claimed to be a “statistically determined” line.

What about the BST$_2$, then? While this interpretation leaves the question of how to define the distinguishing line relatively open, it allows for larger variations in frequency of pathology between different trait types. Still, also according to the BST$_2$ the line must end up below, or far below, the average efficiency level. Hence, it is questionable whether it allows for 30 percent of the trait tokens of a type being pathological. Moreover, there may be pathologies affecting 50 percent or more of the trait tokens, which would be even harder to account for. Of course, one desirable feature missing in the BST$_2$ is an account of how the distinguishing line is determined.

**Normal aging**

A second type of states that causes problems for the BST is what is often called “normal aging.” To clarify the discussion of “normal aging,” I suggest we distinguish between two types of states that are both aimed at by the term “normal aging.” States of the first kind are functional reductions that occur among more or less all individuals while getting older. One example here is perhaps reduction in muscle function due to reduction in muscle mass. States of the second kind are conditions that merely become more common in groups of individuals as they get older. States that could serve as examples here are Schwartz’ cases of urinary dysfunction due to benign prostatic hypertrophy among men older than 70, and senility of the Alzheimer type among people over 85. These states may be described as a subcategory of common diseases. Although there is no sharp line between the two types of normal aging, I think the distinction is useful. While states
of the second kind are thought of as pathological in medical theory, states of
the first kind are not (at least it is controversial to claim they are).

If we consider the two interpretations of the BST, both the BST$_1$ and the
BST$_2$ classify states of the first kind as healthy. Both interpretations also
classify states of the second kind as healthy, although, just as with common
diseases, the BST$_2$ may accommodate some cases of normal aging as patho-
logical. This result has given rise to criticism, which Boorse (1997, 91–2)
has answered by stating that there is no contradiction between a state being
healthy and health care aiming to treat it. Yet, while this is correct, there
may be other reasons for thinking that some of these states should count as
pathological. Considering pathologists’ classification of states into healthy
and pathological, it is a cost for the BST that it does not harmonize with
medical theory here.

III. DEVELOPING AN ALTERNATIVE ACCOUNT

Having considered the BST and two of its problems, I now develop an alter-
native account of how to distinguish between health and pathology. I take
Boorse’s theory as my point of departure and make some crucial amend-
ments, aimed at dealing with the two problems described—(i) what is ef-
ficiency and (ii) how, among the various efficiency levels, should the line
distinguishing health from pathology be drawn? In section “Efficiency,” I pro-
pose a measure for efficiency. In section “Where to Draw the Distinguishing
Line,” I propose to distinguish health from pathology using this efficiency
measure. In this essay, I simply work under the assumption that there is a
sound account of reference classes and a sound account of physiological
functions to be made, presumably more or less along the lines of the BST.
For reasons of simplification, I define the health and pathology of a trait
token by reference to that trait token’s performances of its physiological
functions, rather than by reference to its dispositions to perform its physio-
logical functions. However, I believe that the suggested account can be
modified into an account that makes health and pathology a matter of dis-
positions rather than actual performances.

Efficiency

Let us then consider how to understand efficiency of trait token perform-
ances of physiological functions. First, I discuss the relation between the two
goals of survival and reproduction and suggest that we measure separately
how efficiently they are met (section “The goals of survival and reproduc-
tion”). Then, I make some clarifications about performances of physiological
functions (section “Performances of physiological functions”) and introduce
“feature types” and “feature values” (section “Feature types and feature
values”). Then, I provide definitions of “survival chances” and “reproduction
chances” (section “Survival chances and reproduction chances”). I then suggest that, relative to reference classes, we use a standard context for all efficiency evaluations (section “A standard context for efficiency evaluations”). Last, I define measures for survival-efficiency and reproduction-efficiency (section “Efficiency of trait token performances”).

**The goals of survival and reproduction**

I agree with Boorse that survival and reproduction are both relevant goals for evaluations of health or pathology statuses of trait tokens. However, in contrast to the BST, which has a single concept of efficiency, which covers both survival and reproduction chances, I think that the two goals should be given separate efficiency measures. With only one efficiency measure encompassing both survival and reproduction chances (and without any further qualifications), the efficiency level of a performance will sometimes be indeterminate. In some cases a trait token’s performance enhances the possessor’s chances of survival in comparison to the reference class more than it enhances the possessor’s chances of reproduction in comparison to the reference class (or vice versa). For example, think of a human female at age 23, whose hypophysis secretion of hormones is abnormal in a way that makes her ovulate very infrequently. The abnormal hormone levels, however, do not affect her notably in any other way. Here we have a situation where the hypophysis abnormal secretion of hormones reduces the woman’s chances of succeeding in reproduction relative to her reference class, but does not reduce her chances of survival, relative to her reference class. Operating with only one efficiency measure here (and no further qualifications), it is indeterminate what the efficiency of the hypophysis performance is. Generally, in all cases where a trait token’s performance enhances survival chances more (or less) than reproduction chances, in relation to the reference class, it will be indeterminate what the efficiency level of that performance is.

