

Chapter 7

The Archaeology and Philosophy of Health: Navigating the New Normal Problem



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Abstract It is often taken for granted that notions of health and disease are generally applicable across the biological world, in that they are not restricted to contemporary human beings, and can be unproblematically applied to a variety of organisms both past and present (taking relevant differences between species into account). In the historical sciences it is also common to normatively contrast health states of individuals and populations from different times and places: e.g., to say that due to nutrition or pathogen load, some lived healthier lives than others. However, health concepts in contemporary philosophy of medicine have not been developed with such cross-lineage, non-human, or diachronic uses in mind, and this generates what I call the ‘new normal’ problem. I argue that the new normal problem shows that current naturalistic approaches to health (when based on biological reference classes) are worryingly incomplete. Using examples drawn from evolutionary archaeology and the human fossil record, I outline an alternative, function-based strategy for naturalizing health that might help address the new normal problem. Interestingly, this might also reconstruct a certain uniqueness for humans in the philosophy and science of health, due to the deep history of obligate enculturation and cultural adaptation that archaeology demonstrates.

Keywords Health concepts · Pathology · Paleopathology · Cultural evolution · Biological function · Biological anthropology

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7.1 Introduction: The New Normal Problem

The bones of our ancestors can tell us many things about the quality of their lives at different times and places. In many cases, bones and other material traces generate clear signals of transitions in health and wellbeing, or at least in the normal indicators of health and wellbeing. One example of this is life expectancy. Our hominin lineage has become unusually long-lived compared to our closest extant relatives, the great apes, who become ‘elderly’ by their mid-30s. Dental evidence suggests that the developmental maturation of our *Homo erectus* ancestors was inconsistent with our own (Robson and Wood 2008), and some researchers argue on the basis of skeletal evidence that modern forager life expectancies were not achieved by *Homo sapiens* populations until approximately 40,000 years ago (Caspari and Lee 2004). A more recent and archaeologically unambiguous story can be told about how the hunter-gatherers who lived just prior to the advent of agriculture were healthier than the first generations of sedentary farmers. The remains of early farmers show them to have been significantly shorter, less robust, and comparatively malnourished: smaller, weaker bones exhibiting more signs of injury and disease, with missing teeth and in generally worse condition. It seems that their forager predecessors lived healthier lives, and (in this respect at least) were straightforwardly better off. Much ink has been spilled in addressing the obvious question accompanying this evidence, i.e., if the agricultural way of life made the lives of individual people worse, why was it adopted? Regardless though, the intuitive thing to say is that early Holocene populations were less *healthy* than their ancestors in the late Pleistocene, and such talk is treated as unproblematic in the literature on archaeology and biological anthropology.

However, there is a long-standing philosophical problem for such diachronic assessments of health. As Canguilhem noted in his 1943 *Essai sur quelques problèmes concernant le normal et le pathologique* (and its subsequent editions and translations), many conditions which in modern populations are seen as pathological were extremely common facts of life for prehistoric populations (Canguilhem 1989, p. 172). One small but well-evidenced example here is the hypoplasia, or underdevelopment of tooth enamel, which afflicted about 75% of Neanderthals (Ogilvie et al. 1989). Conversely, certain common facts of life for our contemporary populations, such as crooked teeth, were far rarer and stranger for those same ancient humans (and indeed foragers in general), because diets and dental development were more closely aligned (Ungar 2004). What is normal and typical changes over time as populations and/or their environments change, which means there is a *prima facie* problem in projecting the normal-pathological distinctions of one population (such as ourselves) onto discontinuous populations for whom those distinctions may be less relevant. This is the broad issue that this chapter aims to grapple with: understanding biological norms in ways that support attributions of health and pathology over evolutionary timescales.

This basic problem persists in contemporary philosophy of medicine. Health and disease are inescapably normative and evaluative concepts, in that the health/

disease states of individuals are ways of describing how well things are going for them, at some basic level. There is a voluminous literature on the nature of these judgements, roughly divided between the so-called ‘naturalists’, who see health or disease states as grounded in objective biological facts, and ‘normativists’, who argue that health or disease states should be seen as relativized (in some way) to factors external to the natural sciences, such as values, interests, medical practices, or experiences; and therefore as culturally constructed, subjective, or in some other way *irreducibly* normative. This brief sketch does not do justice to the diversity of debate within the literature (especially on the normativist side), with many nuanced theories and interpretations on the table (Boorse 2011; Cooper 2017; Kingma 2014) including hybrid positions (see, e.g., Wakefield 1992). In broad brush strokes though, we can characterize naturalism and normativism as trading off against two competing desiderata, i.e., (i) that being in a healthy or diseased state is to some degree scientific, in the sense of being a matter of mind-independent fact, and (ii) that the notion of health and disease we arrive at should be *humane*, in the sense of being relevant and immediate to the human experience of health/disease and/or medical practice. Much of the debate is centered around the acceptability of the trade-offs, especially with respect to how they deal with problem cases (such as in mental health).

