

HUMAN PSYCHOLOGICAL NATURE 1:

Why Attacks from Philosophy of Biology Fail

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I. Introduction

Do human beings share a richly detailed mental nature? Since the term ‘human nature’ has been used in many different senses by different writers, we must be as clear as possible about the question from the start. My overall topic in these two papers is: Is it plausible that human psychology shows a complex structure of part-functions similar to that of the human body? Does the human mind, like the human body, have an elaborate constant functional design or architecture, worthy of Pinker’s phrase “a universal complex human nature” (2002, 73)? Or is human psychological nature a “superstition,” as Michael Ghiselin called it (1997,1)? This is the central theoretical question for both human psychology, the study of normal mental function, and psychiatry, the study of psychopathology -- just as its physical counterpart is for physiology and ordinary medicine.¹

I shall defend three theses. First (part 1), there is a scientifically legitimate concept of species nature immune to all recent criticisms by philosophers of biology. Second (part 2), it is indeed plausible that the human species has a complex mental, as well as bodily, functional architecture in this sense; and third, this is the only human nature needed to fulfill the traditional normative functions of the concept in ethics, policy, and law.

As will be abundantly clear below, in discussing human psychological nature, it is crucial to bear in mind the physiological analogy and its features. To emphasize the analogy, I will sometimes write ‘HΦN’ and HΨN’ for human physiology and psychology, respectively. To begin with, it is uncontroversial that typical human anatomy and physiology – HΦN – can be specified in enormous detail. Evolutionary psychologists Tooby and Cosmides put the point reasonably well:

[T]he “architecture” or physiological design of humans is both distinctively species-specific and species-typical. When one examines the organs, with their complex design and interlocking architecture, one finds (within a sex, and to a large extent between sexes) monomorphism of design. Virtually everyone has two lungs, one neck, a stomach, a pancreas, a tongue, two irises, 10 fingers,

¹From 1975 onward, I have called the functional organization typical of species members the “species design,” and argued that it is the basis of the concepts of health and disease in scientific medicine. Specifically, on my analysis a pathological condition is a state of statistically subnormal part-functional ability, relative to species, sex, and age. See Boorse (1975, 1976b, 1977, 1987, 1997, 2011, 2014).

blood, hemoglobin, insulin, and so on. And, although there is a great deal of superficial variation – no two stomachs are exactly the same size or shape, for example ... – each organ system has the same basic design. The locations and connections between organs are topologically the same, and the internal tissue structures and physiological processes have a uniformity of structure and functional regulation. One has to descend to specific enzymatic pathways before design differences – as opposed to quantitative variation -- start showing up. (1990, p. 224 of Downes and Machery)

Even a basic description of the universals of human anatomy and physiology requires a gigantic textbook: the two most popular undergraduate texts have 960 and 1264 pages respectively (Marieb and Hoehn 2015, Silverthorn 2015), while the standard text for medical students has 1171 (Hall 2015).

At the same time, this bodily human nature – HΦN – lacks many features often treated as part of the human-nature concept. First, contrary to Pinker’s phrase, it is not universal, for at least two reasons. One is that functional design varies between age groups and, in sexual species, between sexes. Females differ qualitatively from males in their organs and quantitatively in many aspects of their physiology, while immature organisms may differ from adults in both ways as well. Also, many organisms – virtually all, I argue – deviate from the species design by having pathological conditions. Not every human being has ten fingers; some have lost one or more to trauma. Not every human being has a pancreas able to secrete insulin; some have type-1 diabetes. Although every feature of the functional design is “species-typical,” not all features are found in every member. At the same time, biologists, not just physicians, distinguish normal and pathological variation. Normal variation can be purely quantitative, as with height or IQ, or qualitative, as with polymorphisms such as blood type.

Secondly, contrary to Tooby and Cosmides, most of bodily human nature is not, in fact, “species-specific.” At least qualitatively, we share nearly all our anatomy and physiology with other primates; most of it is also mammalian or even vertebrate physiology as well, and, at the biochemical level, we share some design features with all organisms on earth. In reality, for any species there are at least two distinct concepts of species nature. One includes all features typical of species members, the other only that subset unique to species members. Remarkably, this distinction does not seem to have any standard name, which may explain the

frequent confusion of the two concepts in human-nature discussions.² We can call it the distinction between *general* (or *generic*) species nature and *special* (or *specific*) species nature. It is general species functional nature that defines health and disease, though of course differences between the patient and other species could be relevant in some medical contexts.

Finally, since, for these reasons, it is false that all human beings and only human beings show the features constituting normal human physiology, a species functional design cannot possibly be a species membership criterion – let alone a causal essence of the species as a natural kind. As we shall see in §II, disregard of these features of human bodily nature (HΦN) vitiates nearly all the arguments by philosophers of biology against a richly detailed human psychological nature (HΨN).

In a useful recent survey article, Kronfeldner, Roughley, and Toepfer argue for the “untried” theoretical option of “pluralism” about human-nature concepts. “Different scientific fields,” they suggest, “are in need of different concepts of human nature, each fulfilling an independent epistemic role” (2014, 649). They distinguish three such roles:

First, there is a classificatory concept of a *definitional human nature* This concept has applications in taxonomic sciences delineating species. Second, ‘human nature’ can refer to features of the characteristic *human life form*.... [T]his is a mere *descriptive nature* that is quite independent of any classification or explanation of that life form. ... Third, the characteristics of the human life form can be explained by evolutionary and developmental mechanisms or even by structural features of the human life form that are explanatory for the other features. If the term ‘human nature’ refers to any of these, we have an *explanatory human nature*. (2014, 649)

Such distinctions are all to the good. In these writers’ terms, species functional design is descriptive, but not definitional since not universal. It also seems to be explanatory in three ways, if not in others. Species functional design describes the mechanisms by which species members manage their characteristic