By adding further qualifications, one could solve the problem of indeterminacy. However, what I propose and also think coheres well with physiological and pathological theory is to distinguish between survival and reproduction as goals of two distinct efficiency measures. The suggestion is to talk about survival-efficiency on the one hand, and reproduction-efficiency on the other. Then, with regard to the concepts of health and pathology, we have two options. Either we define pathology as reduction in survival-efficiency or reproduction-efficiency and health as absence of reduction in survival-efficiency and absence of reduction in reproduction-efficiency. In the above example, we would then get the result that the hypophysis is pathological, since its reproduction-efficiency is reduced. Alternatively, we also distinguish between two health/pathology concepts, one for each efficiency measure: reproductive health/pathology on the one hand, and
health/pathology pertaining to survival on the other. If we adopt the latter approach, in the above example we get the result that the hypophysis is reproductively pathological but healthy with regard to survival. Which of these two alternatives should we choose? This I take mostly to be a matter of terminological taste. However, if one of the alternatives coheres better than the other with physiological and pathological theory, that would be a reason to favor that alternative. In this essay, I just opt for the second alternative and distinguish between two health/pathology concepts.

Performances of physiological functions

“Survival-efficiency” and “reproduction-efficiency” apply to trait token performances of physiological functions. Above, I said that I would continue under the assumption that there is a sound account of physiological functions to be given. However, there are some things I presuppose of such an account. I take physiological functions to be tied to trait types. Hence, even if there are more than one trait type in organisms of a reference class that are responsible for a certain effect, the physiological functions of these trait types are distinct. For example, both the human liver and the human kidneys are responsible for filtering the blood. Yet, we should distinguish between the liver’s filtration of blood and the kidney’s filtration of blood as two distinct physiological functions. Presumably, we should also distinguish between the left kidney’s filtration of blood and the right kidney’s filtration of blood as two distinct physiological functions. It should also be pointed out that trait types can be described at different levels. A trait type (at a higher level) may comprise several trait types (at lower levels). For example, the circulatory system may be described as one trait type, the heart as another, and the mitral heart valve as yet another one. These three trait types all have their own physiological functions.

Whereas the physiological functions are shared by the individuals of a reference class, the performances of these functions differ. In order to ascribe efficiency levels also in cases where a physiological function is not carried out at all, from now on I use the term ‘performance’ to denote both the carrying out of a function and the omission to do so. So, even a heart token that does not pump blood at all performs, in this sense, the function of pumping blood (maximally deficiently). How, then, are performances of physiological functions individuated? A performance of a physiological function is tied to its trait token and its time of performance. Hence, different trait tokens will have different performances. Also, one trait token can have different performances of a certain physiological function at different times. (Otherwise, we would not be able to judge a trait token healthy at one time and pathological at another time.) How individuations of performances of the same physiological function by the same trait token are to be made I take to be a matter of theoretical fruitfulness, and, within those frames, of
convention. Some individuations are more useful than others in determining survival chances, or, respectively, reproduction chances, but some individuations may be as useful. There may even be several individuations of performances of a physiological function that are all (together) adequate for determining survival chances, or, respectively, reproduction chances.

The individuation of performances may be done in terms of absolute time units or in terms of non-absolute time units, e.g., biological cycles. It should be noted, though, that in cases where the physiological function is not carried out at all (and, possibly, in some cases where the physiological function is carried out, but not “as it should”), it may not be possible to individuate performances in all ways adequate for that physiological function. For example, say that the blood pumping of hearts is adequately individuated by the cycle of one contraction. That individuation principle cannot define the blood pumping of a heart that does not contract at all. Rather, that heart token’s blood-pumping performance will be an instance of the special case where the physiological function is not carried out at all.

Feature types and feature values

We can describe and compare performances of a certain physiological function by looking at features of the performances. For example, we can describe and compare different heart tokens’ blood pumping by considering feature types such as beating rhythm and stroke volume. These feature types are relevant since they matter for the organism’s survival chances (and indirectly also for the organism’s reproduction chances). Just as I assumed that physiological functions are tied to trait types, I assume that feature types are tied to physiological functions. So, even if there is some physiological function besides the heart’s blood pumping, for which it is relevant to look at beating rhythm, we should distinguish between the beating rhythm of the heart’s blood pumping and the beating rhythm of the performance of the other physiological function.