Despite Canguilhem’s early contributions though, diachronic comparisons have been largely absent from the debates between normativists and naturalists, which tend to focus on concepts of disease and health suitable for contemporary clinical practice.¹ Conversely, the cognate science of paleopathology is concerned with differences over time with respect to the incidence and expression of disease conditions, largely without considering the philosophical question of why conditions count as diseases (Grauer 2012, 2018; Klepinger 1983). But from such a perspective it is intuitive to side with naturalism to some degree, i.e., to assume (as in our motivating transition cases) that there are some mind-independent facts of the matter about whether this or that individual was more or less healthy in some regard. This becomes especially salient when considering other hominins and non-human animals. For example, cancer has been observed within almost every branch of the tree of life where complex multi-cellularity evolved (Aktipis et al. 2015). All sides of the debate would recognize cancer as a biological phenomenon or *condition* that these organisms suffer from, but at least some non-naturalistic approaches would struggle to recover the intuition that it is still a *pathological* one for creatures lacking the cognitive or cultural sophistication to appreciate it as such, or where the spotlight of human attention is absent. The intuition to make sense of (or explain away) is that cancers (and other ‘fundamental’ diseases/injuries) represent ways in which things ‘go wrong’ for an organism entirely at the biological level. While we can recognize that disease, injury, and health are value-laden within the human social context, it is not unreasonable to hope for something to underpin this which is more biologically

¹ See for example Murphy (2020), and the absence of such considerations in significant handbooks and collected volumes (e.g., Carel and Cooper 2014; Humber and Almeder 1997; Huneman et al. 2015; Marcum 2016; Schramme and Edwards 2017; Solomon et al. 2017).

general and objectively grounded. The minimal naturalistic intuition is that at least *some* of the norms of health are firmly grounded in physiology, fitness, or other biological facts.

But if biological norms are to be objective in some sense, they should still be relativized to relevant biological populations. This seems unavoidable. For example, while the maximum life expectancy for humans is decades longer than it is for even the most pampered of chimpanzees in captivity (and by more than a factor of two when considering the oldest known individuals from each species), it is absurd to treat this a *health* comparison. What constitutes a healthy lifespan for a chimp (or for our most recent common ancestor) is not a matter for human biological norms to weigh in on, any more than humans should be subject to the biological norms of 200-year-old Bowhead whales. Unless strictly figurative or otherwise un-scientific, the terms 'long' and 'healthy' with respect to the lives of individual organisms only make sense in the context of the biological norms of their relevant biological populations (at a first pass, their species). Some sort of relativization to biological populations seems like a plausibility constraint. But coupled with the fact of common descent, this means that objective biological norms must also change and diverge over time.

This generates a problem: normative change makes archaeological evidence of diachronic health differences potentially ambiguous. It might be evidence that earlier or later populations were healthier or more diseased. But without assuming invariant biological norms, the difference might instead constitute evidence that those norms have changed, such that comparative health judgements between the two populations become problematic. After all, we know that biological norms must have changed in various ways during the evolution of our lineage. The challenge then is to filter the archaeological record of hominin health changes in a principled, suitably objective manner: which of the changes we see are changes in health status, and which are changes in what it takes to be healthy? Updating Canguilhem's terminology, the difference is between normal vs pathological on one hand, and the normal vs the 'new normal' on the other. This is the new normal problem.

The phenomena to save, in the face of the new normal problem, are common sense attributions of health differences across human/hominin populations, and the challenge is to do so in a way that fits what the archaeological evidence can tell us, without circularity. Objective biological norms cannot be invariant across lineages and over time. But it is equally implausible that they change whenever the population does—this cannot be the case if they are to be useful in describing the health effects of novel innovations, mutations, environmental changes, etc. The challenge is finding a useful middle ground. And the key question raised by the new normal problem is: what *kind* of changes license drawing a line between discontinuous regimes of biological normativity? Failure to address that question leaves naturalism about health incomplete, and unable to support attributions of health and pathology over evolutionary timescales.

This chapter is an attempt to explore that problem, and to consider how evidence from evolutionary archaeology might help guide formulations of biological normativity which avoid it. The ideal goal for biomedical naturalism would be a positive

account that is archaeologically informed, anthropologically sound, and which accounts for human health evaluations within an evolutionary context. The more limited goal of this chapter is to make progress in this regard by (in Sect. 7.2) providing an analysis of the failure points of one well-known naturalistic account, and (in Sect. 7.3) outlining several bodies of archaeological evidence for major transitions in health/wellbeing, which will assist in carving up that problem space in a principled manner. I will argue that a naturalistic account of human biological norms must be bolstered by a robust notion of biological function, but that human archaeology cautions against over-generalizing from non-human biology. This is because *cultural* evolution plays a key role in each of the problematic transitions. The unique reliance on culture to direct the development of human phenotypes raises difficult questions about how to demarcate characters as relevant or not to health and pathology, but (as I will cautiously suggest) the answering of those questions might also allow naturalism to be more humane.

7.2 Bolstering Biomedical Naturalism Against the New Normal Problem

The new normal problem for biomedical naturalism can be better explored by considering how it manifests for the most well-known naturalistic theory of health: the biostatistical theory of health, or BST, proposed by Christopher Boorse (1977) and further defended and updated in a series of responses to critics (Boorse 1997, 2014). Though there are more recent starting points for biomedical naturalism (Chin-Yee and Upshur 2017; Griffiths and Matthewson 2018; Matthewson and Griffiths 2017; Neander 1995), the BST is a useful reference point because the new normal problem is closely related to a known criticism of it. And the way that it falls short permits lessons to be drawn for naturalism in general.

7.2.1 Reference Class Problems

By way of brief summary, the BST defines health as the absence of disease, and diseases as impairments or limitations that prevent relevantly important functionality of an organism (or ‘normal functional ability’) from operating at levels that are statistically typical, given the reference class of the organism in question. Which functionalities are important and relevant is determined by their typical contribution to survival and reproduction, with the reference class being “a natural class of organisms of uniform functional design; specifically, an age group of a sex of a species” (Boorse 2014). Health then is constituted by having all your biologically significant functions operating as expected for your sex and age cohort.

