

A RELIC OF DESIGN: AGAINST PROPER FUNCTIONS IN BIOLOGY

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The notion of biological function is fraught with difficulties - intrinsically and irremediably so, we argue. The physiological practice of functional ascription originates from a time when organisms were thought to be designed and remained largely unchanged since. In a secularized worldview, this creates a paradox which accounts of functions as selected effect attempt to resolve. This attempt, we argue, misses its target in physiology and it brings problems of its own. Instead, we propose that a better solution to the conundrum of biological functions is to abandon the notion altogether, a prospect not only less daunting than it appears, but arguably the natural continuation of the naturalisation of biology.

INTRODUCTION

Biological functions pose a conundrum: they refer to something teleologically-loaded in a context that was freed of any purpose. The way we see it, the core philosophical problem is to articulate the highly successful practice of functional ascription in physiology (which appears to investigate organisms *as if* they had been engineered) with the theoretical framework of evolutionary biology (which is supposed to eliminate any residue of purpose-talking).

This problem should be contextualized and characterized in light of the widely-shared, tacit idea that the notion of function represents (or should represent) a sort of unification of physiology and evolutionary biology: physiology uses functions to explain *how* traits or variations contribute

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to organismal maintenance and/or capacities, which in turn may perhaps explain why some said traits/variations got selected through evolutionary history, which in turn explains *why* an organism's physiology is the way it is. The selected effects account of functions has been purported to provide this unification and naturalize the physiological notion of function. However, in this article we show that this strategy is fraught with difficulties and a mismatch remains between the two, leading to irremediable confusion. Moreover, we also show that selected effect *per se* is problematic. Therefore, function-talk in all its ramifications seems to be misplaced and misleading. In the light of these problems, we propose to eliminate the notion of function altogether.

The structure of the article is as follows. In Section 1, we describe the various meanings functions can have in biology, their relationship, and the problems created by ambiguities between them. Rather than being due to conceptual laxity, we argue throughout the paper that these difficulties are inherent to biological functions. In Section 2 we propose a reading of the history of functions in physiology according to which physiological practice has always treated organisms as if they were designed, even if that idea had been forsaken. Attempts to resolve this tension by appealing to selected effects remain problematic because of the mismatch between ascriptions. In section 3, we furthermore show that attempting to reform functional ascription to conform to selected effects might not be a desirable solution. In Section 4 we propose a solution to avoid the ambiguities of the problems with function-talk, which is to eliminate the word 'function' altogether, and we show that in the relevant fields losing the word 'function' is not very costly, given also the payoffs of avoiding the epistemic problems highlighted in sections 1-3.

1. THE PROBLEM(S) OF BIOLOGICAL FUNCTIONS

1.1 Four notions of biological function

Given the debate on the meaning of function, it is worth beginning by distinguishing different possible meanings, suspending for a moment judgment as to whether these should be called biological functions. Wouters (2003) identifies four main notions which, although not necessarily exhaustive, capture the most important and interesting connotations of function-talk in biology:

- 1 *Biological activity*, namely “what an organism, part, organ or substance by itself does or is capable of doing” (Wouters 2003, p 636). This notion is very broad and, if used

without care, can lead to spurious or problematic functional ascriptions, such that a function of the heart is to make noise.

- 2 *Biological role (BR)*, also called causal role, describes “how a certain item or activity contributes to the emergence of a complex capacity of an organism” (Wouters 2003, p 638). This notion of function is usually attributed to Cummins’ (1975) analysis in terms of causal roles. The difference between this notion and biological activity is that a functional analysis requires to establish first an explanandum or a context. Once we establish the higher capacity to be explained (e.g. flying, digesting a food product, etc.), the analysis is constrained, and ascribing functions is not arbitrary. As Craver has argued in the context of selecting which factors are relevant and they should appear in a mechanistic explanation, “[w]hich information is relevant varies from context to context, but that a given bit of information is relevant in a particular context is as objective a fact about the world as any other” (Craver 2006, p 360). Analogously, if the activity of the respiratory system is what we want to explain, then the fact that the heart emits noise will not count as a function, as it does not contribute in any way to respiration. Rather, the function of the heart in this system will be to pump blood, thus enabling gas exchange between the lungs and the rest of the body. The heart could have other roles in other higher-level capacities. In other words, a functional analysis situates an entity in the organization of a system. Nevertheless, even with specific contextualizations à la Craver, critics often see such functional ascriptions as subjective, because in principle one could define the explanandum arbitrarily. Evolution has been proposed to provide an objective framing (see the notion of biological advantage, below).
- 3 *Selected effect (SE)*, namely “the effect for which a certain trait was selected in the past” (Wouters 2003, p 649). This is a historical (or etiological) notion of function, meaning that functional analysis is not necessarily aimed at explaining what an item does, but rather at explaining why a certain trait is present in the current population, or in general the presence of the function-bearer (Wright 1973; Millikan 1989; Neander 1991). As such the explanation depends entirely on evolutionary history. More precisely, a trait T has a function F iff T 's performing F is the reason why T has been selected and maintained in the course of evolution. For instance, moth light in

color were predominant before the 19th century in England because they were concealed from predators on light-colored trees. However, as soot started to darken the trees during the industrial revolution, there was a selective pressure in favor of darker-colored moths, and hence the function of the dark color is, under this account, to make the moth less visible on darkened trees.

