**FUNCTIONS AND HEALTH AT THE INTERFACE OF BIOLOGY AND TECHNOLOGY**

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Synthetic biology promises to eliminate the distinction between biology and engineering by delivering a philosophically interesting new kind of entity: a biological organism that is wholly designed and constructed by humans. The possibility of such organisms raises interesting questions in three domains: the analysis of (1) biological functions, (2) engineering functions, and (3) health and disease. This paper identifies and systematically answers these questions. This does not only establish how we should think about functions and health and disease in synthetic biological organisms, but it also reveals insights that are of broader relevance: (1) aetiological accounts of biological function need to omit or reinterpret reference to *natural* selection. This results in complete continuity between aetiological analyses of function in engineering and philosophy; (2) considering synthetic biology prompts interesting further questions about heritability, ancestry, and biological individuals; and (3) accounts of disease as biological dysfunction do not straightforwardly map onto our intuitive health and disease judgments regarding non-human animals. In response to the latter point I examine three possible avenues, and tentatively defend one on behalf of the ‘disease as dysfunction’ theorist.

Keywords: Biological Function, Functional Analysis, Health, Disease, Synthetic Biology, Co-evolution, Cultivation, Selected Effect, Artifact, Engineering Function.

Synthetic biology promises the possibility of a philosophically interesting type of entity: a biological organism that appears to have no direct ancestry in similar organisms, but that is both wholly designed and constructed by humans. The possibility of such *de novo* organisms raises interesting questions for functional analysis and health and disease: without being the reproductive output of similar organisms, can *de novo* organisms have aetiological functions? What is the *proper* function of such organisms if their intended or designed functions conflict with what appear to be their biological goals: survival and reproduction? And how, if at all, can we determine in such cases whether the organism is healthy, or has a disease?

Section 1 gives a brief overview of approaches to functional analysis in biology and engineering, as well as their relation to health and disease judgments. I will not defend a particular account of function in this section. Both in the context of biology and engineering there is a broad consensus that a pluralist approach is to be preferred: multiple approaches to functional analysis can usefully co-exist and are more or less useful depending on the pragmatic context of purpose of the enquiry. In keeping with this pluralism I will outline the two main types of function account–*aetiological accounts* and *causal role accounts*–so that they can be applied to synthetic organisms in sections 3, 4, and 5.

My discussion of accounts of health and disease will only consider so-called *naturalist* approaches. These define disease at least in part in terms of biological dysfunction, and are therefore interesting to discuss alongside questions about functional analysis.

Section 2 briefly introduces what synthetic biology is and how it differs from earlier forms of biological engineering. As we will see, the type of organism that I will discuss is promised, but not yet realized, by current practices.

Section 3 examines whether *de novo* synthetic organisms can have aetiological functions. I first present the *prima facie* argument that they cannot; they lack a reproductive history on which selection could have acted. I then respond by giving independent arguments for two claims. First, selection processes that generate aetiological functions must include entirely human-imposed selective pressures. Two, reproduction does not require the direct transfer of information-bearing biological material between generations, but can include the (re)creation of organisms by human actions, based on information that is conserved as knowledge in the human (extended) mind. These arguments not only result in a new and decisive answer on whether synthetic organisms have aetiological functions, but they have upshots for functional analysis and wider debates in philosophy of biology: they (1) reveal a continuum between aetiological functions in engineering and biology, and (2) raise further questions about ancestry, heritability, biological individuality and the boundaries of life.

Section 4 considers synthetic organisms whose ‘designed’ function appears to undermine, rather than promote, their survival and reproduction. An example is an organism that models terminal disease processes. On the face of it, such organisms put pressure on causal role analyses of biological function. But building on the arguments in section 3, I contend that if we take an encompassing view of reproduction here, such organisms pose no problem for causal role accounts.

They do, however–as I argue in Section 5–challenge our intuitions about health and disease. This puts pressure on naturalist analyses of health and disease. I discuss three ways of responding to this pressure, and tentatively endorse one. Again sections 4 and 5 have upshots beyond synthetic biology: they reveal (1) that questions raised by synthetic biology should already be widespread, and (2) that our intuitions about health and disease in the non-human animal kingdom are far less straightforward than is generally assumed.

1. **Functions in Biology and Engineering**

This section introduces the two main types of functional analysis that are applied to both domains where function-talk is widespread: biology and engineering. These are *causal role analysis* and *aetiological analysis*.[[1]](#endnote-1)I do not take a stance on which of these accounts is to be preferred, in keeping with a pluralist approach that is widely accepted in both domains.

*Functions in Biology*

*Causal Role Accounts* (Cummins, 1975) are present- or forward-looking. They analyse functions as the causal contributions that traits make or are disposed to make to a capacity of a system. In order to specify *biological* function on a causal role analysis, the relevant capacities are defined as the goals of the organism: survival and reproduction.[[2]](#endnote-2) For example, the biological causal role functions of my kidneys are to filter my blood of waste and help regulate blood pressure, because those are the contributions they make to my survival and reproduction.

*Aetiological Accounts* are backwards-looking: they identify the function of a trait as the effect of that trait that explains the trait’s existence (Wright 1973). When specifying biological function, this explanatory role is usually understood by reference to evolution by natural selection. (Griffiths, 1993; Godfrey-Smith, 1993, 1994; Millikan 1984; 1989; 1993; Neander 1991a, 1991b; 1995; Wakefield 1992). Because the ability of my ancestor’s kidneys to filter blood and regulate blood pressure explains the existence of my kidneys, these are the biological aetiological function of my kidneys.[[3]](#endnote-3)

Much ink has been spilled over the relative merits of causal role and aetiological accounts, but the literature appears to have reached limited consensus on two issues. First, a pluralist stance is justified: both function attributions have their uses.[[4]](#endnote-4) Second, within that pluralist stance, aetiological accounts are thought to have two distinct advantages. One is that they better account for the *normativity* of functions; unlike causal role functions, which can only describe what a trait is actually capable of doing, aetiological accounts can describe what a trait *should* do, even when it is not capable of doing it. Thus only aetiological accounts can tell us that, and when, traits *malfunction*.[[5]](#endnote-5) A second advantage of aetiological accounts of function is that they alone accommodate the apparent *explanatory role* of functions.[[6]](#endnote-6)

*Functions in Engineering*

Besides biology, the other main domain where *function-talk* seems appropriate is engineering. Here, the same two types of analysis–causal role and aetiological–are found.