One line of critique here (though by no means the only one) is to question the objectivity of these reference classes, the synchronic version of which is succinctly laid out by Kingma (2007). Boorse's rationale with reference classes (again, broadly speaking) was to follow medical practice in recognizing that some variable biological characters, such as hormone levels, play different adaptive roles for different people: men and women, young and old. Having a specific level of some specific hormone might be normal and healthy, e.g., for a young man, but aberrant and pathological in women and older men. In this way, Boorse hopes to keep statistical normality in alignment with intuitive judgments about health with as little 'splitting' as possible, being ambivalent about treating race (for example) as another reference class category (Boorse 2014, p. 702). Boorse is right to be resistant, as arbitrarily fine-grained reference classes would allow an absurd gerrymandering of the BST (until we each end up in a reference class of one, where being healthy is tautological). But what Kingma points out is that Boorse's line-drawing is dangerously ad hoc. It is one thing to identify a convenient set of reference classes which generate right-sounding results, but the biological objectivity of BST's health judgments depends on these reference classes themselves having a biological rationale. Boorse, in reply, is instead happy with having a *medical* rationale, i.e., to allow current medical practice to instead precisify the reference classes for BST, fit for current day clinical application (see Boorse 2014, p. 693). Whether this gives away too much of BST's aspirations for biological objectivity will not be considered here, though it will depend on how value-laden, conventional/pragmatic the reference classes of current medical practice are taken to be.

This debate becomes relevant for present purposes when it is generalized diachronically. The reference classes used in health statistics (e.g., of life expectancy or infection rates, etc.) are typically synchronic snapshots of populations, often broken down according to Boorse's envisioned reference classes, but also usually by territory.² Using such snapshots as reference classes for BST purposes, the average British man in his 40s (for example) will have biological norms against which his health can be assessed. Assuming his relevant functional abilities are indeed operating near the statistical mean for 40-year old British men today, the BST should rule that he is healthy. But it should also have made the same assessment (i.e., 'healthy') of the average British man of 150 years ago based on the statistical norms of that era, even if this person would be horribly unhealthy as measured by today's standards. Alternatively, if we rigidly adhere to snapshot reference classes then we cannot even make that comparison, as these two individuals simply have different biological norms, relativized to different reference classes. This recreates Canguilhem's discontinuity on an uncomfortably short timescale: there would be no objective basis for saying that British 40-somethings now are healthier than British 40-somethings 200 years ago (when an alarming proportion of them were dead or dying).

²It should be noted that this casts doubt on the *biological* objectivity of the reference classes used in medical practice, as 'you are healthy for a man/woman your age' typically carries with it an implicit '... in your country/territory/ethnic group'.

7.2.2 *Functions and Norms*

The full suite of problems arising from the diachronic reference class problem (for both the BST and biomedical naturalism in general) is something I explore more fully elsewhere. To be fair to Boorse, it is not clear he has any ambition to make biomedical naturalism applicable to diachronic contexts. The point is not that it is a bad idea to take the BST out of the clinic in this way (though it is a bad idea), but rather to illustrate and generalize the new normal problem in more detail.

The BST (at least the 2014 version) has three features which combine to make it unsuitable for application to evolutionary timescales:

- (i) it relativizes health evaluations for individuals against defined populations,
- (ii) it defines those populations synchronically and at least partially non-biologically, and
- (iii) its assessments of disease/health are based on statistical typicality within the populations.

The easiest of these to point to is the statistical assessment procedure (iii). Condition prevalence can ebb and flow wildly due to contingencies like epidemics and environmental conditions, and it seems implausible that biological norms too are tossed around this way. An assessment procedure which was less hostage to such contingencies seems preferable. As suggested, we should also address (ii) here, and find some objective way of fixing the qualitative and spatial/temporal limits of reference classes that does not rely on inference from standard medical practice (though perhaps at the price of diverging from that practice). However, we should also question relativization to populations *per se*. The BST uses different reference classes to grade people on their own curve, as it were. But this means that, even synchronically, it is hard to see how it can say of two average specimens from different reference classes whether one is more healthy than the other, as we intuitively can between typical 30 year olds and typical 80 year olds. Ideally, naturalists should want biological norms that can partially overlap, leaving some (but not all) inter-class comparisons intelligible.

I would argue that the BST's main rivals in the naturalist literature can already do this.³ Whereas the BST bases health/disease assessments on class-typical levels of class-typical biological function, these views typically attend to biological functions directly, where functions are either defined systemically or as selected effects. Rather than explore these alternatives in depth though, we can imagine functional approaches in the abstract, as a third potential relativization strategy in contrast to 'normativist' relativization and BST-style relativization to populations. The

³Approaches appealing to common accounts of biological function (such as the selected effects account) include Chin-Yee and Upshur (2017); Griffiths and Matthewson (2018); Matthewson and Griffiths (2017); Neander (2016); Wakefield (1992, 2007). The organizational account of function has also been appealed to for health-naturalist purposes (Saborido and Moreno 2015). The arguments in this chapter are intended to be broadly compatible with any of these, though I err on the side of language friendly to the selected effects account.

longevity comparisons between humans, chimps and whales show that health judgments must be relativized in *some* way, as health *simpliciter* makes less and less sense the further we range over the tree of life. Normativists of various stripes relativize to human values, conventions, interests, etc., and Boorse (despite protestations of sufficient objectivity) ends up relativizing the reference class component of his view to conventions concerning medical practice. The obvious move was to relativize the health of organisms to the natural populations they are members of. But, as Kingma points out, we all belong to many, varied natural populations defined by various combinations of (arguably) natural features—sex, species, lineage, sexuality, developmental stage, race, blood type, handedness, etc. At the very least, this requires a further naturalistic story to fix upon one of the many ‘natural’ groupings we fall under as normatively special for us, without over-compartmentalization.