²An interesting example of such confusion is a fallacy too common among Catholic ethical writers. Such writers often argue that physical pleasure, as in eating or sex, is not intrinsically good for human beings, since it is a capacity we share with animals. But of course our animal qualities are just as natural to us as our specifically human ones. In Aristotelian terms, both genus and differentia characterize the species, not differentia alone. This fallacy might be called bootlegging neo-Platonism into Thomism, or Paul into Aquinas.

life pattern allowing their survival and reproduction, so it certainly explains individuals' fitness. And deviations from this design – dysfunctions – explain the phenomena we call disease. Finally, since HΦN has a genetic as well as a phenetic component, the former can explain how normal human gross structure and function develop across a highly variable range of typical environments. In all of these ways, HΦN does explain why species members, in Sober's phrase, "are the way they are" (1980, 352).³

My species-nature concept has some features in common with other recent nonessentialist ones, such as Boyd's (1991, 1999a, 1999b), Ramsey's (2013), and, perhaps especially, Machery's "nomological" idea of human nature (2008, 2018). Machery defines human nature as "the set of properties that humans tend to possess as a result of the evolution of their species" (2008, 323). My concept, like his, is of general, not specific, human nature and allows species nature to change over time (Machery 2018, 19-20). Unlike his, my concept excludes purely structural traits with no functional importance, so in that way it might be seen as a restriction of his. But there are also important differences. My functional traits do not need to result from evolution, since I defend a goal-contribution, not a selected-effects, analysis of function (Boorse 1976a, 2002). I would also not use the term 'nomological', since there is nothing lawlike about health. Unlike Machery (2008, 324), I view functional polymorphisms like sex as part of human nature. Finally, while Machery explicitly rejects any role for his concept in defining normality (2008, 326), my concept is precisely a model of the normal-pathological distinction in scientific biomedicine.

II. Philosophers of Biology Who Attack Human Psychological Nature by Attacking Species Nature in General

From 1977 onward, a series of philosophers of biology, including Lewontin, Sober, Hull, Ghiselin, and Buller, expressed varying degrees of skepticism about the idea of human psychological nature and its applications to ethics, law, and policy. All but two of their arguments rest on attacking species nature in general. Since this strategy invalidates HΦN as much as HΨN, it must fail. As we shall see,

³Roughley's own magisterial survey of human-nature concepts has an even finer-grained classification of elements (2021, §§1.2-1.3) than the Kronfeldner *et al.* paper. My species functional design best fits Roughley's fourth type of theory. It is not classificatory, but is explanatory – though not only of development, since it includes phenetic as well as genetic elements. It also describes a "privileged state," *viz.*, normal function. But it is not "normative" (Roughley's fifth element) in the true sense of value judgments, since there is only a strong presumption that health is good for its bearer.

this failure is concealed mainly by their neglect or misdescription of physiological examples and their misguided attack on the biomedical concepts of normality and pathology as lacking scientific basis. The simple fact is that Volume I of *The Great Book of Human Nature* already exists, and can be ordered overnight from Amazon. The only question is whether Volume II can be written, which is a wholly empirical question on which philosophy of biology has almost nothing to say.

Hull and Sober are not as narrowly focused as Buller on discrediting a rich psychological theory, let alone the specific one of evolutionary psychology. But both argue that since the idea of species nature has no biological basis, the notion of normal function therefore has none as well. Both writers also draw social and political morals from their analysis. I shall begin with Hull's brief essay (1986), since he seems to have started this line of argument with his (1978).

A. Hull

Hull (1986) begins by describing the idea of human nature as the belief that "all human beings are essentially the same, that is, they share the same nature, and that this essential similarity is extremely important" (24). This description already overstates the view, by minimizing all sensible modern HN theorists' acceptance of extensive human variation as natural and normal. At any rate, Hull next clarifies that, in biological terms, his target is the notion that a typical biological species, including ours, is "characterized by one or more characters which are both universally distributed among and limited to the organisms belonging to that species" (24) – in other words, found in all and only species members. Further, by 'characterized' he means "defined," since he takes species nature to be assumed to explain why organisms belong to a given species. It is a species-membership criterion, often reflecting an assumption that species are natural kinds of which such natures are the essences. He then proceeds to contrast this typological, perhaps essentialist, concept of species with the contemporary cladistic approach, in which an organism's species is defined by its ancestry. He denies that there are any characters found in all and only species members, mentioning human "retardates and dyslexics," and says that if there were traits universal to a species, an organism could have all of them yet be in a different species (25).

None of this, for the reasons I gave at the outset, threatens the kind of species physiological nature found in any textbook of anatomy and physiology. HΦN is neither universal to nor limited to human beings, so it is not a membership criterion, much less the essence of a natural kind. Yet Hull represents HN theorists as showing "consternation," feeling that

[b]iological species cannot possibly have the characteristics that biologists claim that they do. There *must* be characteristics which all and only people exhibit, or at least *potentially* exhibit, or which all *normal* people exhibit – at least potentially. (25)

Before attacking the “conceptual contortions” behind these ideas of potentiality and normality, however, Hull turns to the topic of “Universality and Variability.”

Hull’s goal in this section is argue for cultural variability – *i.e.*, to cast doubt on human cultural universals – by appeal to the analogy of genetic variability, which is well-established. He first faults anthropologists for assuming a connection between universality and innateness. He quotes Eisenberg as moving from the universality of language to its species-specificity, and thence to its genetic basis: “One trait common to man everywhere is language; in the sense that only the human species displays it, the capacity to acquire language must be genetic” (Eisenberg 1972, 126). To this Hull replies, first, that some human beings do have a genetic inability to acquire language, which refutes universality; and second, that the “actual distribution of language use” among humans or other animals implies “nothing ... about any ‘genetic basis’ for language capacity.” He then spends three paragraphs emphasizing the variability of human blood types: at the various genetic loci determining blood types, only one in 500 human beings has the most common joint genotype and phenotype.