For every physiological function, I assume a set of relevant feature types:

\[
U: \text{physiological functions} \rightarrow \text{sets of feature types}
\]

\[
U(F):= \text{the set of feature types that are relevant for the physiological function } F.
\]

For each performance of \(F\), every feature type in \(U(F)\) takes a feature value. As an example, say that the relevant feature types for hearts’ blood pumping are beating rhythm and stroke volume. Then, for each heart’s blood pumping, beating rhythm is assigned a value and stroke volume is assigned a value. Since different feature types may be adequately expressed by different types of values, let us allow for all kinds of values (e.g., qualitative as well as quantitative values, and exact values as well as sets or intervals of values, and possibly an “undefined” value).
Let us call the values assigned to each of the feature types in $U(F)$ taken together a ‘configuration of feature values’. For each physiological function $F$, the set of its possible feature value configurations is the set of functions $U(F) \rightarrow \text{values}$.

What we now want to define is the feature value configuration for the performance of a physiological function. For convenience, we first define the following function:

$f: \text{performances of physiological functions} \rightarrow \text{physiological functions}$

$f(p):= \text{the physiological function that } p \text{ is a performance of.}$

Then, for every performance of a physiological function, we have a configuration of feature values:

$P: \text{performances of physiological functions} \rightarrow \text{feature value configurations}$

$P(p):= \text{the feature value configuration of } p$, i.e., the function

$c_p: U(f(p)) \rightarrow \text{values}$

$c_p(t):= \text{the value } p \text{ takes for the feature type } t.$

As a concrete example, consider the feature value configuration of the blood pumping of Emma’s heart (let us still assume that the relevant feature types for hearts’ blood pumping are beating rhythm and stroke volume):

$P(\text{Emma’s blood pumping}): \{\text{beating rhythm, stroke volume}\} \rightarrow \text{values}$

$P(\text{Emma’s blood pumping})(\text{beating rhythm}):= \text{60 beats/minute}$

$P(\text{Emma’s blood pumping})(\text{stroke volume}):= \text{60 mL/beat}$

**Survival chances and reproduction chances**

How should we define “survival chances” and “reproduction chances”? The term “survival chances” could mean different things, e.g., chances of surviving for another day, or chances of surviving for fifty years. If we measure “survival chances” by chances of surviving for a certain period of time, we get the problem that what is a reasonable period of time may differ between species and reference classes. A better idea is to measure “survival chances” by expected length of life. This measure can be used univocally for all species and reference classes.

I assume that, given a certain environment, the expected length of life of an individual is (at least approximately) determined by the complete set of feature value configurations of that individual. By “complete set of feature value configurations” I mean the set comprising all feature value configurations of all performances of physiological functions in that individual (i.e., the set of all feature value configurations $P(p)$ for the performance $p$ of
every physiological function in the organism). So, instead of talking about expected lengths of life of individuals, we can talk about expected lengths of life of complete feature value configurations.

We let \(\text{ell}(I)\) denote the expected length of life of the complete set of feature value configurations \(I\). We then have:

\[
\text{ell}: \text{complete sets of feature value configurations} \rightarrow \text{non-negative real numbers}
\]

\[
\text{ell}(I):= \text{the expected length of life of the complete set } I \text{ of feature value configurations.}
\]

What about “reproduction chances”? The best way to measure this calls for more discussion. For example, should the measurement be about number of offspring, or number of viable offspring, or about spreading of genes? For the purposes of this essay, as an example, I measure chances of succeeding in reproduction by expected number of offspring. In case one prefers another measurement, expected number of offspring could be exchanged for that.

We let \(\text{eno}(I)\) denote the expected number of offspring of the complete set of feature value configurations \(I\). We then have:

\[
\text{eno}: \text{complete sets of feature value configurations} \rightarrow \text{non-negative real numbers}
\]

\[
\text{eno}(I):= \text{the expected number of offspring of the complete set } I \text{ of feature value configurations.}
\]

A standard context for efficiency evaluations

Before we can further account for survival-efficiency and reproduction-efficiency, we need to consider the issue in what context the efficiency-evaluation of performances of physiological functions should be made. Should the efficiency of a trait token’s performance be relativized to the unique individual organism, or rather to some common standard for the reference class? This question is relevant, since two tokens of a certain trait type may perform exactly alike in two individuals of the same reference class but affect the respective individuals’ expected length of life, and respectively their expected number of offspring, to different extents, because of differences in feature value configurations of other trait token performances in these individuals. In such a case, should we say that the two tokens have performances that are as efficient, or that they differ? I do not provide an exhaustive discussion of this issue here. However, what I think we should say is that both tokens’ performances are of the same efficiency. Hence, I assume that efficiencies of trait token performances of physiological functions are not purely individual-relative. Rather, for every reference class, I assume a standard context in which we measure efficiencies of trait token
performances. That context, I take it, is the “exemplary combination” of feature value configurations for the reference class. This “exemplary combination” is complete in the sense that it comprises feature value configurations for all physiological functions of the reference class. When measuring the efficiency of a trait token performance, we consider how that performance’s feature value configuration would affect the expected length of life of an organism with the exemplary combination of feature value configurations. On the one hand, we have a combination of feature value configurations that is exemplary for (high) expected length of life. That is the relevant context for evaluations of survival-efficiencies of trait token performances. On the other hand, we have a combination of feature value configurations that is exemplary for (large) expected number of offspring. That is the relevant context for evaluations of reproduction-efficiencies of trait token performances.