The alternative relativization strategy I would like to suggest is relativization to functional design itself. Adaptive functional homologies might be a way of making sense of ‘ideal’ functional design across branching lineages, where this is independent of the statistics of contingent functional attainment,⁴ and of value-laden projection. In simple terms, this would involve considering functional complexes (like longevity or cardiac function) one at a time, and assessing health *in those respects* based on achieved functionality relative to the functions as evolved. On this approach, the population subject to a biological norm would be derivative of and relative to the relevant functions and their homologies.⁵ For example, chimpanzee and human biological norms of longevity presumably diverged because of selective pressures for longer lifespans in the post-divergence hominin lineage. We lack relevant functional homology with chimps in this respect, but (depending on the sequencing of the divergent selection) we share a normative clade in this respect with a smaller set of hominids with a more recent common ancestor. This will be similar for many cognitive functions (for example). But with respect to many other adaptive functions where things can go wrong, we would be part of clades with a deeper homology, for example with respect to aspects of cardiac health, or endocrine function, or vulnerability to certain cancers or mitochondrial diseases. Details will of course matter a great deal here, but function-relativization of biological norms might allow a more flexible regime of health judgments, some being very narrow and specific to human populations (e.g. as regarding dyslexia), and others with which we can make meaningful comparisons of health and pathology further across time and between lineages and morphs with shared functional heritage.

⁴Boorse, when pressed, makes a similar suggestion: “to the problem of typical disease, I see no solution but to retreat to a concept of ideal design which, so far, I am unable to define” (Boorse 2014, p. 707).

⁵I am using ‘homology’ here in a very loose sense. Functionally homologous groups (in my sense) would be the result of both common selective pressures that explain the origin of that function, and subsequent maintaining selection. They would therefore often be paraphyletic, i.e., subsequent divergent selective pressures could split some lineages away from the functionally homologous group. This may be a problematic use of the terminology (Griffiths 2006).

This is of course more of a theory schema than a theory itself, and I will not be defending it in any depth here. In any case, it can serve as a framework for what follows. We can assume that the best-pass version of biomedical naturalism will involve some appeal to biological function. To the extent that biological function is construed and constructed in evolutionary terms, naturalism will be highly dependent on the kinds of functional changes evidenced in the archaeological record: how robust they are/were, and whether they are likely to have been adaptive (as opposed to being the result of some other process of change). While the overall goal of this chapter is not to articulate a complete positive view, I will return to consider some directional recommendations in this regard in the concluding section, in light of the case studies to which I now turn.

7.3 Health Transitions in the Archaeological Record

So far, the argument has been both negative and abstract. In what follows, I consider what we can infer from the archaeological record regarding how our lineage changed during some distinctive physiological transitions, and what a philosophical account of health should ideally say about those transitions. Using modeling terminology, the goal is both to establish data points that an eventual theory of health should try to fit, and to look at how plausible human biological functions (and their evolution) might figure into such a theory.

7.3.1 *Gross Human Transitions and Discerning Adaptive Functions*

Human archaeology, broadly construed (i.e., to include human teeth and bones as well as material culture, the narrow conception of archaeology), is invaluable here. The majority of our biological functions of course have their origins much deeper in time; the functional homologies we have with other primates, mammals and so forth. But fossil evidence at those timescales is likely too low resolution to provide many clear transition examples. But archaeology can supply higher temporal resolution for recent transitions, including information about context, order, and causation.

There are numerous examples available, and we can begin by considering gross characteristic changes. Human beings are replete with evolved features that are unusual in the context of our nearest extant relatives, many of which are part of the suite of adaptations associated with foraging and the diet it allowed. The genus *Homo* is broadly marked out from preceding *Australopithecus* and divergent *Paranthropus* taxa as being more gracile and behaviorally sophisticated, with an overall trend toward less robust bones, smaller teeth, shorter guts, larger brains, and

complex lifeways and social interdependencies, up until anatomically modern humans. There is a (descriptive) sense in which this gross transition was an enfeeblement, especially for anatomically modern humans: we are much less robust and physically resilient than many of our ancestors and other archaic species. Is there also a normative sense in which this is true? I.e., are we less *healthy* in this respect? That seems like an implausible comparative judgement to make, at least in contrast to considering parasite load, or other incidences of common diseases, injuries, and afflictions. For example, Neanderthal skeletal evidence appears to show (i) that they were stronger with bones somewhat thicker and more robust than anatomically modern humans,⁶ but (ii) that they suffered from a higher incidence of serious injury (Berger and Trinkaus 1995; Spikins et al. 2019). It seems reasonable to say that they were no more or less healthy than us for having the bones they had (i.e., that these differences are just descriptive differences), but that they were less healthy for having them damaged more often. At least, the aptness of these propositions seems like an empirical matter that a philosophical framework for health judgements should respect, rather than pre-empt. Functional relativization would seem to allow this: morphological differences in the bones of the two lineages could be signs of innocent functional specialization, but they would also share a deeper common function (i.e., not to break).

Various evolutionary narratives seek to explain human enfeeblement as the result of tool use. Tools became force multipliers, enabling hunting and other high-value food sources, and a way of outsourcing much of the food-processing work previously done by chewing. One of the best sources of data here, fossil teeth, shows a nuanced and piece-wise trend (Ungar 2017). *Homo habilis*, one of the presumed basal species for our genus, appeared to have molars which were roughly similar size to *Australopithecus* (relative to body size), and larger incisors. It is not until *Homo erectus*, about 500,000 years after the first known stone tools, that teeth are appreciably small enough to indicate that a significant degree of food-processing is being outsourced to technology.⁷ Though fossil evidence at this the time-depth is by no means voluminous, it is still higher resolution than in deeper time periods, and indicates a likely sequencing of changes in environment and organism.

The usual cautions against adaptationist thinking should of course be kept in mind. But we can still draw reasonable conclusions. For example, the inference made by Ungar is that the reduction in human dental (and skeletal) robustness is adaptive, due to an alleviation of the selection pressures that previously demanded developmental investment in those structures. This is reasonable and again fits function-relative naturalism, given the evolutionary trajectories discernible across plausible reconstructed evolutionary trees for the hundreds of attested early hominin fossils. Even if we did not have modern humans as a baseline object of study, it is beyond implausible that, e.g., the hundreds of *H. erectus* specimens we have

⁶Though this should not be over-stated, see Pearson et al. (2006).