Causal role analyses of engineering are exactly like those in biology, except that the capacity or ‘goal’ in engineering is usually some human aim. Thus the causal role function of my bicycle chain is to transfer the power exerted by my legs into wheel-rotation, because that is its causal contribution to my goal of moving forward-by-bike. If I use that same chain to ward off an attacker, than its causal contribution is to inflict injury (or look threatening). Note that such ‘use’ functions can apply to anything: artifacts can be re-appropriated (as in the defensive use of a bicycle chain) but so can biological entities (as I could also try to scare the attacker with my pet rat). Preston (1998, 2013a) points out that when it comes to artifacts, such new ‘use-functions’ can come to completely trump original design, and redefine the artifact. A rather useless kitchen implement, for example, is now in permanent and very effective use as the window-ice-scraper in my car.

Aetiological accounts of function in engineering, as in biology, appeal to the effect of an artifact that explains its existence. Traditionally, this effect was deemed the effect that the designer intended. On this account, even the wings of early airplanes would have had the aetiological engineering function of facilitating flight, even if they were incapable of doing so; the designer’s intent explains why the airplane exists, why it has the designed features that it does, and – indeed – even *that* it is an ineffective airplane (rather than, say, an ineffective washing machine). However, more recent work in the philosophy of technology has deviated from exclusive reliance on designer’s intent, for not all designed engineering functions are intentional: engineering systems may have parts or features that make essential contributions to the overall working of the system, whose contribution the designer did not intend. This can happen by chance, or (more often) because in the creation of artifacts – as in the creation of organisms – there is a significant role for reproduction and selection. This line of work emphasizes that it is naïve to think that the creation of artifacts is a one-off, top-down, uni-directional, idea-to-artifact procedure. Instead, designing and reproducing artifacts is a *process*–and often a complicated one–that is not unlike evolution: artifacts, ideas for artifacts, and prototypes are created and reproduced with both intentional and non-intentional variations, on which both intentional and non-intentional human selection subsequently operates (Houkes & Vermaas, 2010; see also Holm, 2013a). [[7]](#endnote-7)

As in biology, then, a pluralist stance regarding engineering functions is customary. However, in engineering there is even more to be pluralist about. First, there are causal role functions and aetiological functions. Second, aetiological engineering functions can be either determined by the designer’s intent, or by the differential ‘success’ of an artifact’s prototypes or ‘ancestors’ in the context of a selectively reproductive and/or iterative design process. Third, both causal role functions and ‘selected’ aetiological functions can be intentional or non-intentional. (Preston, 1998; 2003; 2009; 2013a; Houkes & Vermaas, 2010; Vermaas & Houkes, 2003).

Functional analyses in biology and engineering therefore have clear parallels: both employ both causal role and aetiological functions. Nevertheless, the precise interpretation of both types of functional account is different. Thus, despite sharing a common origin,[[8]](#endnote-8) biological and engineering functions now tend to be analyzed separately, and are discussed in largely separate literatures.

*Health, Disease, and Dysfunction*

There is a close relationship between analyses of biological function and accounts of health and disease: so called ‘naturalists’ analyse ‘health’ and ‘disease’ (at least in part) in terms of biological function and dysfunction. Boorse (1975; 1977; 1997) does so by adopting a causal role account of biological function. Wakefield (1992, 1995) employs an aetiological account.

1. **Synthetic Biology**

Synthetic biology is described as the “engineering of biology” (European Commission 2005): the “design and construction of new biological parts, devices and systems” ([www.synthetic](http://www.synthetic)biology.org) “which display functions that do not exist in nature” (European Commission 2005). The supposed[[9]](#endnote-9) key difference between synthetic biology and earlier forms of biological engineering is that, where earlier forms merely re-combined or modified *existing* biological systems, synthetic biology aims at the design and creation of *entirely new* organisms or biological systems “from scratch” (AAAS 2010). Biological engineering, for example, involves the building of human DNA into yeast cells so that they produce human insulin, or the growing of a human ear out of a mouse. Synthetic hopes to create, say, a human-insulin-producing organism without the use or copying of existing cells or DNA.

Attempts to create synthetic organisms take two main forms. One focuses on *BioBricking* (see, e.g., Gibson et al 2010; biobricks.org).This is an attempt to build a ‘library’ of modular DNA elements–BioBricks–that can be combined into organisms. This allows for the ‘rational’ design and construction of organisms with particular features. For example, if one wishes to create a basic organism that produces human insulin and is fluorescent, one would identify all the ‘BioBricks’ required to form a ‘basic organism’, as well as the BioBricks coding for ‘fluorescent protein’ and ‘human insulin’. One would string these BioBricks together into a numerical DNA code, transform this code into *actual* DNA through a DNA-printing process, and then insert the DNA into an existing (micro-)organism. This would allow for the DNA’s transcription such that proteins or even (in the next generation) entire organisms ‘encoded’ by the synthetic DNA code are produced (e.g. Gibson et al, 2010).

The second form of synthetic biology avoids reliance on existing organisms altogether. It aims to design and print self-assembling proteins that self-assemble into simple organisms–so called ‘protocells’–without the need to employ the DNA-transcribing and protein-synthesizing ability of an existing living unit (see e.g. Hanzyck, 2011).

In this paper I focus on the questions that arise if both methods are fully successful. At present they are not; self-assembling protocells have not yet been produced, but there *has* been (limited) success using the BioBricking method (Gibson et al. 2010). Even in such cases, however, the details of the processes are not as ‘clean’ as described above. Unsurprisingly, given what we know about genetic interactions, epigenetics and the general lack of straight geno- to phenotype mapping, the practice of a modular library is less straightforward than it sounds. The products of modular design processes, for example, can give unpredicted and interactive results that have to be ‘debugged’ in subsequent rounds of ordinary selective reproduction: a process known as *kludging* (O’Malley, 2009, 2011).

Nonetheless, this paper considers the question that would arise in the–as-yet hypothetical–case of a self-assembling protocell. Call this a *de novo* organism. *De novo* organisms are philosophically interesting because, on the face of it, they are simultaneously (1) biological organisms, (2) wholly ‘human-created’ designed artifacts and (3) entities without any ancestry in other biological organisms.[[10]](#endnote-10) They therefore raise interesting questions for functional analysis, which we shall now consider.

1. **Synthetic Biology and Aetiological Functions**

*De novo* organisms appear to lack direct biological ancestors whose differential reproductive success explains the present generation of organisms. This has motivated several authors to argue that *de novo* organisms cannot have *biological aetiological functions* (BAF)*.* Insofar as these organisms do have aetiological functions they must be a species of *engineering aetiological functions* (EAF): for example, those derived from the intention of their human bio-engineers (see, e.g., Basl, 2012; Holm 2012, 2013a, 2013b, 2014; Douglas, Powell & Savulescu, 2013). Insofar as these organisms, qua biological organisms, have biological functions, these must be *biological causal role functions* (BCRF) (Holm, 2014).