What does it mean, then, for a combination of feature value configurations to be “exemplary”? In very rough terms, the idea is that the survival-exemplary combination of feature value configurations of a reference class is the complete combination of feature value configurations that gives the highest expected length of life that is relatively easy to achieve for a considerable portion of the members of the reference class in normal environments of the reference class.9 Mutatis mutandis for the reproduction-exemplary combination of feature value configurations. The idea is that, for each reference class there is only one survival-exemplary combination of feature value configurations and only one reproduction-exemplary combination of feature value configurations.10

In accordance with the above discussion, for every reference class $R$ I assume a survival-exemplary combination of feature value configurations, notation $\text{Ex}_s(R)$:

$\text{Ex}_s: \text{reference classes } \rightarrow \text{ sets of feature value configurations}$

$\text{Ex}_s(R):= \text{the survival-exemplary combination of feature value configurations of reference class } R.$

Also, for every reference class, I assume a reproduction-exemplary combination of feature value configurations, notation $\text{Ex}_r(R)$:

$\text{Ex}_r: \text{reference classes } \rightarrow \text{ sets of feature value configurations}$

$\text{Ex}_r(R):= \text{the reproduction-exemplary combination of feature value configurations of reference class } R.$

**Efficiency of trait token performances**

Now, the idea that I put forward is that the survival-efficiency of a trait token’s performance is determined as follows. We take the survival-exemplary combination of feature value configurations of the reference class of the bearer of the trait token. We note the expected length of life of this combination
of feature value configurations. Then, we again take the survival-exemplary combination of feature value configurations of the reference class of the bearer of the trait token, but make a modification. We modify the feature value configuration of the performance of the physiological function that the performance under evaluation is a performance of: we modify this feature value configuration to be the same as the feature value configuration of the performance under evaluation. We then note the expected length of life of this modified combination of feature value configurations. The survival-efficiency of the trait token’s performance is then calculated by dividing the expected length of life of the modified combination of feature value configurations with the expected length of life of the exemplary combination of feature value configurations. Mutatis mutandis for reproduction-efficiency.

Let us put this formally. Let \( p \) be the performance of a physiological function of a trait token in some individual, e.g., the pumping of blood by the heart in some particular human. \( p \) has the feature value configuration \( P(p) \). Let \( I \) be a complete set of feature value configurations. We can then think of a(whole) complete set of feature value configurations which we get by setting the feature value configuration of the performance of \( f(p) \) in \( I \) to \( P(p) \).

We denote this modified set of feature value configurations \( M(I, p) \), thus:

\[
M(I, p) := \text{the set of feature value configurations that results from the modification of the set } I \text{ of feature value configurations, such that the feature value configuration of } I \text{'s performance of } f(p) \text{ is set to } P(p).^{11}
\]

When modifying certain feature value configurations of an exemplary combination, this may also affect feature value configurations of performances of other physiological functions. It might even result in feature value configurations of performances of other physiological functions indicating pathology. Some diseases are such that when a certain trait token becomes pathological, that affects other trait tokens of the body, sometimes to the extent that they also become pathological. For example, in individuals with diabetes mellitus the beta cells of the pancreas are not producing insulin. Without proper intervention, this leads to pathological states of other parts of the body, e.g., of the eyes (in the worst case blindness), and of the feet and the legs (which in the worst case need to be amputated). Thus, when we measure the efficiency of a trait token’s performance of a physiological function, we cannot just assume that the feature value configurations of the performances of all other physiological functions are still the same as in the exemplary combination. Rather, we must look at the expected length of life, or respectively the expected number of offspring, of the combination of feature value configurations such that it becomes after the specific modification.
We may now define survival-efficiency and reproduction-efficiency of performances of physiological functions. Let $R$ be a reference class of individuals. Let $p$ be the performance of a physiological function, for example, the pumping of blood by the heart in some particular human. The survival-efficiency of a trait token’s performance relativized to $R$, notation $e_s(R, p)$, is the ratio of the expected length of life of the combination of feature value configurations resulting from a modification of the exemplary combination of feature value configurations such that the feature values for the performance of $f(p)$ have been set to $P(p)$, and the expected length of life of the exemplary combination of feature value configurations:

$$e_s: \text{reference classes and performances of physiological functions} \rightarrow \text{non-negative real numbers}$$

$$e_s(R, p) := \frac{\text{ell}(M(\text{Ex}_s(R), p))}{\text{ell}(\text{Ex}_s(R))}^{12}$$

The reproduction-efficiency of a trait token’s performance relativized to $R$, notation $e_r(R, p)$, can be formulated similarly:

$$e_r: \text{reference classes and performances of physiological functions} \rightarrow \text{non-negative real numbers}$$

$$e_r(R, p) := \frac{\text{eno}(M(\text{Ex}_r(R), p))}{\text{eno}(\text{Ex}_r(R))}^{13}$$