⁷However, tooth shape in early *Homo* species had already changed to become more capable of eating tougher and more elastic foods than the hard, brittle foodstuffs that *Australopithecus afarensis* specialized in (Ungar 2004).

found (scattered over Africa and Eurasia, and across almost two million years) were all pathological individuals whose dental development had been stunted in more or less the same way.

However, interesting debates sometimes arise between adaptive transformation and maladaptive pathology, especially when it comes to describing new species from initially small sample sizes. For example, the famous and surprising discovery in 2003 of the LB1 partial skeleton in Liang Bua cave on Flores, one of the Lesser Sunda islands in Indonesia, led to conflicting claims about whether this was a new species, or merely a pathological specimen of a known species. Because it was initially dated as living during the known settlement time of *H. sapiens* in the region, and because of its unique ‘mosaic of primitive and derived traits’, small stature and low endocranial volume, the describing authors considered and rejected the possibility that LB1 was a modern human afflicted with IGF-related postnatal growth retardation, pituitary dwarfism, or microcephalic dwarfism (Brown et al. 2004; Morwood et al. 2004). Their description of LB1 as a new species, *Homo floresiensis*, was challenged however by further claims of alternative, pathology-based explanations of its condition. These included secondary microcephaly (Henneberg and Thorne 2004), myxoedematous endemic cretinism caused by congenital hypothyroidism (Obendorf et al. 2008), and Down Syndrome (Eckhardt et al. 2014; Henneberg et al. 2014). These explanations have been rejected by subsequent comparative studies (Baab et al. 2016; Brown 2012). Subsequent re-dating and phylogenetic analyses of morphological characters now suggest that the *H. floresiensis* lineage originated prior to *H. erectus* and was likely extinct before *H. sapiens* arrived in the region (Argue et al. 2017). Teeth and statistical inference also come to the rescue here, as dental fragments from several other individuals were also found in the Liang Bua cave, and there is enough commonality of characteristics to make it unlikely that LB1 is radically unrepresentative of that small population (Kaifu et al. 2015). It is likely then that *H. floresiensis* is a genuine hominin lineage with distinctive small-bodied physiology and associated biological norms evolved (presumably) according to insular dwarfism (Montgomery 2013; Tucci et al. 2018).

One point to emphasize here is that scientific methods can tell the difference between functionally defined health and pathology in a population by pinning down the variation within it. There are more ways of being unhealthy than being healthy, in the same way that there are more ways of being far from the fitness maxima than being near it. The function-relative theory schema therefore sounds plausible at this level. To a first approximation, selection and adaptive optimization will tend to drive convergence to a functionally adaptive phenotype, with pathologies then impinging to divert organisms from this (healthy) phenotype in a variety of ways and degrees. It would be very difficult even for an endemic pathology resulting from persistent, detrimental environmental conditions, to mimic the distribution of a stable, more-or-less optimally adapted population. Pathology should therefore leave at least a

statistical signal in the fossil record, given enough available specimens.⁸ Evidence of convergence on a trait within a biological population (again, generally speaking) might then be a good sign that the trait is associated with health, if health is understood in some way as approximating adaptive well-functioning.

There will no doubt be devils in the details, but at this level of description at least, relativizing health judgments to adaptive functions allows us to draw some reasonable conclusions about health in the face of evolutionary change. Regarding the gross human transition: rather than an enfeeblement, we can see the shortening of our gut, reduction in bone mass and teeth size (etc.) as healthy adaptations which coevolved with our larger brains and the technologies (physical and social) that they made available—stone tools, fire control, cooperative complexity, paternal investment in children, etc. We are considerably less robust than Neanderthals, and weaker than chimpanzees. But given our ancestors' other advantages, including their complex suite of cultural adaptations (Henrich 2015; Laland 2017), they did not need to invest as heavily in developmentally expensive bone and muscle, and these resources were better spent furthering survival and reproduction in other ways. So, while *H. sapiens* might have been descriptively enfeebled, we are no less healthy for it, assuming that the weakening was part of overall phenotypic adaptation. We share many functional adaptations with our cousins and ancestors, and so we can recognize many of the same diseases across the clade which run counter to our common biological norms by impairing those functions. But insofar as we have our own distinctive adaptive biological functions, our biological norms (and potential pathologies) will differ in those respects.

Function-relative naturalism seems to get the basic intuitions right here, and successfully addresses the change-vs-new-normal problem. In principle, it offers a biologically grounded, objective and mind-independent way to determine the scope of distinctive human biological norms, in a way which still lets some of them extend to archaic populations. If so, valid comparisons of health and disease can still be made qua specific common biological functionality and associated pathologies. And this approach may allow us to push back against Canguilhem's concerns about the normal/pathological distinction in archaic populations. Unlike the BST, it can recognize that, as it is for us, dental hypoplasia *was* pathological for Neanderthals despite being endemic among them. For both lineages, dental hypoplasia is not a functional adaptation, but instead a developmental impairment⁹ working against a functional adaptation with a deeper homology. Of course, the success of this sort of move depends on there being plausible evolutionary narratives to support it, and this would have to be defended on a case-by-case basis. But the reliance on a plausible narrative is also the point: this is an approach which leaves discernible empirical facts in the driver's seat.

⁸Though lack of convergence is by no means definitive proof—drift and other evolutionary processes can be sources of variation that are fitness-neutral and not pathological.

⁹As hypothesized by Ogilvie et al. (1989), the widespread underdevelopment of the tooth enamel in their sample (of 699 crowns) is indicative of nutritional stress from weaning to adolescence.