I think Hull is straightforwardly wrong about the genetic-basis claim: if typical members of one species but no members of another can develop a certain trait, such as language, this phenetic difference can only be caused by genetic differences. Of a baby, a kitten, and a goldfish raised in the same family, only one will acquire language. This must be due to a difference in their genes; in a common environment, how else could it arise? But my main criticisms are two. First, it is a non sequitur to attack the thesis “Cultural universals derive from genetic universals” by stressing how diverse the genetic basis of some traits is. At best, what Hull needs to show is not that some human traits are polymorphic, but that all of them are. But, second, that is of course false. There is no normal variation among human beings in their number of eyes, or in any of thousands of other anatomical and physiological features in a textbook. By contrast, such a textbook will present blood type as a normal polymorphism -- normal because such variation in no way significantly affects the functions of the blood, whereas comparable variation in hemoglobin level or blood acidity, like variation in the number of eyes or heart chambers, would have drastic implications for these organs’ functions. To this Hull might reply that his critique is only of traditional HN theorists, who assume that HN is something that all and only humans have.

That is not so, however, as shown by his attack in the next section on normality as a “conceptual contortion.” Someone who says that HN is possessed by all *normal* humans is precisely granting that it is not universal, a species-membership criterion, an essence, and so on.

Hull next emphasizes intraspecies variability, both genotypic and phenotypic, and the lack of correlation between genetic and phenetic similarity. He says that HN theorists “discount” these facts by sophistry: “What do biologists know about biology? Organisms that lack a particular trait actually possess it potentially or else are abnormal for not possessing it” (28). I will agree that it would be silly to claim that anencephalic babies (28) are “potential” language users. But the standard biomedical view that anencephaly is objectively a dysfunction -- a total lack of normal human mental function -- is not sophistical, and nothing Hull adduces tends to show that it is.

He first notes that the normality concept has been abused in the past, to declare women defective men and blacks defective whites. But, as he grants, false judgments using a concept do not invalidate the concept; if they did, the species concept (as he does not mention) would be equally defective, since some 19th-century scientists believed human races to be distinct species. Hull therefore suggests searching for a “biologically respectable” notion of normality in one of three areas: embryology, evolutionary biology, and “functional morphology.” As to embryology, his argument against the concept of “normal development” is essentially Lewontin’s norm of reaction, which I will discuss in the next section; his use of it has the usual defects. He writes:

There is a fairly clear sense of “normal development,” but it is not very significant. As far as I can see, all it denotes is that developmental pathway with which the speaker is familiar in recent, locally prevalent environments. (30)

How can this remark apply to his own example of anencephaly? Until recently, there was no environment anywhere in which anencephalics could live for more than a day or two. How could the difference between life and death -- or even between typical mental abilities and total lack of them -- not be “significant”? As for evolution, Hull thinks that it shows that a species “nature,” if universal, is only temporary, even if change is very slow. But this criticism applies only to HN concepts that include universality, as our HΦN does not. Likewise, our HΦN need have nothing to do with “the individuation of our species” (31).

Hull finally makes a half-hearted gesture in the direction of our HΦN when he writes:

If by “human nature” all one means is a trait which happens to be prevalent and important for the moment, then human nature surely exists. Each species exhibits adaptations, and these adaptations are important for its continued existence. (30)

Moreover, he grants that “[t]he central notion of normality relative to human nature ... seems to be functional” (31). So once again one wonders whether Hull has any real disagreement with human nature as species-typical functional design. In any case, nothing he then says about functions is a threat to HFN as a concept of human nature, or, in most cases, is even relevant to it.

First, biomedicine does not use functional normality to “dismiss variation” (31); rather, it distinguishes normal variation from the abnormal variation that causes dysfunction. Second, there is no reason for anyone, HN theorist or not, to need a one-to-one relationship between structures and functions. Who denies that “a single structure can perform more than one function” and conversely (31)? The supposed difficulty of describing the “normal function of the hand” is illusory. Normal hand function requires full typical capacity for those hand and finger movements supported by typical human bones, muscles, and nerves in that area. It is false that “anything we can do with [our hands] is ‘normal’”: a broken bone, a ripped muscle, nerve damage, or a paralyzing disease like rheumatoid arthritis can make normal movement impossible. It is absurd to deny that the function of the testes, or of sexual intercourse, in producing sperm for reproduction is not a “biological” function (31). As writers of his ilk so often do, Hull is here just expelling anatomy and physiology from biological science.

Hull’s last example shows his unwillingness to make any serious engagement, not just with functional normality, but with the concept of function itself.

Finally, having blue eyes is abnormal in about every sense one cares to mention. Blue-eyed people are very rare. The inability to produce brown pigment is the result of a defective gene. The alleles which code for the structure of the enzyme which completes the synthesis of the brown pigment found on the surface of the human iris produce an enzyme which cannot perform this function. As far as we know, the enzyme produced performs no other function either. However, as far as sight is concerned, blue eyes are perfectly functional (32)

Earlier, Hull mentioned the “huge literature” on the function concept in philosophy

of biology. Yet he again elects to disregard it completely. If blue eyes are functionally equivalent to brown, and the deviant enzyme has no other ill effects, then the deviant gene is not, in fact, “defective” in any functional sense.

According to any of the four major types of analysis of biological function – based on past selection, present causal role in fitness, contribution to organism goals, or value judgments – there is no dysfunction in Hull’s example. So blue eyes are not, in fact, “abnormal” in the biomedical sense, nor are they so classified by anyone.