Since neither expected length of life nor expected number of offspring can be negative, $e_s(R, p)$ and $e_r(R, p)$ will never take values lower than 0. If the expected length of life of the exemplary combination of feature value configurations is the same also when the feature value configuration of the performance of $f(p)$ is set to $P(p)$, then the performance will have the survival-efficiency measure 1. Now if the expected length of life of the exemplary combination of feature value configurations decreases when setting the feature value configuration of the performance of $f(p)$ to $P(p)$, then the efficiency measure will be below 1. Mutatis mutandis for reproduction-efficiency. The efficiency measures may also take values above 1. That happens when a trait token has a performance $p$ of a physiological function such that the expected length of life, or respectively the expected number of offspring, of the exemplary combination of feature value configurations gets enhanced when the feature value configuration of the performance of $f(p)$ is set to $P(p)$.

Let us look at some examples of performances of physiological functions pertaining to survival. First, consider the blood pumping of hearts. Different heart tokens may have rather different survival-efficiency measures. Some heart tokens are probably such that the feature value configuration of their blood pumping would not at all affect the expected length of life of the exemplary combination of feature value configurations. Their blood pumping has the survival-efficiency measure 1. Many heart tokens are probably such that the feature value configuration of their blood pumping would only to
a limited extent affect the expected length of life of the exemplary combination of feature value configurations. Their blood pumping has a survival-efficiency measure slightly below or slightly above 1. Some heart tokens are probably such that the feature value configuration of their blood pumping would considerably decrease the expected length of life of the exemplary combination of feature value configurations. Their blood pumping has a survival-efficiency measure significantly below 1. Also, although probably very rarely, the feature value configuration of some heart tokens' blood pumping may significantly increase the expected length of life of the exemplary combination of feature value configurations. Their blood pumping has a survival-efficiency measure significantly over 1. Now, contrast the heart’s pumping of blood with a single melanocyte’s production of melanin (pigments protecting the human body from UV-radiation). No matter how well-functioning or not the melanin production of a single cell is, the impact on expected length of life is incredibly small. This means that for a single melanocyte the survival-efficiency measure will always be (very close to) 1. If we think of the whole system of melanocytes, on the other hand, the melanin production’s impact on expected length of life is larger; although not as large as the heart’s blood pumping. (One may survive without the melanin’s protection against UV-radiation, but not without the heart’s blood pumping.) What we can conclude is a tendency that the more important the performance of the function is for the goal, the more its efficiency measure may vary.

Where to Draw the Distinguishing Line

Having accounted for survival-efficiency and reproduction-efficiency, let us turn to the question of what determines at which level of efficiency the line between health and pathology to be drawn. I first provide my account. Then, I explain how it avoids the BST’s problems of common diseases and normal aging. The general idea is that a trait token is pathological if and only if the efficiency of at least one of its performances of a physiological function has a particularly low efficiency. Let us put this formally. For convenience, we first define the following function:

\[ Z : \text{trait tokens} \rightarrow \text{sets of physiological functions} \]

\[ Z(a) := \text{the set of physiological functions for the trait type of which } a \text{ is a token.} \]

Let \( x_s \) be a constant that represents the highest survival-efficiency measure that counts as particularly low. The suggested definition of pathology pertaining to survival, pathology, is then:

\[ \text{Pathology} : \text{A trait token } a \text{ is pathological if and only if at least one of its performances } p \text{ of a function in } Z(a) \text{ is such that } e_s(R, p) \leq x_s. \]
From this, we also get a definition of health pertaining to survival, health_s. A trait token is healthy_s if and only if it is not pathological_s, that is:

**Health_s**: A trait token \(a\) is healthy_s if and only if each of its performances \(p\) of the functions in \(Z(a)\) is such that \(e_s(R, p) > x_s\).

Let \(x_r\) be a constant that represents the highest reproduction-efficiency measure that counts as particularly low. Then, we also have the corresponding definitions of pathology and health pertaining to reproduction, pathology_r and health_r:

**Pathology_r**: A trait token \(a\) is pathological_r if and only if at least one of its performances \(p\) of a function in \(Z(a)\) is such that \(e_r(R, p) \leq x_r\).

**Health_r**: A trait token \(a\) is healthy_r if and only if each of its performances \(p\) of the functions in \(Z(a)\) is such that \(e_r(R, p) > x_r\).