7.3.2 *Recent Transitions and the Question of Culture*

Before acknowledging the open questions for a function-relative approach (e.g., what ‘function’ is supposed to mean, and the difference between functional adaptation and adaptive function, etc.), there is another important point to acknowledge: the causal role of culture. As mentioned, we should be cautious of biasing attention toward ‘human uniqueness’ and human-distinctive adaptations, since the majority of biological functions and norms will have deeper origins. But it is an unavoidable feature of recent functional adaptations that they involve cultural traits as well as classically biological characters.

Technology is strongly implicated in the gross human transition, and is treated as characteristic of the later hominin phenotype, including (accurately or not) the *Homo* genus as a whole. Contemporary and ethnographically described foragers such as the Hadza and Inuit simply could not survive in their environments without specific tool kits, and detailed skills and knowledge with respect to their manufacture and use (Henrich 2015). This dependence of course evolved earlier in the *Homo* lineage, with respect to more archaic technologies such as stone axes and cleavers, and the control of fire. Fire provided material advantages in the form of heat for cooking, and for generation of further cultural adaptations, such as resins and heat-tempering. The shortening of the gut as the result of liberated calories is just one physiological change this supposedly fed into (Wrangham 2010). Another apparent consequence is our truncated sleep cycle: fire effectively extended the hours of daylight and the time available for social activity. Adult humans sleep less than our great ape relatives (who sleep from dusk to dawn), though we appear to compensate for this by sleeping more deeply (Nunn and Samson 2018; Samson and Nunn 2015).

But characters like the control of fire or mastery of stone knapping (as well as undoubtedly many other early *Homo* technological skills which did not preserve as well) are not transmitted vertically as part of our classical biological heritage—as anyone who has tried their hand at knapping will appreciate. They are culturally transmitted, sometimes through social learning and/or teaching, and sometimes through trial and error learning in an environment which scaffolds that learning (Sterelny 2012). But it is reasonable to think of stable, reliably reproduced, culturally transmitted skills and knowledge as integrated into the human phenotype—at least, if ‘phenotype’ is understood in the ecological sense of how well an organism can do in a given environment. A forager who does not know how to forage is not really a forager. Such skills and knowledge are straight-forwardly referred to as ‘cultural adaptations’ by some (Boyd et al. 2011; Henrich 2015; Perreault 2012), in that they provided unequivocal fitness advantages and were ‘selected for’ because of those advantages,¹⁰ but via social learning mechanisms rather than (or in addition

¹⁰Cultural evolution theory is divided regarding whether ‘selection’ is the best way (or even necessary at all) to describe the mechanisms by which successful cultural traits are propagated and/or upregulated. See for example Tim Lewens’ taxonomy of cultural evolutionary thinking and related discussion (Brusse 2017; Heyes 2016; Lewens 2015). Indeed, clarifying relevant notions of suc-

to) natural selection. To cover the semantic bases here of transmission and upregulation (selectively or otherwise) we can call them ‘culturally propagated’.¹¹

Cultural traits and social learning might therefore be quite deeply implicated in human evolution, certainly in its most recent chapters, and this poses interesting questions for the function-relative approach to health we are considering. Suppose, as seems likely, that some of our traits are i) culturally propagated, ii) selected as ecologically adaptive, and iii) developmentally integrated, in the sense that they are built up over time, especially in early life—examples here might include specialized forms of cognition (Heyes 2018). There is no obvious, non-question begging reason to rule out such traits and characters from inclusion in the suite of adaptive functions we might otherwise take as underpinning biological normativity. Therefore, some failures of adaptive enculturation or cultural development might be just as pathogenic as deleterious mutations, malnutrition, or environmental mismatch. Though this could be a theoretical minefield (for reasons alluded to in passing), it could also be an advantage for biomedical naturalism. For example, depending on the details of cultural function, it might allow naturalism to take a more nuanced approach toward (for example) some mental illnesses than under a more crudely biological ‘brain malfunction’ paradigm. At the very least, it makes conditions such as dyslexia more naturalistically intelligible as pathologies, given the likely reality that dyslexia is the failure of an underlying cognitive system that has only recently been exapted by cultural evolution. These are of course open questions that will depend on how biological/cultural functions (and their relationships to health) are specified.

7.3.3 Functional Adaptation and Culture in Behavioral Modernity

With these frameworks and caveats in mind, we can now return to the two candidate cases for human health transitions we began with: the (allegedly) dramatic increase in longevity in humans of the Early Upper Palaeolithic, and the Neolithic revolution. In both these cases, cultural processes are clearly implicated which may challenge the function-relative theory schema, and in both cases difficult interpretive questions are opened up, illustrating the ways in which the viability of this approach will have to be decided.

cess and cultural analogues of fitness and adaptiveness are non-trivial (Ramsey and De Block 2017), meaning that the idea of a cultural adaptation should be approached with reasonable caution. I am assuming that some such useful account is possible for the purposes of this discussion, though perhaps only in a very loose sense.

¹¹ This is not to say that the basic cognitive capacities to acquire traits did not evolve through biological natural selection, but even this is disputed (e.g., by Heyes 2018, who argues that there is a degree of cultural propagation even for the enabling capacity-traits of cultural propagation).