Hull ends by asking: “[W]hy is it so important for the human species to have a nature?” (32). He says a “likely answer” is to serve as a “foundation for ethics,” as suggested by Ruse and Wilson (1986). On the contrary: practically no HN theorists have ever taken Ruse and Wilson’s “Darwinian approach,” turning ethics into “applied science.” Much more typical is Hull’s suggestion that human psychological nature is viewed as a ground for human rights. His objection that the human species contains subgroups, such as males and females, who are not “essentially the same” in no way disproves the existence of sufficient commonality for these groups to have equal rights. And I shall argue in this paper’s sequel that a HYFN analogous to HFN, in my sense, is an entirely suitable foundation for human rights in ethics and law.

B. Sober

Elliott Sober’s “Evolution, population thinking, and essentialism” (1980) is a classic in history and philosophy of biology. But for all its many virtues, it includes a gratuitous attack on the distinction between health and disease, normal function and pathology, as unscientific – an attack unsupported, we shall see, by anything in Sober’s paper, and very likely later abandoned.

Sober’s aim is to clarify and expand upon Mayr’s contrast (1959) between pre-Darwinian essentialism in biology and post-Darwinian “population thinking.” According to Mayr, essentialism is the ancient view that

[t]here are a limited number of fixed, unchangeable “ideas” underlying the observed variability [in nature], with the *eidos* (idea) being the only thing that is fixed and real, while the observed variability has no more reality than the shadows of an object on a cave wall. ... [In contrast,] the populationist stresses the uniqueness of everything in the organic world. ... All organisms and organic phenomena are composed of unique features and can be described collectively only in statistical terms. Individuals ... form populations of which we can determine the arithmetic mean and the statistics of

variation. Averages are merely statistical abstraction, only the individuals of which the population are composed have reality. The ultimate conclusions of the population thinker and of the typologist are precisely the opposite. For the typologist the type (*eidos*) is real and the variation an illusion, while for the populationist, the type (average) is an abstraction and only the variation is real. No two ways of looking at nature could be more different. (1959, 28-9)

Although Sober agrees with Mayr that there is a crucial distinction between the two approaches and that Darwinian evolution replaces one by the other, he disagrees with how Mayr and other writers describe the difference. In fact, he notes (351-2), both essentialists and population thinkers grant the reality of both individuals (including individual variation) and groups or types. And contrary to what is often supposed, neither the mere fact of evolution – species change – nor the vagueness of species boundaries is fatal to essentialism (356-8). The former is because classic essentialists like Aristotle and Linnaeus countenanced the appearance of new species, and the latter is because essences, like “practically every concept applying to physical objects” (358), can be vague. Moreover, Sober thinks that essentialism does not even entail that species are natural kinds, as opposed to being historical individuals, the popular current view.

What is central to essentialism, instead, is, first, that species have essences. A species essence is a property that “all and only the members of that species possess,” (353) and possess *necessarily* by virtue of their membership. Moreover, this essence must be *explanatory*.

The essentialist hypothesizes that there exists some characteristic unique to and shared by all members of *Homo sapiens* which explains why they are the way they are. A species essence will be a causal mechanism which works on each member of the species, making it the kind of thing that it is. (354)

So analyzed, essentialism about any domain is “a scientific thesis” (354) -- a “perfectly respectable claim about the existence of hidden structures which unite diverse individuals into natural kinds.” And this thesis is sometimes validated, as in chemistry, where the essence of each chemical element is its “atomic number,” the number of protons in its atoms’ nucleus (355). Moreover, a classic example of essentialism, Aristotle’s natural-state model of explanation, can have valid scientific applications, as in Laplace’s analysis of the solar system (366). But in biology, Sober thinks, evolutionary theory invalidates the natural-state model in

two ways. First, that model fails as an explanation of individual variation within a species; and second, population thinking violates the essentialist requirement of “constituent definitions,” which define properties of populations in terms of properties of their members (350).

Sober illustrates the change from pre-Darwinian essentialism to post-Darwinian population thinking by various historical figures. Aristotle, the supposed⁴ classic essentialist, used his natural-state model in biology as well as in physics. Each type of thing had a natural state, but might be in an unnatural state due to interfering forces. In physics, gravity was explained as heavy objects’ seeking their natural place at the center of the earth. In biology, for higher organisms, Aristotle views perfect reproduction of the male as the natural state, with all other outcomes due to unnatural interfering forces. Thus all males who differ even slightly from their fathers, all females, and even some entire species are unnatural (361-3). It is at this point that Sober first makes his mystifying charge against health concepts: “Notice that our ‘modern’ conceptions of health and disease and our notion of normality as something other than a statistical average enshrine Aristotle’s model” (363). Next, Sober chronicles how Aristotelian essentialism gradually yielded to population thinking in biology. After the discovery of the Gaussian bell curve as a law of measurement errors, Quetelet, like Aristotle, continued to regard all individual variations as unnatural errors, in this case errors of nature rather than of human observers. By 1889, however, Galton had firmly rejected this view.

Bell curves are normal; they are found everywhere, Galton thought. This change in nomenclature crystalized a significant transformation in thinking. Bell curves need not represent mistakes made by fallible observers or by sportive nature. Regardless of the underlying etiology, *they are real*; they enter into explanations because the variability they represent is lawful and causally efficacious. (369)

Now given this summary of Sober’s historical story, we must ask: how can he possibly use it to charge the contemporary medical, and also (as we shall see) biological, concept of health with Aristotelian essentialism? No biomedical scientist today believes, like Aristotle and Quetelet, that all individual variation is pathological -- only that some is, namely, variation involving failure of species-typical part-function. Yet this is the logical gulf that Sober seems to leap over. At

⁴For a summary of recent work concluding that Aristotle was not, in fact, a taxonomic essentialist after all, see Roughley (2021), §1.3.

first sight, it seems that he cannot even run a guilt-by-association argument, since his target has no historical correlation with Aristotelian essentialism. Just as one cannot argue “Nazis built roads, therefore roads are bad” if everyone built roads, so one cannot argue “Essentialists distinguished normality from pathology, therefore the normal-pathological distinction is bad” if everyone uses that distinction. To discredit the distinction, what Sober needs to show is not that essentialism entails normality, but that normality entails essentialism.