On the face of it this argument seems compelling. And it implies that *de novo* organisms present a real-life example of that familiar counter-example to aetiological accounts of function: swamplion. Swamplion emerges, by a freak of nature, fully formed from the molecules in a swamp and is indistinguishable from other lions. She is meant to provoke the intuition that, surely, the function of her heart is still to pump her blood, as it is in all other lions, from which she cannot be distinguished. But since she lacks a reproductive history, aetiological accounts must deny this intuition. (Boorse, 1976; Davidson, 1987; Neander, 1996) The very same, it seems, should be said about *de novo* organisms: their possibility threatens aetiological accounts of biological function.

I shall now contend, however, that this is false. *De novo* organisms do have biological aetiological functions, albeit unusual ones. The argument rests on two key points. First, selection by humans is no different from–and indeed *is*–natural selection. Second, what I shall call ‘self-reproduction’, or the direct transfer of biological or information-bearing material between generations, is not necessary for inheritance, biological reproduction, or aetiological biological norms.

*The Aetiology of Synthetic Organisms.*

What are my grounds for thinking that *de novo* organisms, appearances notwithstanding, do have a selectively reproductive history? The answer is that nearly all artifacts do. As philosophers of technology have emphasised (e.g. Houkes & Vermaas, 2010), it is a mistake to think of engineered functions as a direct ‘idea-to-product’ relationship, or of artifacts springing directly from their designer’s intent. Design processes involve elaborate rounds of copying, selection, and reproduction, as well as the spread, cooption, and alteration of ideas between engineers. Cars are a good example: every car and every engine now draws upon the designs, mistakes, and successes of previous cars. Indeed, this is one reason why our earlier discussion of engineering functions included un-intended selected functions: in the process of trial and error, copying, and selection of different prototypes, the performance of non-intended functions can become part of the design.

What holds for artifacts in general certainly holds for synthetic organisms–even self-assembling protocells. Even if we were to ignore the very real and evident role of selective reproduction in the development of synthetic organisms (see the literature on ‘kludging’), *and* ignore the fact that in BioBricking the BioBricks are all still modified versions of existing natural systems with reproductive histories, *and* furthermore ignore the reliance on an existing organism to transcribe and synthethise DNA–so we imagine an instantly successful self-assembling proto-organism–then this organism still has a reproductive history.

Consider such a *de novo* organism called FAP: a simple fluorescent self-assembling protocell. Suppose that creating FAP was an (extremely unlikely) case of instant success. Being in possession of the (not yet existent) academic paper detailing the construction of a successful simple self-assembling protocell (SAP), the team of bio-engineers intending to make FAP simply followed the instructions but added some fluorescent molecules, and got lucky the first time around: FAP self-assembled and turned out to be viable. Even in this unlikely scenario, FAP has a reproductive history, in that FAP still is a modification of a previous proto-type–SAP–which itself will have had a design history full of trial and error, reproduction and selection. The same is likely to hold for our ability to create and isolate the fluorescent molecules that were selected; these, too, will have an experimental engineering history, and thus an aetiological function.

Like other artifacts, then, synthetic organisms–even self-assembling protocells–have a history that involves the selective reproduction of prototypes. We can therefore conclude that this results in the sort of intended and non-intended *engineering* aetiological functions that such a history confers. The crucial question, however, is whether such a selective history–which appears to be a highly unusual one compared to other biological organism–can result in *biological* aetiological functions. First, at least on the face of it, the selection involved, which is entirely imposed and mediated by humans, is a far cry from what we have in mind when we talk about ‘evolution by natural selection’. Second, the mode of reproduction involved is, again, decidedly unlike what we think of in talking about exemplary cases of biological reproduction.

*Question 1: Human-Driven Selection*

The first issue is quite easily addressed: do human mediation and the imposition of selection criteria and selective environments disqualify subsequently selected traits from having a biological function? The answer is no. We have a *very* long history of such mediation and involvement. Think of dogs, horses, dairy cows, or *any* organism involved in agriculture or animal husbandry. The traits of these organisms have been heavily influenced by their co-evolution with humans, which sometimes involves intentional, and often un-intentional, manipulation of selective pressures (see also Sperber 2007; Preston 2013b). Surely these traits have retained, adjusted and acquired functions that are still appropriately considered *biological* functions.

One might be tempted to resist this, however. Think of a Holstein dairy cow, an animal that is selectively bred to produce quantities of milk that far exceed the needs of its own offspring. We can quite easily, and indeed do, specify functional norms for a Holstein’s milk production in various circumstances. A Holstein that is only able to produce enough milk to nourish its own calf is not functioning as it should. Nonetheless, one might think that these norms merely specify the cow’s *engineering* functions. When it comes to its *biological* functions, by contrast, one might argue that a cow *should not* produce more milk than is needed to feed its own offspring. Indeed one may well think it obviousthat a Holstein, which (as a vet once put it to me) “is bred to the brink of physiological collapse”, no longer has a milk production that can be legitimately described as *biologically functional.* It certainly does not seem healthy. A similar response is likely to be triggered by, for example, the exceedingly rapid growth of breast tissue in chickens grown for human consumption, which impairs adult chickens’ ability to walk.

Whilst this response is revealing about our intuitions about health–which we shall revisit in section 5–it is unconvincing at the level of biological function. Holstein dairy cows are, in evolutionary terms (that is, in terms of outcompeting other types of cows and spreading their numbers) extraordinarily successful. They have become so successful by enlisting human protection and human assistance. But this ‘enlisting’ of other species is a recurrent occurrence in the natural world. Frequently such ‘enlistment’ involves developing features to attract or reward these ‘enlisted’ species, at some energetic cost. Thus the Holstein’s high milk yields and the big breasts of chickens are, from the point of evolutionary success, no different from a flower’s nectar, which attracts bees, or a tree’s tasty fruit, which attract birds, or indeed from the aphid’s production of a sweet discharge that attracts and feeds ants, which in turn protect aphid colonies. These are all features that at first sight are energetically costly to the organism, but that have a very precise biological function: attracting reproductive vectors and/or protectors in a way that maximizes fitness in the actual evolutionary context that the organisms faces.

The consequence of this line of thought is that we should no longer think of the biological function of a cow’s milk as (solely) the feeding of its own offspring, as in other mammals. Instead, the function of the cow’s milk is to attract the protection and reproductive assistance of another species: *Homo sapiens* (see also Sperber 2007). Similarly, the function of chickens’ hypertrophic breast tissue is not flight, but pleasing humans.