7.3.3.1 Longevity

In a pair of papers (Caspari and Lee 2004, 2006), Rachel Caspari and Sang-Hee Lee used dental age-at-death estimates of fossil hominids to estimate the ratio of mortality between old and young individuals for hominid groups in different times and locations. In the first study, 768 fossil hominids in four groups were analyzed: later australopithecines, Early and Middle Pleistocene *Homo*, West-Asian/European Neanderthal, and European Early Upper Palaeolithic *H. sapiens* (50–30 kya). The authors found both a chronological trend toward increased longevity (consistent with a gradual increase in longevity over human evolution), and a five-times higher old-to-young ratio in the Early Upper Palaeolithic group compared to the Early and Middle Pleistocene group. Caspari and Lee hypothesized that this striking difference was due to an increased reliance on cumulative cultural innovation in the Middle to Upper Palaeolithic transition, whereby cultural complexity meant that senescent individuals (i.e., beyond reproductive age) became more valuable and prestigious as reservoirs of skills and knowledge. In their 2006 paper, the old-to-young ratios of four populations were compared: European Early Upper Palaeolithic *H. sapiens*, and three populations were from the prior Middle Palaeolithic—West Asian *H. sapiens*, West Asian Neanderthals, and European Neanderthals. At this time (prior to known *H. sapiens* migration into Europe) the *H. sapiens* and Neanderthal populations in West Asia are culturally indistinguishable in the archaeological record. Overall, these populations had three cross-cutting domains of similarity which allowed meaningful comparisons to be made: the three Middle Palaeolithic populations were culturally similar, the two West Asian Middle Palaeolithic populations (*H. sapiens* and Neanderthal) had similar environments, and the two *H. sapiens* and two Neanderthal populations of course each had closer phylogenetic affinities. According to their analysis of comparative age-at-death ratios, the two *H. sapiens* populations (separated by time and geography) were found to be significantly different, which the authors see as attributable to either environmental or cultural differences. The contemporaneous West Asian and European Neanderthals were different, implying environmental differences (as might be expected). But the contemporaneous *H. sapiens* and Neanderthal populations in West Asia were similar, suggesting that merely biological differences between the lineages was not a strong causal factor.

These papers are intriguing both in their purported findings, and for their place in the context of two contentious debates at the time: i) concerning the so-called grandmother hypothesis and its sequencing, and ii) concerning the now mostly debunked ‘human revolution’ theory of a rapid transition to human behavioral modernity in the European Middle-Upper Palaeolithic (McBrearty 2007). Briefly, the grandmother hypothesis agrees that longevity increase was driven by social changes whereby the contributions of post-reproductive individuals became more valuable. Advocates of this hypothesis such as Kristen Hawkes typically attribute this effect to grandmothers contributing to the fitness of reproductive relatives (i.e., their daughters), but at a much earlier stage than the Middle-Upper Palaeolithic, and proceeding more incrementally, driven by kin selection rather than cultural

evolution. Hawkes and co-authors have expressed skepticism about Caspari and Lee's results, arguing (a) that skeletal assemblages are unlikely to represent unbiased samples of the age-structure of populations, and (b) that longevity and old-to-young statistical ratios are by no means proxies for one another, as evidenced by the similarity of old-to-young ratios in living non-human primates despite very different life expectancies (Hawkes and O'Connell 2005; Hawkes and Coxworth 2013).

Regardless of the wider debates, this summary can still serve as an instructive hypothetical case study. Assume for sake of argument that Caspari and Lee are correct, and there was a dramatic increase in longevity because of a cultural change. How should a function-relative grounding of health judgments treat this case? That might depend on exactly how the culture changed and how we interpret that change. Would this be a case of better health achieved though cultural change alleviating excess mortality, or would it be an alteration of the altered *H. sapiens* phenotype via cultural adaptation, and therefore a non-valent change with respect to the relevant norms of human health? This is effectively a resurgence of the new normal problem with respect to the culturally-driven functional traits being considered as candidates for normative relevance alongside paradigmatically biological functions.

But the problem here has a somewhat different aetiology, and stems from a lack of interpretive unity within contemporary cultural evolutionary theory. On one hand we might take robust, intergenerational cultural innovations to be part of an extended human phenotype, and therefore take Boyd, Henrich et al. literally: cultural adaptations are adaptations, and cultural functions are functions. Alternatively, cultural innovations, their substratum being stored externally to the traditional organism in the form of practices, rituals, institutions or artifacts, might instead be seen as more like an engineered niche, à la cultural niche construction, in being just a conveniently, reproducibly modified part of the environment. On this reading they would scaffold behavior and development, but they are not themselves part of the phenotype, and so are not functions in the relevant sense. These two approaches would give very different answers to the new normal problem, yet at least some argue that the choice between them is effectively a modeling decision (Sterelny 2010). If so, this seems like a liability all of a sudden for function-relativism: it seems like a bad thing if a modeling decision were to decide whether a condition counted as healthy or not. Without a way to resist this sort of interpretational pluralism, the function-relative approach would fail to be clearly objective, and in a similar way as the BST, i.e., with respect to grounding out in convention or utility (in this case explanatory convention/utility rather than clinical).

This is not to say that this problem is unanswerable, but answering it is beyond the scope of the current discussion. It will hinge on the nature of cultural adaptations and how and when (if ever) to take them seriously as adaptive functions in the literal, phenotypically-constitutive sense, and it is therefore part of a much broader

debate about the overall status of cultural evolution and the extended evolutionary synthesis.¹²

7.3.3.2 The Neolithic Transition

The final transition I want to revisit in more depth is the Neolithic transition from foraging life to sedentary farming (in the Middle East, from about 11 kya), which presents another interesting problem case. Prior to the work of a number of archaeologists in the 1960s,¹³ the dominant explanations were self-flatteringly progressive. The assumption was that settled agricultural life was a straightforwardly rational improvement on forager life with more security, ease, and leisure (Barker 2006), and so simply inventing the technology was enough to kick off settled civilization. If that were really the case, then health and ‘civilizational’ cultural adaptations would seem to go hand in hand. However, ethnographic studies of hunter-gatherers like the !Kung-San put lie to this, as even in inhospitable environments foragers enjoyed relatively easy and secure lives compared to hard-toiling traditional farmers, with more healthy, diverse, and fungible food sources, and a lower parasite load. This comparison would have been much worse for early farmers with a much narrower range and quality of domesticated crops, a fact now conclusively attested to by skeletal and environmental evidence showing a marked decline in bone density, stature, and nutrition, and a corresponding increase in cavities, damage, and conditions which usually serve as markers for pathology and poor quality of life (Larsen 2006).