How should we understand \(x_s\) and \(x_r\)? \(x_s\) and \(x_r\) are to reflect what pathologists take to be such a low efficiency of a performance that the trait token is pathological. Just like Boorse’s distinguishing line, the values of \(x_s\) and \(x_r\) are conventionally chosen. However, the values of \(x_s\) and \(x_r\) should be smaller than 1. Otherwise, we will have pathology without any reduction in efficiency. Definitely for \(x_s\) and reasonably also for \(x_r\), the value should also be greater than 0. Otherwise, for a trait token to be pathological, it would be required that the expected length of life of the survival-exemplary combination of feature value configurations, having the feature value configuration of the performance of \(f(p)\) set to \(P(p)\), is 0. Now, for a trait token to be pathological, it would be required that the expected number of offspring of the reproduction-exemplary combination of feature value configurations, having the feature value configuration of the performance of \(f(p)\) set to \(P(p)\), is none.

As mentioned, \(x_s\) and \(x_r\) are constants. Although performances of different physiological functions may be more or less important for survival, and respectively for reproduction, \(x_s\) and \(x_r\) apply to all trait types, with regard to all of their physiological functions. For example, think again of the blood pumping of hearts, the melanin production of single melanocytes, and the melanin production of systems of melanocytes. We saw that the survival-efficiency measure may vary largely from 1 between heart tokens, but less for token systems of melanocytes, and not at all for single melanocyte tokens. Let us, merely as an example, set the value of \(x_s\) to 0.9. Then it holds equally for the heart, the system of melanocytes, and a single melanocyte that their performances’ survival-efficiency measures must be 0.9 or lower to count as pathological. A perhaps controversial implication of this is that there may be trait types where pathology is impossible. For the account to ascribe pathology to a trait token, the survival-efficiency measure must be particularly low. If a trait token is of a trait type whose physiological
functions generally do not, or do only to a very limited degree, affect expected length of life, then it cannot become pathological. Mutatis mutandis for pathology.

However, this interpretation should not necessarily be regarded as problematic. Nonpathological efficiency reductions at low levels in an organism may still contribute to pathology at higher levels in that organism. For example, consider again type-1 diabetes mellitus. In cases of type-1 diabetes mellitus, the beta cells in the pancreas get destructed. Normally, beta cells are responsible for producing insulin. A person with type-1 diabetes mellitus therefore becomes destitute of insulin. This in turn has very serious consequences, for instance, fatigue, weight loss, and blurry vision. What is noteworthy here is that what causes the detrimental states is “merely” a dysfunction of certain cells. If we look at a single beta cell, its survival-efficiency will be 1 (or very close to 1) irrespective of whether it performs its physiological function of producing melanin or not. This means that, in cases of type-1 diabetes mellitus, my suggested account cannot say of a single defective beta cell that it is pathological. This might sound like an unacceptable result. However, I think it is not. That we cannot say of each beta cell that it is pathological does not preclude that there is pathology in the organism. There are still larger efficiency reductions at higher levels than at the level of the single cell. Considering the system of beta cells, the survival-efficiency of its performance is reasonably well below the value of $\chi_s$. Thus, when an insulin-producing system breaks down, as it does by type-1 diabetes mellitus, its survival-efficiency becomes particularly low, and thus pathological. Moreover, several effects of the insulin deficit on tokens of other trait types in the organism lead to further pathological states.

Another implication of the suggested account is that trait tokens can be healthy, or pathological, and respectively healthy or pathological, only if their bearer belongs to a reference class where the expected length of life, and respectively, the expected number of offspring of the exemplary combination of feature value configurations is $> 0$. This is, as mentioned in footnotes in section “Efficiency of trait token performances” because $e_s(R, p)$ is undefined when $ell(Ex_s(R)) = 0$ and $e(R, p)$ is undefined when $eno(Ex(R)) = 0$. However, this should not be considered a serious problem. With regard to health and pathology, it is doubtful whether there could even exist reference classes with such survival-exemplary combinations of feature value configurations. With regard to health and pathology, on the other hand, such reproduction-exemplary combinations of feature value configurations could perhaps make sense, at least for some reference classes among hybrid species where reproduction is impossible. If the expected number of offspring of the exemplary combination of feature value configurations is 0, then it seems that the concepts of reproduction-efficiency, health, and pathology, are irrelevant to trait tokens within that reference class.
How, then, does the suggested account serve better than the BST with regard to common diseases and normal aging? With regard to common diseases, the account serves better than the BST (regardless of interpretation), since it allows for very large proportions of the tokens of a trait type being pathological, or pathological,_. For example, if dental caries reduces the survival-efficiency of the dental system’s performance below $\chi_s$, and occurs among, say, 20 percent of the reference class, then tooth decay is a pathological, condition. The same goes for canine hip dysplasia in dogs. If it reduces the survival-efficiency of the hip’s performance below $\chi_s$, it is a pathological, state in 30 percent of some breed. My account could even ascribe pathology, or pathology, to 100 percent of the trait tokens. Although such a situation is very unlikely, we can at least think of it as theoretically possible. For example, consider teeth and cavities.\(^{14}\) If the exemplary combination of feature value configurations of a reference class precludes cavities, this means that a considerable number of the members of the reference class could relatively easily lack cavities in normal environments of the reference class. However, if all reference class members despite this choose to eat a lot of sugar, they may all obtain pathological, teeth.