- 4 *Biological advantage*, which is “the biological value (utility) of a certain trait in comparison with another” (Wouters 2003, p 643). Here, *utility* is understood in light of what we know of evolution, e.g. as contributions to survival or extended fitness. While selected-effect is backward-looking, biological advantage is forward-looking (Bigelow and Pargetter 1987; Griffiths 2009) and therefore independent of history. For instance, in an environment where pollinators reach predominantly tall plants, being a tall plant is a biological advantage irrespective of whether it was selected for that reason.

The relationship between these notions should be relatively clear: all selected effects (SE) imply a biological advantage at least in ancestors (though not necessarily in present organisms), which in turn imply a BR and a biological activity (again at least in ancestors). The historical aspect, however, prevents the concepts from standing in a full nesting relationship, as represented in Figure 1.

While one can legitimately debate whether they deserve the label of function, it is fairly uncontroversial that the different notions all have their utility. There is a rich tradition of linking them to different research questions. Ernst Mayr (1961) famously distinguishes *How*-questions (in the sense ‘how does x operate’) from *Why*-questions in the context of biology, where the latter can further be divided into proximate and ultimate causes of biological phenomena. “It is evident”, Mayr writes, “that the functional biologist would be concerned with the analysis of the proximate causes, while the evolutionary biologist [...] with the analysis of the ultimate causes” (1961, p

1503).³ Wouters (2005) notices that this is not uncommon; biologists speak “of functional biology and evolutionary biology as two ‘modes’ and ‘ways of doing biology’” (p 130).⁴

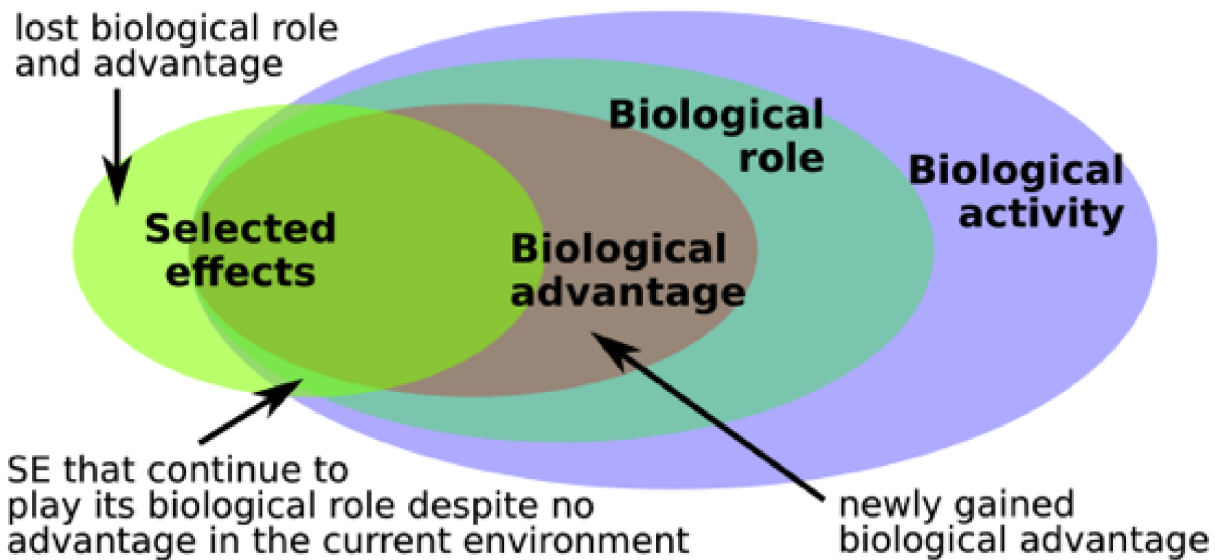


Figure 1. Relations between different notions of function

More recently, Marcel Weber (2005) has supported a similar idea, emphasizing the relative autonomy of some research questions. He follows Mayr in distinguishing between etiological and causal-role types of functional explanation, and he argues that in the former case biologists are interested in explaining the presence of the function, while in the latter case the explanation is geared towards the elucidation of the specific contribution of the function-bearer (the ‘how’) to a system. Next, he also stresses that the ascriptions are established independently of each other. Especially in the case of ‘causal role’, this ascription does not have any implication about why that particular token is present in the system. Conversely, “elucidating the evolutionary history of some

³The interpretation of the two questions is not uncontroversial, and it has for instance been pointed out that functional and evolutionary biology cannot be conducted entirely in isolation because the two constrain and feedback on each other (Laland et al. 2013). Mayr's point, however, was about the distinction between, and respective legitimacy of the questions, rather than about the isolation or independence of the endeavours to answer them.