Explanations of this run the gamut. One is sedentism itself: once farming productivity reached a level capable of providing full subsistence (as opposed to occasional supplementation), a switch to sedentary farming could increase effective fertility by supporting raising more children than a mobile life would allow. The gradual encroachment of farmers onto forager lands might likewise be explicable economically/militarily, via the order of magnitude greater increase in carrying capacity for land under the plough (Diamond 1998). And the increased strategic value of land might also make the difference, so that hundreds of sickly farmers defending fixed investments would out-fight (in both strength and morale) dozens of relatively healthy foragers who have the option (for now) to move on and hunt elsewhere. But the flipside of this is greater vulnerability and deep planning horizons: take away everything a forager owns and they are less well-equipped foragers, but take everything from sedentary farmers and they cannot farm at all. Social solidarity and bonding mechanisms such as collective religions and ideologies, and associated

¹²For example, we might perhaps understand cultural adaptations as adaptations proper to the degree that they are intergenerationally stable and partake in other paradigmatic features of adaptations, in a multi-dimensional conceptual space (*à la* Mitchell 2000), though this obviously presents further challenges.

¹³Famously culminating in the conference and subsequent book *Man the Hunter* (Lee and DeVore 1968).

assurance mechanisms and cognitive biases may have taken on a much greater role as a result, and there are numerous theories and modeling approaches along these lines, both adaptive (Brusse 2020; Bulbulia 2004; Handfield 2020) and maladaptive (Norenzayan et al. 2016). In short, the transition to sedentary farming lifeways might have been i) an unpleasant trade-off for greater fitness, ii) a forced but still rational choice (Sterelny 2015), iii) a fitness trap, or iv) driven by maladaptive or fitness-orthogonal cultural evolution.¹⁴

What to make of the options here? Simplifying greatly, there are two binary judgments to be made. First, are the health differences caused by the transition validly comparative? I.e., is it fair to say that early farmers had worse health than the preceding foragers? Second, were the causes of the transition functional adaptations? Intuitively, the first answer seems like an unequivocal ‘yes’—this is exactly the sort of bio-anthropological judgement we want a naturalistic theory of health to accommodate. But if the answer to the second question is also yes, this will spell trouble for the proposed theory schema, in that it would give the intuitively wrong answer to the new normal problem in this case. If the shift to farming lifeways was the saltation of a suite of cultural adaptations, then this might just be the new adaptive phenotype, which trades off a more miserable form of enfeeblement for increased overall fitness. The problem is that the form of enfeeblement here looks very much like malnourishment, or at least the prevalent environmental susceptibility to it. This prospect seems much harder to swallow than the gradual weakening of *H. sapiens* discussed earlier.

Again, (a) it might be seen as a virtue of the function-relative approach that it is so much a hostage to empirical fortune, but (b) figuring out exactly how much danger it is in here would be extremely involved. Everything hinges on the empirical details and how to interpret them in answering the second question. Over and above clarifying the nature of adaptive (bio)cultural functions, the ideal strategy for a function-relative approach, to retain its full scope and potential, would be to show that only the most plausible transition-drivers met that standard. This would be easier with a restrictive standard for cultural adaptations, though this of course would trade off the potential advantages of cultural function-relativization with respect to ‘humanizing’ biomedical naturalism.

7.4 Conclusions and Prospects

Time to take stock. I began this chapter with the new normal problem, which looked like a curious dilemma for both common sense uses of comparative health judgments in archaeology, and for naturalistic theories of health in general. The main point of the discussion was to demonstrate what it would take to construct a naturalistic conception of health that is *complete*, in the sense of being able to address the

¹⁴This is of course also bracketing off exogenous drivers like climatic change.

new normal problem and also being *applicable* naturalistically, outside narrow clinical applications. It is easy to make the case that naturalism should do better than the BST, and do better than relativizing health and disease to fixed biological populations. Relativization to biological functions solves many problems. And, for at least some cases of health changes in the human archaeological record, a function-based approach also seems to make plausible judgments. Speculatively, there might be advantages if cultural adaptations were taken into account, with respect to a wider scope for the content of such judgements, opening up less narrowly biological assessments of health and pathology, perhaps more in line with the judgements of normativist theories. What is currently missing from this picture are objectively justifiable *inclusion criteria*, to adjudicate such functions as either internal or external to health-relevant biological (or biocultural) normativity. But there is also no apparent justification for ignoring them, or for pretending that culture and biology can be neatly separated in this context.

In any case, this approach is extremely dependent on empirical evidence from archaeology and evolutionary anthropology, and how it is interpreted. In exploring this, a number of sticking points were bracketed off, with the conclusion here largely programmatic and several philosophical questions left wide open. Some of these are very broad, and arguably more imposing than the original problem which led to them: what are biological functions, in this context? To what degree are these functions grounded in culture? How and to what extent are these integrated into human phenotypes? If they are integrated to some degree, are there health states or pathologies which can be objectively associated with them? These questions become more acute when culture becomes a significant driver of phenotypic changes. Pinning down the answers to many of these questions better would be crucial to making progress toward a more complete conception of biological norms, and how to make sense of health and disease from any given naturalist and evolutionary perspective.

In attempting to extend the scope of health judgment beyond the strictly medical context and out to archeologically-relevant populations, it is ironic that human archaeology also makes it plain that grounding these judgements objectively will not be simple. We are likely to miss the mark if we simply use traditional notions of biological adaptation and function which downplay the distinctive contribution of culture to human phenotypes. Humans of every species and era were always cultural organisms to some degree, and our understanding of their health and well-functioning will need to respect the stories that both their bones and their material culture speak to us.

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