Also, in cases of normal aging my account gives better results than the BST. Let us first consider states that affect more or less all individuals while getting older. These states will also affect the expected length of life of the exemplary combination of feature value configurations. Thus, a performance $p$ affected by normal aging will not make $\overline{e[l]}(M(\text{Ex}(R), p)) / \overline{e[l]}(\text{Ex}(R))$ particularly low. Hence, my account will not classify states of this type of normal aging as pathological,_. This is the same result that the BST gives. However, if we consider states that merely become more common among individuals as they get older, there is a difference. In these cases, the exemplary individual’s expected length of life remains unaffected. Hence, here my account gives the same results as it does in cases of common diseases. It ascribes pathology, to more states than the BST. Although pathology pertaining to survival is the more relevant pathology concept in the context of normal aging, a parallel story could be told for reproductive pathology.

IV. COMPETING PROPOSALS

I now defend my account against another suggested idea of how to amend the BST to solve the problem of common diseases. This idea is developed, in somewhat different ways, by Peter Schwartz (2007) and Daniel Hausman (2012, 2014). Unlike me, Schwartz and Hausman take the BST’s problem of common diseases to show that an account distinguishing health from pathology in medical theory needs some further aspect besides survival-efficiency and reproduction-efficiency, namely, an aspect of negative consequences.
I first dispute Schwartz’s argument for adding a further aspect beyond survival-efficiency and reproduction-efficiency to the account (section “Schwartz’s Argument for Adding a Further Aspect”). Then, I argue that Hausman’s addition of further goals besides survival and reproduction conflicts with the project of the BST (section “Hausman’s Addition of Further Disjunctive Goals”).

Schwartz’s Argument for Adding a Further Aspect

Schwartz thinks that some aspect other than survival- and reproduction-efficiency is needed in accounting for the health–pathology distinction. Schwartz’s main reason for this is that pathological conditions need not reduce survival or reproduction. For example, aphasia, blindness, or a missing limb need not be associated with reduced chances of survival or reproduction in a society that is suitably supportive (Schwartz, 2007, 379).

I think Schwartz is right that pathologists clearly judge aphasia, blindness, and missing limbs to be pathologies, although many societies today have the resources to adapt the environment in such a way that these conditions do not affect survival or reproduction (to the same extent as they once did). However, I do not think that we need a further aspect beyond survival-efficiency and reproduction-efficiency to account for these states as pathological.

First, whether a state is healthy or pathological is not determined by facts about how well or bad the particular individual actually succeeds in survival or reproduction. Rather, that is about the individual’s chances of succeeding in survival or reproduction. If some individual is lucky enough to succeed quite well in survival or reproduction in spite of having reduced chances of doing so, that does not mean that aphasia is not pathological for that person. Here, one may also stress the role of the reference class: health and pathology make sense only in relation to a reference class. As long as aphasia typically reduces chances of survival or reproduction for members in the reference class, it is a pathological condition also for this lucky individual. Second, health and pathology should be relative to actual normal environments, and not to possible environments. Even if we can imagine a nearby world in which society spends more on supporting people with aphasia, so that this condition does not negatively affect survival or reproduction, it does not follow that aphasia is not pathological in the actual world.

However, these clarifications do not refute the argument with the stronger claim that a normal environment actually is such that aphasia, blindness, and a missing limb do not reduce chances of succeeding in survival or reproduction. Let us understand Schwartz as making this claim in arguing that a further dimension of negative consequences is needed. The question, then, is whether this claim is true. Let us concentrate on the example of aphasia.
In which sense does aphasia not reduce chances of succeeding in survival or reproduction today? It is hardly the case that one can get severe aphasia and go unnoticed (not seeking help), without having lowered survival- or reproduction chances. Communication, I would say, is still crucial for these goals. We sometimes need to communicate our needs to other persons, and we need to understand both verbal and written language to manage in society. However, because of health care and social systems a person with, for example, aphasia can get support in various ways. One could roughly categorize these ways of getting support into four types of interventions (which may be combined). One type of intervention has as its goal to increase the capability of the trait token that is pathological (in cases of aphasia, e.g., trait tokens responsible for word mobilization). A second has as its goal to strengthen some other trait token(s) by which the individual can compensate for the pathological trait token (e.g., trait tokens responsible for body language). A third kind of intervention consists in providing tools for the individual to use in order to compensate for the pathological trait (e.g., a folder with pictures to point at). A fourth kind of intervention aims at adapting the environment in a sense that is more passive in relation to the individual (e.g., educating the individual’s family in how to optimize conditions for communication).