If one is still unconvinced, then this might be because one perceives the co-evolution of aphids and ants, or of flowers and bees, as somehow ‘natural’ and therefore resulting in biological function. Human interference, for example by selectively breeding cows, may in contrast be seen as artificial: it distorts evolution, rather than being part of it. However, such a response is confused. It presupposes an entirely anthropocentric narrative, placing humans above and beyond ‘nature’ or biology. For a contrast, consider the perspective of cows. If we asked them, they’d tell us that they have successfully domesticated *Homo sapiens*, modifying the species by exerting a selective pressure on them to overcome the natural lactose-intolerance in adults. This has made humans ever keener to reproduce, defend, and tend to cows. Indeed the Holstein will proudly tell you how she has ‘outcompeted’ other cows by being the best at harnessing *Homo sapiens* for her own ‘evolutionary ends’.

Perhaps the claim is more intuitive if we consider wheat. Surely in wheat it is now the biological function of the husk, which keeps seeds concentrated to facilitate harvesting rather than (as in other grasses) scattering them to the wind, to promote reproduction via its main reproductive vector: humans.

We have no reason to think that the human mediation and alteration of selective pressures stands in the way of those very same selective pressures producing traits in organisms that have aetiological biological functions. This means that the appeal to ‘natural selection’ in biological aetiological accounts must include co-evolution, including co-evolution with humans. Another way of explaining this point is to say that any attempt to draw a sharp line between human agriculture and other forms of coevolution is an attempt to separate ‘culture’ from biology. However, we are cultural entities as biological entities. The co-evolution of aphid-herding ants and aphids is just as ‘cultural’ as human domestication of livestock and agriculture. It does not follow that it is therefore ‘not-biological’. Although such co-evolution involves cultural, perhaps even intentional, behavior, this simply does not place it outside the biological world. Similarly, the fact that synthetic organisms are (re-)produced in the context of entirely human-made selective pressures should therefore not be a barrier to performing an aetiological analysis of their biological function.

*Question 2: Non-Self Reproduction*

What about the second issue, the apparent lack of *self-*reproduction in synthetic organisms? As much as aphids recruit ants, dairy cows recruit humans, and flowers recruit bees, aphids still reproduce themselves–they don’t have ants ‘building’ them from scratch. Cows still can reproduce themselves, even though often they do not.[[11]](#endnote-11) Whilst flowers essentially rely on bees as part of their reproductive cycle, there is still a direct transfer of ‘packets of information-bearing biological material’ from flower to flower, in the form of pollen. The bees don’t create this pollen themselves. Synthetic organisms, by contrast, reproduce in a much more unusual way. Their heritable material (i.e. the ‘information’ transferred through generations or, in more misleading terms, the ‘recipe’ for their construction) is not encoded in DNA but in academic papers, computer codes, and human minds. There is therefore no direct physical connection or direct transfer of physical information-bearing material between generations. This appears to be a significant difference between synthetic biology and ‘ordinary’ biological reproduction.

But synthetic organisms are not the only organisms whose reproduction is indirect, involving no direct transfer of biological material between generations. Consider, for example, hybrid flowers, plants and fruits. These–much like mules–are the result of the crossing of two species, and are sometimes themselves infertile. Such hybrid plants–again like mules–are often deliberately created by humans for their valued properties: strength and hardiness in mules, for example, or particularly desirable fruits and flowers in hybrid plants. Indeed, a whole range of familiar agricultural products are such hybrids, including grapefruits, seedless watermelons, and many flowers. Some such hybrids can reproduce; some are infertile. In either case, however, they are often not bred from parental organisms of the same kind, but created and recreated as the first generation hybrid offspring of parental lines. This guarantees predictable traits in the first hybrid generation, which in later generations of in-breeding cannot be guaranteed. Such parental lines are frequently in-bred, kept and cultivated entirely for the purpose of producing desirable hybrids.[[12]](#endnote-12)

The reason for considering these hybrids is that they, like synthetic organisms, do not reproduce in a way that involves the direct transfer of information-bearing biological material between the relevant generations of hybrids. Although there is direct transfer of such material between parental and offspring organisms, these organisms are not similar; it is not the hybrid *itself* that reproduces by direct transfer of biological information. Instead, the hybrid–say a seedless (and thus infertile) watermelon–reproduces by enticing humans to do something particular, namely to recreate seedless watermelons from two parental lines, neither of which are seedless watermelons themselves. Thus the *information* that encodes the ‘recipe’ for its creation is partly the human knowledge of how to combine parental lines to recreate the specific hybrid.

This has implications when we consider the *biological* aetiological functions of hybrid organisms. Consider what an aetiological account would say about the biological function of the hybrid watermelons’s trait ‘seedlesness’. What explains the existence of this trait is the delight that humans take in seedless watermelons, which prompts them to keep recreating them. But the trait ‘seedlessness’ is not possessed by any of the seedless watermelon’s direct genetic ancestors, its parental lines. These aren’t seedless. The trait ‘seedlessness’ is only possessed by previous generations of seedless watermelons that did not, and could not, themselves produce further generations. The way in which ‘ancestor’ seedless watermelons transmit the trait of seedlessness to ‘offspring’ generations, then, is via human knowledge of how to (re)create seedless watermelons.

This starts to look an awful lot like ‘non-self reproduction’ as we found in synthetic organisms: in both cases, desirable traits prompt humans to (re)create the organism; in both seedless watermelons and synthetic or *de novo* organisms, there is no direct transfer of biological material between generations of organisms of the same kind, although there may be use of or reliance on existing biological material and systems; and in both seedless watermelons and synthetic or *de novo* organisms, at least some of the heritable information responsible for certain traits is transmitted through human knowledge of how to (re)create these traits. The ‘recipe,’ so to speak, for creating them is partially maintained–as propositional knowledge–in the extended minds of cultivators. I do not deny that there are differences. Seedless watermelons are still the product of ‘ordinary’ reproduction between parental lines, whereas *de novo* organisms are not. But that wasn’t the main objection to *de novo* organisms having aetiological functions. The objections were, first, that such organisms lacked a reproductive history on which selection has acted. This, we have established to be false. The second objection notes that these organisms are recreated by humans rather than by their ancestor organisms, which involves ‘unusual’ modes of information transfer. But such ‘unusual’ transfer of information occurs in other places as well.

It is particularly helpful here to consider that aetiological functions are supposed to give rise to functional norms. It seems clear that a seedless watermelon *should* be seedless, just as a Holstein *should* produce lots of milk. However, that functional norm cannot derive from, or be explained by, the success of the two biological parents that combined their gametes to create the seedless watermelon. Instead, it has to be derived from the comparative ‘reproductive success’ of previous generations of seedless watermelons, which is their ability to create future generations of similar organisms by enticing humans to do so.