Nor can anyone believe that HΦN, in my sense, is either universal to or limited to our species, and nothing in biomedical science requires thinking of any part of HΦN, or any other collection of traits, as a necessary species-membership criterion. As I explained in 1977, three years before Sober’s paper appeared in the same journal, “species design,” or HΦN, is just

the typical hierarchy of interlocking functional systems that supports the life of organisms of [a given] type. Each detail of this composite portrait is statistically normal within the species, though the portrait may not exactly resemble any species member. (557)

Although Sober does not mention my paper, perhaps his proviso “our notion of normality as something other than a statistical average” is a concession to it. But he offers no evidence that contemporary biomedicine has, in fact, any other notion of normality than the one I described: statistically typical part-function, which has no essentialist presuppositions.

In reality, both biology and medicine constantly employ attenuated natural-state models to describe and explain life phenomena -- but nonessentialist ones. To begin with, biology implicitly employs a notion of normal structure and function in its most basic activity, species description. To use Kass’s example, a “half-eaten butterfly” is not recorded as a morphological variant in the species. Rather, it is a case of normal structure partially destroyed by the environmental “interfering force” of a predator, such a bird. That is why an insect atlas does not show butterflies with every conceivable type of wing damage, as though they were polymorphisms. When entomologists write that ants “invariably” have six legs (Keller and Gordon 2009, 13), they are making a true scientific statement about normal ant anatomy – though in reality, the world is full of ants with different leg numbers, due either to injury or genetic or to developmental defect. This standard practice in no way conflicts with “population thinking.” Here is Mayr:

Modern biologists are almost unanimously agreed that there are real discontinuities in organic nature, which delimit natural entities that are

designated as species. Therefore the species is one of the basic foundations of almost all biological disciplines. Each species has different biological characteristics, and the analysis and comparison of these differences is a prerequisite for all other research in ecology, behavioral biology, comparative morphology and physiology, molecular biology, and indeed all branches of biology. (Mayr 1988, 331).

Here Mayr, Sober's arch-population-thinker, explicitly refers to a nonessentialist species design in the area of "morphology and physiology."

Similarly, any explanation of disease, in humans or any other organisms, involves a clash between the "natural state" of normal physiology and an interfering factor. Such a factor may be external, as when we explain syphilis or cholera by the effects of infection by specific bacteria, or internal, as when we explain phenylketonuria or Tay-Sachs disease by the effects of abnormal genes, or type-1 diabetes by the lack of the normal function of insulin secretion. Moreover, biologists, not just physicians, make explicit use of the disease concept. There are numerous journals, such as *Journal of Wildlife Diseases*, *Journal of Invertebrate Pathology*, and *Plant Pathology*, chronicling the diseases of all kinds of plants and animals, from elms and grasses to wasps, earthworms, crabs, salamanders, magpies, alligators, and hundreds of other wild creatures great and small (Boorse 2014, 698). Furthermore, leading evolutionary biologists, not to mention Sober himself, freely use the vocabulary of health and disease across the whole biological spectrum.⁵ In all these cases, it is hard to see how any such descriptions and explanations "emit even a whiff of essentialism" (Boorse 1997, 38).

If biology constantly uses health concepts implicitly and often explicitly, how can Sober imagine that they are without scientific foundation?⁶ Besides his

⁵See Boorse (2014}, 698 and 719-20 note 25, for such quotations from Maynard Smith, Haldane, Simpson, Dawkins, Huxley, Gould, Mayr, Williams, and Sober.

⁶In 1980, Sober stopped short of the conclusion that normality and health are not biological concepts. He wrote:

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

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

failed gesture at a guilt-by-association argument, his thinking is based on Lewontin's discussion of the "norm of reaction" of a given genotype to various environments (Lewontin 1977, 10-11). Here is Sober's summary of his own view in 1984:

All possible phenotypes of a genotype are "natural," since all are possible. This radically non-Aristotelian idea is codified in the genotype's *norm of reaction*.... The norm of reaction shows the different phenotypes a given genotype will produce in different environments. So, for example, we might graph the height that a corn plant with a particular genotype will attain as a function of how much water or nutrition or sunlight it receives. There is no such thing as its "natural" height. *We* may prefer a taller plant to a shorter one, and natural selection may, too. But the norm of reaction makes no such distinction. (1984, 160, 161)

So far, as an argument that the distinction between normal function and pathology is unbiological, Sober's reasoning does not even get off the ground. That is because it applies equally to the distinction between life and death, the most basic idea in biology. Surprisingly, Sober mentions this *reductio*.

When a corn plant of a particular genotype withers and dies, owing to the absence of trace elements in the soil, the Natural State Model will view this as an outcome that is not natural. When it thrives and is reproductively successful, one wants to say that *this* environment might be the natural one. Given these ideas, one might try to vindicate the Natural State Model from a selectionist point of view by identifying the natural environment of a genotype with the environment in which it is fittest. (375)

The perspective on development offered by the norm of reaction cuts very deep. ... Malnutrition while in the womb may reduce the child's intelligence; a well-nourished child may have its intelligence boosted by various enriching experiences. But through the complexity of these thousands of variables, we may think we discern the child's "natural" level of intelligence. ... The norm of reaction, on the other hand, makes this whole system of thinking look like a delusion. (Sober 1984, 160-1)

Given this shocking dismissal of the whole idea of normal vs. pathological child development as unscientific, I think it is fair to attribute to Sober the position in my text. [Footnote quoted from Boorse 2014, 719 n. 20]

Sober then says that this view violates Natural-State intuitions by making a drugged-up stud bull the healthiest bull, and also fails to vindicate the intuition that the natural state may be an ideal state never yet realized.