If we consider an intervention of the first type, there is a pathological condition, which is then (completely or partly) treated, so that after the intervention there is no (or alternatively, a less severe) pathology. Observe that the fact that we today have the resources to treat many conditions does not imply that these conditions are not pathological. Now compare this to interventions of types two, three, and four. In these cases, the trait token with an efficiency-reduced performance is not intervened on. Instead, the compensatory trait token or tool or environmental adaption is needed as long as the performance’s efficiency is reduced. With regard to aphasia, it can seldom be completely treated by interventions of type one. It is the availability of interventions of kinds two, three, and four that seems to be what Schwartz takes to make the environment today, such that conditions like aphasia do not reduce survival or reproduction chances. However, when the environment is altered in this way, I think it should not be regarded as a normal environment, but rather as a special environment. When we provide these interventions, we do so because we judge that in the normal environment (without interventions) the individual will have a hard time. Hence, normal environments are non-intervened environments, whereas intervened environments are special environments. According to this description, aphasia comes out as pathological also today. However, should our environment change into one where considerations about interventions need not be made, because it has become standard for all of us to use the tools or technics in question, that environment would become the normal
one. The line between normal and special environments is, however, not clear-cut, but vague.

Hausman’s Addition of Further Disjunctive Goals

My account is partly inspired by Hausman (2012, 2014). According to Hausman’s account, a trait token is pathological if the efficiency level is significantly worse than the “maximal level” (Hausman, 2012, 536). This sounds similar to my account. However, Hausman has a different view on what counts as a significantly worse level. He says that significantly worse efficiency levels are (at least) “. . . differences [from the maximal level] that seem worthy to note because of their consequences for valued traits and activities, that make large differences in the functioning of other systems, or that make large differences in the probability of survival and reproduction. . .” (2012, 537, my emphasis).

Although Hausman may get satisfying results in cases of common diseases, I think that his way of solving the problem is unsound in relation to the project of the BST. I have emphasized that the verdicts of an account of the theoretical concepts of health and pathology should harmonize quite well with the verdicts made in physiology and pathology. However, there are also other constraints to keep in mind. Remember the aim of the project: to account for health and pathology as naturalistic phenomena, being the same for all organisms. But with this aim it seems that goals about valued traits and activities cannot consistently be included in the account. The feature of being valued is mind dependent, and hence cannot be applied to plants, and only very doubtfully to animals. For example, what would be a valued activity for cactuses?15 By adding the goals of valued traits and activities Hausman also has to step out of the project of the BST.

V. CONCLUDING REMARKS

I have proposed definitions of health and pathology of trait tokens. By this, I do not exclude that there are health and pathology of other entities to be defined in physiology and pathology. For example, it is reasonable to think that when an organism lacks a token of a trait type which has an important physiological function, there is pathology. However, we cannot say of something nonexistent that it is pathological. Hence, it is questionable whether my suggested definitions can account for missing trait tokens. My account may accommodate some cases. This is when the trait type missing is part of another (higher level) trait type, of which the organism has a token. However, if the trait type missing is not part of another trait type of which the organism has a token, it is questionable whether the suggested account can account for it.
NOTES

1. There is a discussion about whether the concepts of health and pathology should be accounted for as mutually exclusive or as a continuum (Hausman, 2012; Schroeder, 2013; Rogers and Walker, 2017). My suggestion will be described in terms of a clear distinction but could be used as a basis for an account described as a continuum.

2. There are several issues with regard to reference classes. See, for example, Kingma, 2007.

3. What occasions count as typical, Boorse says “is an empirical fact about the reference class” (1977, 562). It is not obvious from this claim how typical occasions are accounted for. For discussion of this issue, see, for example, Kingma (2010). In this essay, I leave the issue aside.

4. Thanks to an anonymous referee for this interpretation.

5. More precisely what weight current medical theory should be given in this project is an important methodological question, which I do not go into in this essay.

6. Here, it should not matter how, precisely, efficiency is accounted for.

7. There is more to say about feature types and feature values than what I have space for here.

8. I do not have the space in this essay to discuss this claim further.

9. This idea is inspired by Hausman’s (2012, 536) idea of readily attainable states in normal environments.

10. For the purpose of this essay, I just make this assumption without any further argument. This claim, and exemplary combinations of feature value configurations in general, calls for more discussion, for which I do not have the space in this essay.

11. \( M \) is a partial function since \( M(I, p) \) is defined if and only if \( I \) comprises feature value configurations for a performance of the physiological function that \( p \) is a performance of.

12. \( e \) is a partial function since \( e(R, p) \) is defined if and only if \( elk(Ex(R)) \neq 0 \). I discuss implications of this in section “Where to Draw the Distinguishing Line.”

13. \( e \) is a partial function since \( e(R, p) \) is defined if and only if \( eno(Ex(R)) \neq 0 \). I discuss implications of this in section “Where to Draw the Distinguishing Line.”

14. I have taken this example from Hausman (2012, 536).

15. One could of course say that what counts as a valued trait or activity for cactuses gets determined by human minds. This, however, is a strange view to hold. Biologists do not classify states in animals and plants as healthy or pathological on grounds of what they appreciate and not appreciate about them.

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