One possible response to this argument is to conclude the opposite, and say that the above argument just means that we should restrict our interpretation of biological aetiological functions in ways that exclude not only the traits of synthetic biological organisms, but also the seedlessness of watermelons, on the grounds *either* that they only are there to please human reproductive vectors, *or* that they are not possessed by any of the seedless watermelons’ direct ancestors. Seedlessness may have an aetiological function, on this view, but only an *engineering aetiological function*.

I remain unconvinced by this move. In response to the first point, we might imagine bees claiming that their selective breeding of nectar-producing flowers means that nectar does not have a biological function, but only an engineering function created by bees. We would not be convinced by that either. In response to the second point, examples of the ‘indirect’ reproduction of traits already exist outside of human agriculture, where we would not hesitate to concede that these traits have *biological aetiological functions*. Consider, for example, eusocial insects such as ants. Soldier ants who are successful out-reproduce genetically unrelated soldier ants who are less successful (namely, those from other colonies). They do so, not by self-reproducing, but by making their queen more likely to reproduce. This produces future generations of soldier ants that are genetically and phenotypically similar to the present one. The traits of these soldier ants are thus explained by–and derive their biological aetiological function from–the effects of traits of previous generations of ‘reproductively successful’ soldier ants. But these previous soldier ants were not the *direct* ancestors of present soldier ants in terms of the transfer of information-bearing reproductive material. Their *direct* ancestors–a lineage of queens and their mates–never possessed the phenotypic traits that make soldier ants soldier ants. However, no one would deny that soldier ants have very precise, normative biological functions on an aetiological account. For the same reason, we should not deny that seedless watermelons or synthetic organisms have such functions, even if the traits that have them were never possessed by any *direct* ancestors of theirs. It is enough that what we shall call ‘indirect’ ancestor generations improved their ‘reproductive fitness’, not by reproducing directly, but by improving the chance that others with dissimilar phenotypic traits–such as queen ants or humans–would recreate genetically and functionally similar generations of organisms that bear the relevant trait.

*Implications*

I have argued that, initial appearances not withstanding, *de novo* organisms *do* have reproductive history subject to the sort of selection that can result in their possessing aetiological functions. Moreover, I have argued that these aetiological functions are *biological* aetiological functions.[[13]](#endnote-13) The former claim rested on the general point that most complex artifacts have design histories involving the experimental and selective copying of prototypes with more or less variation, and the plausible claim that successful synthetic biology would certainly have these features (O’Malley, 2011). The second claim rested on two key points. First is the claim that selection pressures that are mediated or entirely created by humans can still qualify as *natural* selection, which can give rise to biological aetiological function. Second is the claim that the transfer of ‘information’ between generations need neither be direct, nor non-human mediated. Traits can be heritable in the sense relevant for biological aetiological function, even when the organisms bearing the trait are not produced by parents who also possess those traits. These claims are independently plausible, as borne out by our function-judgments in hybrid agriculture, animal husbandry and other forms of co-evolution, and eusocial insects. Here we find, first, that both human-governed selective breeding and other forms of co-evolution frequently result in (possibly energetically costly) traits with the biological aetiological function of attracting reproductive vectors and/or protectors. Second, we find that direct and even indirect human-mediated heritability results in legitimate biological aetiological functions of traits.

I now want to sketch two implications of my claims so far. The first is specific and relevant for our analysis of biological and engineering function. The second is a series of wider questions about delineating ancestors, biological individuals, and life.

*Implications for Functional Analysis in Biology and Engineering.*

It has been noted before that synthetic biology, instead of being radically new or different, simply lies at the extreme end of a spectrum of intensifying co-evolution or human influence on other species’ traits–a spectrum that includes human agriculture and the domestication of animals (e.g., Preston, 2013b; Lewens, 2013). My arguments add that what holds for organisms holds for their functions: engineering aetiological functions and biological aetiological functions are not radically different, but lie on a spectrum too.

One implication of this is that we should reject the idea that biological aetiological functions are only the product of a particular kind of selection, namely *natural* selection. Or rather, I have argued that if *natural* is interpreted narrowly–to mean ‘not human mediated’–then it is a nonsensical view of evolution, and moreover one that rules out far too many biological functions. But if the term is interpreted broadly–as it should be–to include human selection and even human-mediated reproduction, then it might as well be omitted. Rather than viewing synthetic organisms, then, as systems that have both biological and engineering aetiological functions, which may conflict, we should see them as a place in which a continuum between aetiological functions in biology and engineering function becomes apparent, and where any difference between them disappears. This suggests that the literatures on engineering and biological functions, which are now largely separate (Preston, 2013a), may have to return to the more unified approach from which they originated. After all, the very attraction of an aetiological account of function derives from its ability to provide both normativity and explanation, and these are produced by any copying mechanism that is subject to selection–whether in engineering, biology, or both domains at the same time.[[14]](#endnote-14)

What does that imply for artifacts that *aren’t* organisms? Are their functions biological too? This is not a question I shall answer here. However, it strikes me that one could approach it in at least two ways. One is to think that the adjective ‘biological’ simply indicates the *type of system* that we are dealing with: organisms, or their functioning and living parts (functioning and living because, of course, many artifacts are built of non-living biological material, such as wood). On this view, aetiological functions are simply aetiological functions, regardless of domain, intent, or the source of a selective pressure, but there is a subset of things–organisms–whose aetiological functions are picked out as *biological*. On this view, it is not the kind of selection (i.e. ‘natural selection’) that determines whether aetiological functions are biological, but the kind of system in which the relevant trait operates.[[15]](#endnote-15)

A second, and different, approach would make all aetiological engineering functions *biological*. It could claim that human-created non-living artifacts, such as guns or cars, are, like the nests of birds, something like the biological functions of the extended organism. Such a view may well end up capturing not only aetiological engineering functions, but also some so-called ‘use’ functions of artifacts, which once again are not restricted to humans. Think of birds dropping rocks to open ostrich eggs.[[16]](#endnote-16)

Whichever view one takes, the suggested new idea of an engineering function, or the difference between biological and engineering functions, is to pick out a rough subset of functions about which we can ask particular questions: the role of human intent, for example, or other sorts of anthropomorphic concerns in the context of engineering, and the role of a particular kind of familiar reproduction and selection in organisms. But these functions aren’t in any way fundamentally different; they just pick out rough domains or identify specific questions.

*Further Questions.*

My arguments pinpoint further questions and avenues of exploration that relate to wider debates in philosophy of biology. First, whom should we think of as the ancestors of an individual or a trait? In humans that appears clear: the parents. But in seedless watermelons and soldier ants we could distinguish different kinds of ancestors. Direct ancestors, such as parental plants and ants, create the present individual. Indirect ancestors, such as the previous generations of seedless watermelons and soldier ants, have traits that (also) play a role in explaining the existence of the present individual, but that are inherited indirectly.