What these remarks show is that Sober's real argument is not just the norm of reaction, which would, of course, be moronic. After all, there is no principle "If two things are both possible, there can be no scientific distinction between them." Rather, his complaint is that normality vs. pathology cannot be defined in terms of fitness.

Our current concepts of function and dysfunction, of disease and health, seem to be based on the kinds of distinctions recommended by the Natural State Model. And both of these distinctions resist characterization in terms of maximum fitness. For virtually any trait you please, there can be environments in which that trait is selected for, or selected against. Diseases can be rendered advantageous, and health can be made to represent a reproductive cost. And even if we restrict our attention to historically actual environments, we still encounter difficulties. A perfectly healthy phenotype may be historically nonexistent; the optimum actually attained might still be some diseased state.⁷

Now a first problem with this charge is that, once again, it seems to apply equally to life and death. One cannot, for example, define death as zero fitness. Since fitness is measured by the number of offspring in the next generation, a dead organism can already have nonzero fitness; and as for future contributions to fitness, a live organism, such as a postmenopausal woman, may be just as unable to make them as a dead one. Sober apparently assumes that, despite the lethal regions of the norm of reaction, the life-death distinction is scientifically respectable. But we have not yet seen why he thinks so.

The key reason, surely, is shown by his references to "maximum fitness" and "the natural environment." Namely, for some reason Sober is assuming that there can only be one healthy height for the corn plant: the one achieved in its one natural environment. But that is not, of course, how our biomedical health concept works. Scientific biomedicine, unlike Aristotle, certainly recognizes normal variation, whether due to heredity or environment. And as one example, there is

⁷1980, 377. Note another mystery in Sober's reference to "both" the distinctions: why does he think that disease is something other than part-dysfunction?

no one unique healthy height for a corn plant. Rather, just as in human and veterinary medicine, plant pathologists separate pathological conditions from normal ones by part-dysfunction.

One standard textbook states:

When the ability of the cells of a plant or plant part to carry out one or more ... essential functions is interfered with by either a pathogenic organism or an adverse environmental factor, the activities of the cells are disrupted, altered, or inhibited, the cells malfunction or die, and the plant becomes diseased. (Agrios 2005, 5).

Thus, for example, some nematode species

puncture a cell wall, inject saliva into the cell, [and] withdraw part of the cell contents ..., resulting in dead or devitalized root tips and buds, lesion formation and tissue breakdown, and swellings and galls of various kinds (*ibid.*, 833)

Such injuries to root tissue often “decrease the ability of plants to take up water and nutrients from soil and thus cause symptoms of water and nutrient deficiencies in the aboveground parts of plants”(844). Some common infectious causes of stunted growth in corn, specifically, are high plains virus and maize dwarf mosaic virus.⁸

Naturally, the same effects could result from a lack of water or nutrients in the plants’ environment. Of such “abiotic stress” (Ashraf and Harris 2005), Agrios writes:

Environmental factors cause disease in plants when abiotic factors, such as temperature, moisture, mineral nutrients, and pollutants, occur at levels above or below a certain range tolerated by the plants.

Within that range, there is a corresponding normal range of plant height – not one unique normal height, as Lewontin and Sober assume. Outside that range, part-dysfunction occurs; within it, all heights are normal. But even within the range, a

⁸Nyvall (1999), 316, 319. To “stunt” a plant, according to Merriam-Webster, is “to hinder the normal growth, development, or progress” of it.

seemingly normal output can be masked by biotic injury. All of this is substantially identical to disease judgments in human medicine.⁹

In sum, therefore, Sober, Hull, and Lewontin go astray by failing to pay attention to basic features of scientific health concepts. Actually, Sober does this in two ways. The first is to suppose that the “healthy phenotype” is a “historically nonexistent” ideal state, rather than merely statistically species-typical part-function. And the second is, that, as we just saw, he assumes that scientific medicine, like his historic essentialists, denies the possibility of normal variation. Thus, while in the end we can clearly grasp his cornstalk argument, it rests simply on false premises.

Perhaps one or both of these false premises explain why Sober ignores the explicitly nonessentialist analysis of health and disease that I offered in the same journal three years before (1977). Theoretical health, I argued, is species-typical functional ability of every species-typical part. In disease, at least one species-typical part-function fails, while in death all species-typical part-functions fail. At least on selectionist or “S&R” analyses of biological function, this definition meets Sober’s challenge to define health in terms of fitness, whether past (selectionist) or present (S&R).¹⁰ But another reason may be that in 1980, as the above quotation indicates, Sober was suspicious of the notion of biological function itself. Later, however, he conceded that “[i]f function is understood [as by Wright] to mean adaptation, then it is clear enough what the concept means” (1993, 86). Perhaps we can presume that this concession renders his earlier discussion of health obsolete. What is clear is that he deleted his whole attack on normality from the reprint of his essentialism paper in a later collection (1994), and never returned to the topic of health and disease. So when Buller, in 2005, repeats Sober’s points to discredit evolutionary psychology, he may be relying on an argument that was already defunct in the mind of its author.

In rejecting a normal-function account of health despite its total freedom from any trace of essentialism as he describes it, Sober shows that he is simply unwilling to use the term ‘unnatural’ for anything but the impossible – which is

⁹It is also worth noting that a character’s normal range of variation differs in different species. A normal human child’s height ceases to increase somewhere around age 21, while a catfish or oak tree can continue growing throughout its lifespan. Normal human branching must stop at two arms and 10 fingers, while trees have no such limit.