These questions are more extreme, but not different in kind to the case of synthetic organisms. Especially when we consider BioBricking, synthetic organisms may be assembled from pieces that have a hugely diverse range of more or less direct reproductive lineages, as well as ancestry in the ‘previous generation’ of experimental organisms that caused the experimenters to attempt to recreate them.

A second, and closely related, question, concerns what the relevant organism or *evolutionary or biological individual* is in cases that display both direct and indirect inheritance. In the case of ants and other eusocial insects, there is certainly a lively debate about whether the individual ant, or the ant colony as a whole, is the organism (See e.g. Clarke, 2010, 2013). Seedless watermelons might prompt us to ask similar questions, and these questions will only get messier in the case of synthetic biology.

Answering such questions may be useful outside the specific cases of synthetic biology and eusocial insects. Take the example of a rare, highly engineered stem cell culture, or a lineage of knockout mice in a particular lab.[[17]](#endnote-17) In both cases the ‘parental lab’ may send (and sell) its ‘offspring’ all over the world. Suppose these offspring do not ever get to self-reproduce, but their use by medical researchers means that funding keeps pouring in to sustain the parental line, to generate further offspring. First, in this example as in synthetic organisms, reproduction and selection are not just entirely human-governed, but various traits of the offspring explain the continued recreation of similar offspring without any *direct* reproduction or transfer of material. The account we give of functions in synthetic biology and agriculture is thus likely to be the account we should give of functions here. Second, in this case, too, we may well start to wonder who the relevant (evolutionary) individuals or organisms are: the individual cells, or the (very spatially extended) lineage as a whole. Third, synthetic organisms may further complicate the debate about the boundaries of life. Think, for example, of viruses, which are traditionally not considered ‘alive’ because they cannot self-reproduce. Now consider a self-assembling protocell that needs to be recreated by humans (and successfully entices them to do so). Even if the protocell has many marks of life – indeed, it looks just like a bacterial cell – its dependence on humans may raise questions about its status as a living thing.

1. **Synthetic Biology and Causal Role Functions**

I have argued, contra Holm (2012, 2014) that aetiological functions do not pose a problem in synthetic organisms. Nonetheless, in the spirit of pluralism, we can still ask: what about causal role functions?

When it comes to causal role functions, we appear to come up against the following problem: consider a synthetic organism that is specifically designed either to self-destruct (to boost our immune system, for example) or to model disease processes such as cancer or infertility. In other words, think of the synthetic equivalent of the OncoMouse.[[18]](#endnote-18) Such organisms are intentionally designed and constructed to have *engineering* functions that appear not to be their *biological* functions according to causal role accounts; they do not contribute to, but rather detract from, survival and reproduction.

In these examples, engineering functions and biological causal role functions appear to conflict. An organism that fails to self-destruct or model the disease process is malfunctioning in terms of it engineering function, but this malfunction contributes positively to its capacity for survival and reproduction. Organisms that function as designed, however, appear to be malfunctioning in terms of their biological causal role function; their traits are *less* capable of contributing to survival and reproduction, at least when compared to the organism that fails to perform the engineering function.

One might not think this is a problem; surely I can use an organism as an artifact in ways such that its proper function as an artifact detracts from its biological function? I could use a cat as a ballistic missile, for example. However, the problem goes deeper than that, Synthetic organisms are not merely used as an artifact, but designed as artifacts–and in fact *wholly* designed as artifacts. Their engineering functions are their ‘proper’ functions. Thus, insofar as biological causal role functions successfully lay claim to describing proper biological functions, the conflict remains. Consider the synthetic immune cell that ‘allows’ itself to be infected and then self-destructs, taking the virus with it. How can such a cell be both functioning and malfunctioning when it does the very thing it is supposed to do, as a designed biological organism? This is at best counterintuitive, and at worst a straight internal contradiction.

Building on my arguments in the previous section, however, we can easily see that this problem, too, is only apparent. It is caused by taking too narrow a view of reproduction. For if we consider that it is *humans* who, in synthetic organisms, must do the reproducing and who, in the OncoMouse, decide who lives and who dies, then self-destruction and developing tumors *do* contribute to the inclusive fitness of OncoMice. Humans will only allow further reproduction of OncoMouse lineages that *do* successfully model cancer, or of immune-enhancing ‘warrior’ cells that *do* self-destruct. The same arguments that established the aetiological function of the Holstein dairy cow’s milk production to be ‘enlisting humans as reproductive vectors’ establish that excessive milk production causally contributes to the Holstein’s inclusive fitness by pleasing humans. Similarly, the warrior ant’s fighting, and possible self-sacrifice in the process, causally contribute to its inclusive fitness, just as the OncoMouse’s successful tumor modeling contributes to its inclusive fitness. As long as biological causal role functions focus on the capacity to promote inclusive fitness, rather than the capacity to directly reproduce, the apparent contradiction can be dissolved.

1. **Synthetic Biology, Health and Disease.**

The real intuitive problem here, as we have already established in the case of dairy cows and hyper-breasted chickens, occurs not at the level of functions, but in the clash between the verdicts of accounts of biological function and our intuitions about health and disease. No matter how convincingly I argue that a dairy cow’s barely physiologically sustainable milk-producing capacity, or a chicken’s gigantic breasts, are strictly speaking *biologically functional* on either account of biological function, they do not, intuitively, seem healthy. The problem is even more pronounced for the OncoMouse and its synthetic equivalents. These organisms are *supposed* to have a disease; having that disease, as per the above analyses, is their function. But if we follow naturalist accounts of health and disease, which define normal function as health, and dysfunction as a disease, then it is logically impossible for OncoMice to have ‘having a disease’ as their normal function. This means, *in extremis*, that the OncoMouse *does not have cancer* and does *not model disease.* In the final section of this paper, this is the contradiction on which I will focus. I will suggest three ways of avoiding it: (1) by rejecting the view that disease is a biological dysfunction; (2) by expounding the ways in which model organisms are *model* organisms; (3) by embracing the limits of causal role accounts of biological function.

*1. Rejecting that Disease is Dysfunction*

First, one might infer that we should simply *reject* a naturalist account of disease as dysfunction in favor of some normative or value-driven account of disease: diseases as bad conditions, for example (see e.g. Cooper 2002; Reznek 1987), or disease as a failure to flourish (Foot, 2001; Megone, 1998; 2000). This move adequately accounts for chickens and OncoMice, Regardless of whether their oversized breasts or tumors are strictly speaking biological functions, these simply don’t seem ‘good’ for the animal on some straightforward assumptions about the interests of chickens and mice–not being in pain, for example.[[19]](#endnote-19)

Whether or not this is the right response, the very fact that we have a possible argument in favor of normativism motivated by intuitions about *animal* health is interesting. Non-human disease judgments are usually considered to favor a naturalist analysis; we seem able to determine whether dogs, trees, fungi, ants and amoebe are healthy or ill. However, since all these organisms clearly have biological functions, but not all of them obviously have interests–and certainly lack the ability to form an evaluative judgment of their situation–the argument usually concludes that normativism must be false and naturalism must be true (e.g. Boorse, 1977). The present discussion undermines that argument. It demonstrates that health and disease judgments in animals may *not* be straightforward, and that there can be a clash between our intuitions regarding, or–in the case of the OncoMouse–explicit conceptualization of the animals’ health on the one hand, and its normal biological function on the other. This means that an argument against normativism on the grounds that animal health- and disease judgments are obviously unproblematic and non-value-laden has its work cut out for it.

That said, I am not convinced that the case of the OncoMouse should prompt us to abandon naturalism. If we did, shouldn’t we also say that the stinging bee suffers a disease because it would be better for it not to sacrifice itself for its queen? That the sheepdog is dysfunctional because it would be better for it to eat the tasty sheep or laze about doing what it feels like? Or that the spider that lets itself be eaten by its young, the praying mantis that feeds itself to its mate, and the male salmon that dies after discharging its sperm are all ill because (surely) there would be less miserable things for them to do?[[20]](#endnote-20) Animals make great sacrifices to promote their inclusive fitness. The idea that such actions are not healthy–that such behaviour is not in the cannibalistic spider’s interest–strikes me as deeply unconvincing.[[21]](#endnote-21)

*2. Making Much of Model Organisms*

A second response is to make more of models. This allows one to stick with naturalism, but to avoid the logical contradiction seemingly presented by OncoMice. The claim would be that OncoMice–and, say, a synthetic organism that modeled Huntington’s disease–do not in fact have a disease; all they do is *model* human disease processes. Like all models, they come with an implicit model specification that tells us how to translate its facts to the system that is being modeled (Frigg & Hartmann 2016). Thus, just as computers do not themselves suffer traffic congestion, but merely *model* it, the OncoMouse is not ill, but merely *models* illness.

Whilst this response is possible, I do not find it convincing. First, it does not help us in any way with dairy cows and hyper-breasted chickens. Second, OncoMice don’t just model disease processes in humans, they undergo processes that, should we encounter them in any other mouse (including the lab mouse in the cage next door that is meant to model *diabetes*), we would straightforwardly think *were* a disease–a mouse disease.

*3. Exploiting the Ambiguity in Function Ascriptions.*

The third solution claims that, in our judgments that dairy cows, chickens, and OncoMice are ill–but that self-sacrificing warrior ants are not–we exploit the pragmatic flexibility afforded to us by causal role accounts of function.[[22]](#endnote-22) I find this the most plausible approach.

Remember that aetiological accounts were supposed to have a distinct advantage over causal role accounts: they create naturalistic norms. They alone, by appeal to effects in ancestors, can tell us what a trait *is supposed to do*,and thus, when it does not do something, whether it is malfunctioning. Causal role accounts, by themselves, can do no such thing. In order to make causal role functions amenable to judgments about *normal function* and *dysfunction*, therefore, which is necessary for underpinning an account of health and disease, biological causal role functions must be modified. The most prominent attempt does so by defining normal functions as the causal contributions to survival and reproduction that are statistically typical in a reference class (Boorse 1977; 1997); dysfunction is an adverse departure from these. Thus ovulating roughly once a month is a considered normal function because it is statistically typical in the reference class of 15 to 45-year-old women; not ovulating is considered a dysfunction in this group. However, not ovulating is not statistically abnormal and therefore not defined as a dysfunction in 15 to 45-year-old men, or 5 to 10-year-old girls.

According to a body of recent literature, this simple picture does not determine all our function judgments. First, there is flexibility in reference classes: what are they, and more importantly why do they exist? Adopting different reference classes results in different disease judgments; depending on whether we accept, say, being deaf as a separate reference class or not, deaf people will count as either healthy or ill (Kingma 2007). Second, statistically typical function needs to be determined with respect to a particular set of environments or situations. For example, lactational secretion is normal function in women after they have given birth or when they have breastfed continuously since giving birth, but it is a dysfunction if it happens spontaneously in other circumstances. Importantly for our argument, however, not any environment or situation will do. The reduced ability of hemoglobin to bind oxygen may well be its statistically typical function in the situation of encountering a carbon-monoxide-rich environment, but we still consider it a disease (Kingma 2010, 2016). The reason for this may be that we import a set of values to distinguish healthy and unhealthy environments (Kingma 2010), or that we define normal function with reference to ‘benchmark’ environments (Hausman, 2012) that were evolutionarily plentiful.

I suggest that it is the scope of flexibility that these arguments provide– flexibility both in terms of fixing reference classes and in choosing which environments should, in Hausman’s (2012) terms, provide the benchmark for our normal function-judgments–that we exploit in our judgments of dairy cows, chickens, and OncoMice. Dairy cows and broiler chickens are evidently some kind of subgroup of chickens and cows. One way in which we can resist calling them healthy, even while recognizing that their ‘attractive features’ ultimately promote their inclusive fitness, is by refusing to assign them their own reference class. By comparing hyper-breasted chickens will all chickens, we can conclude that they lack the statistically normal function of being able to support their own weight and walk.[[23]](#endnote-23) Such flexibility is afforded by health and disease judgments, even within humans. We can say that someone with a chronic illness or disability has been ‘very well, lately’. That does not mean her disability or chronic illness has disappeared. Rather, we have temporarily shrunk our class of comparison to people with that disease only. [[24]](#endnote-24)

Alternatively, what may be going on in our judgments of chickens or dairy cows is that we determine normal function by reference to a set of benchmark environments that do *not* include the human milk, egg, and meat industries, but rather some actual or imagined ‘natural’ or historic environment for these animals. In such environments–where they aren’t assisted by humans, and have to find their own food and fend off predators–both hyper-breasted chickens and dairy cows would have statistically subnormal capacities for survival and reproduction.