¹⁰One can, of course, complain that my analysis of health leaves the concept vague, either because it is unclear what parts are species-typical or because dysfunction usually comes in degrees. But such a complaint is not open to Sober, given his earlier sensible recognition that vagueness affects “practically every concept applying to physical objects” (1980, 358).

not, of course, what anyone in biomedicine has ever meant by it. Near the end, he writes:

What happens in nature is simply everything that happens. There is no other sense of “natural.” ... It is no more a part of human nature to be healthy than to be diseased. Both kinds of phenotypes are to be found, and the norm of reaction makes no distinction between them.
(379)

By the same reasoning, it is no more part of human nature to be alive than to be dead. Not even life is part of any organism’s nature. Why? Because it is possible to be dead. But it is hard to see this as the “deep” insight that Sober supposed it to be. On the contrary, it is trivial. And in rejecting the thoroughly nonessentialist HΦN of physiology textbooks, he rejected all of medicine, most of biology, and an increasing fraction of his own specialty, philosophy of biology, as well – since the overwhelming majority of writers on biological function presume that biological traits can have (“proper”) functions, which is all that is required for a scientific concept of health and disease.

C. Buller¹¹

David Buller’s main target in *Adapting Minds* is evolutionary psychology (EP), one version of the subject formerly called sociobiology. But he claims that “the very idea of a universal human nature stands or falls” with some of his theoretical arguments. And his conclusion is that this idea is “antithetical to a truly evolutionary view of our species” (35).¹² Hence human nature is a “superstition,” “an idea whose time has gone.” Along the way to this conclusion, he says the same, more clearly than Sober, of the normal-pathological (or health-disease) distinction: it “has no foundation in biology” (48).

After some analysis of traditional theories of HN, Buller describes EP’s version as follows:

In sum, ... according to Evolutionary Psychologists, human nature consists of a set of adaptations that are presumed to be universal among, and unique to, human beings. (38)

¹¹For a critique of Buller complementary to my own, see Machery and Barrett (2006).

¹²Page references are to the selection reprinted in Downes and Machery (2013).

Actually, however, he quickly concedes – though he takes it back later – that EPists regard their HN as universal only among normal human beings. He says that they offer two arguments for the existence of such a HN. One (which I shall not discuss), based on genetic theory, is the “argument from sexual recombination,” claiming that all complex adaptations must be species-universal. The other, based on common sense, is the “argument from *Gray’s Anatomy*” (38). He quotes Tooby and Cosmides (1992) on the universal human bodily “architecture” found in that book.

[T]he fact that any given page out of *Gray’s Anatomy* describes in precise anatomical detail individual humans from around the world demonstrates the pronounced monomorphism present in complex human physiological adaptations. ... [W]e anticipate that in 50 or 100 years one will be able to pick up an equivalent reference work for psychology and find in it detailed information-processing descriptions of the multitude of evolved species-typical adaptations of the human mind. (Quoted by Buller, 39)

To this inference from physiology to psychology, Buller poses five objections. The first is that our bodies have adapted to fairly “stable and simple” physical features of our environment, like air composition; but our minds would have had to adapt to the variable and rapidly changing environment of social life. Second, Buller says the anatomical uniformity in *Gray’s Anatomy* is at a “relatively coarse scale,” whereas mental adaptations would have to be “much smaller-scale brain mechanisms” (40). This is exactly backwards. A standard textbook describes human physiology all the way down to the cellular and biochemical levels; any EPist would be ecstatic to be able to specify human psychology in even 1% as fine detail. The third and fourth objections are that we share most human physiological traits with other primates, and often with other mammals, vertebrates, and so on; hence these traits did not evolve during human history and cannot serve as a reason to believe in similarly complex human psychology (40). This point – one of Buller’s only two arguments even to touch EP – is entirely correct, and will be discussed in my sequel. Finally, he says that really “there is no single human anatomy and physiology” shared by all humans. Despite *Gray’s Anatomy*, some human beings have one kidney or three, situs inversus, or ambiguous genitalia, and there are more than 20 human blood types (40-1). As we have noted, mainstream biology and medicine view some intraspecific variations as functional or function-neutral polymorphisms (sex,

blood type, situs inversus) and other variations as pathological (one or three kidneys, ambiguous genitalia). The fact that one can live “a reasonably healthy” life (40) with the latter conditions does not show they are not pathological; one can also do so with countless diseases, from arthritis to athlete’s foot.

Consequently, Buller mounts an attack on the normal-pathological distinction – and from now on, what he offers are impossibility, not implausibility, arguments against HN. He says that “[t]his distinction between normality and abnormality, on which all claims regarding a universal human nature must depend, is part and parcel of a doctrine known as *essentialism*” (41). Here he seems to repeat Sober’s error: to discredit normality, he must show that normality entails essentialism, not that essentialism entails normality. At any rate, following Sober, he says that essentialism about a group is the thesis that it is a natural kind, defined by a causal essence that explains why its members are the way they are. He then quotes several EPists – Buss, Cosmides and Tooby, and Brown – explicitly using this language of essences defining natural kinds to describe HN. Such statements may well be unjustified, but they are easily separable from the EP project of finding a HΨN parallel to HΦN. Buller now asks how EP reconciles a species essence with abnormal members, and finds his answer in Sober’s Natural State Model, which distinguishes essence (the natural state) from variation caused by interfering forces. He says that EPists take the causal essence to be “universal developmental programs” that produce “the species-standard physiological and psychological architecture visible in all humans raised in normal environments” (Cosmides and Tooby 1992). But this cannot be true, since deviation from such architecture can be due to genetic, not just environmental, variation, as with anencephaly, where a part of the developmental program itself is missing. The truth is, therefore, that EPists cannot believe that species are natural kinds defined by causal essences, and they should avoid overexuberant language suggesting that they do. It is just false that “Evolutionary Psychologists are … committed to the idea that there are certain things that all humans share” (44) – only all normal humans.