There are ways, then, of making sense of our health and disease judgments–either in terms of normal biological functions and dysfunctions, or in other ways–that are compatible with the account of biological function of synthetic organisms presented in this paper. I have indicated that my sympathy lies with the third option, but that this need not be the only one. Whichever direction we take, thinking about our function and health judgments regarding organisms that are (either wholly or in part) ‘engineered’ by humans is clearly instructive. It shows that health and disease judgments in non-human animals are far from the straightforward exercise that we widely presuppose them to be.[[25]](#endnote-25)

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1. My taxonomy roughly coincides with those of Godfrey-Smith (1993; 1994), Lewens (2004), Neander (1991a), Schlosser (1993; who calls causal role approaches ‘organizational approaches’), Schwartz (2004), and Roszkowski (2010). But see Walsh & Ariew (1996) or Perlman (2004) for alternative and more detailed classifications. See Nanay (2010) for a quite different ‘modal’ account of function that I do not discuss here. [↑](#endnote-ref-1)
2. See e.g. Boorse 1977; Bigelow & Pargetter, 1987 for such accounts. There are subtle differences between them, but these are not relevant to the present argument. In the remainder of the paper I will replace ‘survival and reproduction’ with the more plausible ‘inclusive fitness’, in order to make sense of biological functions in, for example, warrior ants and non-dominant wolves (see section 4). Whether any of the aforementioned accounts are or should be committed to this move is beyond the scope of this paper. [↑](#endnote-ref-2)
3. There is more to say about how exactly selection and aetiological accounts are to be cashed out. We can, for example, contrast accounts that look at more distant evolutionary history in contrast to ‘modern’ history approaches (Godfrey-Smith, 1994). Incorporating these nuances is beyond the scope of this paper. [↑](#endnote-ref-3)
4. See e.g. Griffiths, 1993; Godfrey-Smith, 1993; Perlman, 2004; Amundsen & Lauder, 1994. [↑](#endnote-ref-4)
5. E.g. Neander (1995). Causal role accounts can also attempt to account for malfunction, but only by adding to the account; the most prominent example of this is Boorse’s (1977, 1997, 2002) account of disease/dysfunction as an adverse departure from ‘normal species function’, which is defined statistically. [↑](#endnote-ref-5)
6. E.g. Godfrey-Smith (1993, 1994), Griffiths (1993), Millikan (1989) and Neander (1991a). [↑](#endnote-ref-6)
7. I only summarise and reference the more recent discussion in philosophy of technology. The discussion of non-intended artifact function has a much longer history; see e.g. Boorse, 1976. [↑](#endnote-ref-7)
8. See e.g. the debate between Wright (1973) and Boorse (1976). [↑](#endnote-ref-8)
9. See, e.g. Endy 2005; Preston 2013b. [↑](#endnote-ref-9)
10. I focus on *de novo* organisms as the ‘purest’ examples of a synthethic organism. Contrast this with Lewens’ (2013) discussion of synthetic biology, which considers BioBricking the furthest removed from ordinary biology. This difference is unsurprising; Lewens is interested in claims about the rationality of the design processes, and, arguably, biobricking, due to envisaged greater levels of control and design precision, allows for more ‘rational’ or ‘planned’ design. Since I am mostly interested in claims about the absence of aetiology – of a selectively reproductive history in previous generations of organisms – self-assembling *de novo* organisms appear furthest removed from ordinary biology because they appear not to have any living ancestor. BioBricking, by contrast, requires that synthetic genomes are implanted and transcribed by existing monocellular organisms. [↑](#endnote-ref-10)
11. Cows are mostly artificially inseminated by humans. It is not inconceivable that cows and/or bulls would lose the ability to do the relevant deeds themselves, in which case they would become fully dependent on humans to act as reproductive vectors. [↑](#endnote-ref-11)
12. Hybrids can also occur naturally, especially in plants, with parent species crossing ‘spontaneously’ [↑](#endnote-ref-12)
13. It is this point on which I clearly deviate from both Holm (2013a, 2014) and Basl (2012), who also note that synthetic organisms may have either intended or non-intended aetiological engineering functions, but deny that these are biological functions because human-mediated selection, they claim, is not *natural* selection. [↑](#endnote-ref-13)
14. See also Lewens, 2004. [↑](#endnote-ref-14)
15. As Holm, 2013b, 2014 notes, this may result in ‘health’ talk extending into the artifactual domain. But such a worry is easily blocked; if we can restrict biological functions to a specific domain - the living world – then we can do the same with health-talk; only in living systems are dysfunctions called diseases. [↑](#endnote-ref-15)
16. On this view, Holm’s (2013b, 2014) worry (see previous footnote) is more pressing. [↑](#endnote-ref-16)
17. I am grateful to Paul Griffithsfor suggesting this line of thought. [↑](#endnote-ref-17)
18. I am indebted to Simon Ripponfor the example of the oncomouse and the problem it poses. Boorse (2011) and Holm (2014) also discuss this problem. [↑](#endnote-ref-18)
19. Note that spelling out such interests is not easy. It is often done by appeal to the animals’ teleology (see e.g. Basl & Sadler, 2013). But if this is defined in terms of their biological function then, given what I have argued earlier, we find ourselves in a vicious circle: giant chicken breasts *would* be in the chicken’s interest. See Lewens (2010) for a similar argument in response to Foot (2001). [↑](#endnote-ref-19)
20. Goossens (1980) and Reznek (1987) first mention examples such as the praying mantis as an objections to Boorse’s BST. Boorse (1997) responds. [↑](#endnote-ref-20)
21. One might think that our intuitions about health and disease somewhat run aground when considering the praying mantis. If this is the case, it might be a separate argument in favor of normativism of a different flavor: it is an anthropomorphic notion based on *our* interest that we can project about as far as higher mammals, but not much beyond. I cannot consider this argument here, but there are certainly responses to be made. Plants are quite different from mammals, nonetheless we do attribute health and diseases to plants [↑](#endnote-ref-21)
22. Whether aetiological accounts of function also provide some flexibility is not something I have space to consider. See my (2013) for an argument that *if* disease is a biological dysfunction, it is a causal role dysfunction. The present discussion may lend further support to that claim. [↑](#endnote-ref-22)
23. If hyper-breasted chickens and dairy cows are more numerous than other chickens and cows, this move may not be possible. See the long-standing discussion on epidemic disease (e.g. Boorse, 1997; Guerrero, 2010). [↑](#endnote-ref-23)
24. This argument addresses another worry raised by Holm (2013b, 2014): synthetic organisms can’t be accounted for on Boorse’s account of health and disease because they lack ‘species design’. But as my argument points out, it is not species design, but reference classes that are important here. [↑](#endnote-ref-24)
25. I am grateful to the SYBHEL project (<http://www.sybhel.org>) for their invitation to speak at a workshop–this prompted my interest in the topic. I am grateful to audiences at the 2012 SYBHEL workshop in the The Hague, the 2015 BSPS Conference in Manchester, and the 2015 CLMPS Conference in Helsinki, for useful questions and discussion. I’d like to thank Teresa Baron and Nicole-Standen Mills for comments on the written work. This paper was completed whilst on a project that has received funding from the European Research Council (ERC) under the European Union’s Horizon 2020 research and innovation programme (grant agreement No 679586).  [↑](#endnote-ref-25)