Buller now runs through the Lewontin-Sober cornstalk argument against the Natural State Model, with one addition. Like Sober, he explores the possibility that EP can avoid the problem by specifying a “natural environment” for each genotype – in particular, the “environment of evolutionary adaptedness” (EEA) (45) in which normal human traits evolved. He sees two problems with such a reply. First, he says, it is “arbitrary to call a genotype’s EEA its ‘natural environment’,” because that genotype might have been fitter in a different environment that did not occur, while a different genotype could have been fitter in the one that did. Second, the EEA is defined in terms of selection. But selection is

only one cause of evolution, which can also result from “mutation, recombination, genetic drift, and migration” (47), and all of these processes are equally “natural.” None of this, however, really seems to threaten our model of HΦN. Buller is taking the idea of a “natural” environment far too seriously. All that anatomy and physiology need say is that normal part-function is assessed in typical environments where the species lives and has lived. It need not specify any particular environment, any more than it must pick one height that a healthy cornstalk must attain. Buller is simply recycling Sober’s own error here.

Buller concludes his section on normality as follows:

The distinction between “normal” and “abnormal” phenotypes, which is central to the Natural State Model, can’t be drawn by the norm of reaction. That distinction is imposed on biological theory from a nonbiological worldview. (47)

But it is bizarre to see Buller endorsing this conclusion, since he, unlike Sober, has written extensively on biological functions. Even in this chapter, he grants that the biological function of the human eye is to support vision. He even notes that “the eyes of the blind are human eyes despite not performing the typical visual function of eyes” (60). But the instant one accepts a statement of the form ‘The biological function of X is Y’, one has a distinction between health and disease, normality and pathology. In a normal organism, its X can perform Y in the situations where Y is typically performed; an organism whose X cannot perform Y in such situations has a pathological condition, such as blindness. That is what it means to be a pathological condition. I cannot imagine how Buller can simultaneously grant that the function of the human eye is vision, that there are blind people, and still claim that the concept of pathology has “no foundation in biology” (48). Similarly, if a fox runs off with a man’s testicles in its mouth, biology can certainly distinguish the effects of foxy and foxless environments. It is merely that contemporary natural-state models lack the two objectionable features of Sober’s classic examples: essentialism, and the thesis that all variation, rather than merely some, is pathological. Finally, unlike Sober, Buller fails to mention that life and death are equally regions of the norm of reaction, so, as far as anything he has said goes, the concepts of life and death also lack any biological foundation.

Buller’s third and longest section is on “essentialism about species,” the doctrine that every species is a “*natural kind*, which is defined by a set of essential properties.” This doctrine, he argues, is “absolutely and completely wrong” (49). I can summarize his case briefly, since the whole issue threatens HΨN no more than HΦN. He makes the following points:

1 Contemporary biology defines species by genealogy – relations of descent -- not by any list of intrinsic properties. Two members of the same species need not share any properties, and two organisms with the same properties can belong to different species.

2 Unlike natural kinds, which cannot change, species evolve and change their members' qualities over time.⁶

3 Species are not classes but individuals, since they have the three qualities that make an individual: spatiotemporal localization, continuity, and cohesion. Thus, organisms are not members but parts of species, exactly as cells are parts of organisms. So there is no more reason to expect conspecifics to be similar than to expect your heart and thumbnail to be similar.

4 Since there cannot be natural laws – “exceptionless universal generalizations” – about specific individuals, and because different people’s mental traits are mere homologues, there can be no laws about human psychological nature.

Buller concludes:

A truly *evolutionary* science of human psychology will not only abandon the quest for human nature, but, with it, the quest to be a science in the model of physics or chemistry.⁷ (61)

Unfortunately, he has already given the game away in the previous paragraph.

⁶Buller thinks this point poses a problem for EPists, since it is generally agreed that “we are the same species now that we were 150,000 years ago” (51), yet the qualities EP says “define us as a unique species” had not yet evolved. But this problem does not arise if EP simply stops saying its theoretical properties define *Homo sapiens*.

⁷It seems remarkably perverse to expect HΨN, any more than HΦN, to describe natural laws, simply because of the existence of pathological individuals. In genuine natural kinds, like chemical ones, the normal-pathological distinction has no application. Since the normal-pathological distinction for a kind is *inconsistent* with natural laws, it is absurd for Buller to claim that instead it *presupposes* it. There is no chance at all that the normal could be the nomic.

[A]lthough there can be no laws of nature that pertain exclusively to human psychology, psychology may one day provide us with richly detailed descriptions of human minds. And some of those descriptions may prove general enough to apply to vast segments of our species *for a particular period of time.* (61)

In other words, for all that he has said in this section, the core project of EP – to find a complex human mental functional architecture comparable to human physiology – can enjoy total, brilliant success. Despite EPists' occasional use of essentialist language, their core project no more entails essentialism about species than does physiological science. Actually, it is questionable whether there are any laws in biology at all.⁸ But whether or not there are, no one cares. If psychology reaches the same scientific success as physiology, psychologists will have everything they could possibly desire.

III. Conclusion

In sum, every one of the impossibility arguments by philosophers of biology against human psychological nature fails, simply because they would discredit HΦN equally with HΨN. What our writers offer is only elaborate excuses – not sophisticated, but sophistical – for denying the obvious. None of their arguments threatens the fact that the function of the human eye is to support vision, and therefore blind human eyes, like all other failures in the functions that constitute HΦN, are objectively pathological. Whether there is a HΨN comparable to HΦN is an empirical, not a philosophical, issue, on which philosophy of biology has and can have almost nothing to say. In this paper's sequel, I examine the plausibility of this thesis and its normative importance.

⁸For example, it appears that the Hardy-Weinberg rule, often cited as a law, is merely a theorem of probability theory.

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