⁴Similarly, in his famous analysis of traits in the context of ethology (i.e. behaviors), Niko Tinbergen (1963) distinguished four questions all to be answered in order to understand an organism: 1) causation (the mechanisms by which organisms do what they do), 2) ontogeny (the development of these mechanisms in an organism's lifetime), 3) survival value (i.e. biological advantage), and 4) evolution (the evolutionary history of the trait/behavior).

system or subsystem is supplementary to analyzing its function; it is not part of it” (2005, p 40). Therefore, while SE imply a biological role (at least in ancestors), it serves a different goal.

Others have resisted these arguments, in particular the claim that evolutionary components have no role in answering ‘how-questions’. Neander (2018) for instance argued that a teleonomic aspect has a role in generalizations: molecular biologists for instance aim at providing a description of how types or kinds of complex systems operate. Because this is shaped by multiple variables whose number vary across individuals, such description cannot be exhaustive, and “[t]he experimental work of biologists often focuses day to day on discovering the causal roles of a few features” (p 72). Those ‘few features’ form a ‘normal system’ which, Neander argues, is “one in which each part that was selected to do something” (p 72). We therefore take Neander to be arguing that that reference to the history of a trait is an integral part of a BR-based functional ascription (see also Millikan 1989). Similarly, Garson (2019) argues that to make sense of ‘dysfunction’ one would have to refer to a typical contribution, where this typical “is assessed over a chunk of time that stretches back into the past” (p 1152).

Griffiths (2009) provides an interesting take on similar issues. He recognizes, as Weber does, that BR are established independently from SE⁵, which might legitimate the use of different words for them. However, he thinks that BR are relevant from an evolutionary perspective, as soon as we realize that they can be proxies to establish evolutionary fate of function-bearers: biologists can use biological roles (BR) as heuristics to identify likely candidates for biological advantages, and vice versa. For instance, one may use cancer evolutionary genomics analyses to identify genes more likely to play a role in cancer (Domazet-Lošo and Tautz 2010; Ciccarelli 2010). In particular, we use an evolutionary analysis in order to identify those entities that are more likely to play a biological role in a set of cellular processes – i.e. genes appeared earlier in evolution are involved in basic cellular processes, which are more likely to be disrupted in cancer. These heuristics, however, do not impinge on the nature of what is actually characterized.

1.2 The problems of ambiguity

⁵ Griffiths (2009) distinguishes selected effects from what he calls a 'causal' notion of function which appears to sit somewhere between biological activity and advantage. His argument for the independence of the causal notion is simply that SEs are predicated on the existence of a BR-like functional ascription in ancestors.

One may take the variety of notions related to biological functions and the aforementioned considerations on their relative autonomy as implying a sort of pluralism. On this view, there are indeed different notions of function and which counts as the most relevant one depends on the context. Accordingly, the notion of function would not be problematic. The challenge, however, is to make sure that, when using the term ‘function’ with a given meaning in mind, one is not misunderstood as using a different meaning, or brought to make inferences that would be enabled by another. But ambiguity, we argue, is very difficult to avoid in function-talk.

Even if one can argue that there is a value in ambiguity⁶, this is not the case here. There is certainly a heuristic value in hypothesizing a SE from a biological advantage, or a biological advantage from a biological activity. Similarly, knowing that something has been selected does suggest its possible involvement in relevant biomedical phenomena we may seek to understand. Yet such heuristics are entirely within reach without using the same word in both contexts. We argue that using the same word for two meanings of function can lead to important problems, making the pluralistic approach unstable.

The first family of problems raised by an ambiguity on functions is well-known: the risk of *panadaptationism* (Gould and Lewontin 1979; Lloyd 2015). The problem of panadaptationism arises when a scientist is keen to analyze any trait in terms of ‘just-so stories’ of perfect adaptations. In the present context, panadaptationism can be understood as assuming that a biological activity or a BR is SE-function. Stated in this way, it is close to Godfrey-Smith’s (2001) characterization of ‘empirical adaptationism’. Recently, in the debate on ENCODE’s use of ‘function’ (see Germain et al 2014), the ENCODE consortium has been accused of claiming that any instance of BR-function is a SE-function, an accusation to which we will return in section 2.3.

⁶There are indeed examples of harmless ambiguity, and even utility of some conceptual vagueness in science. Francis Crick is reported to have “observed that if he and James Watson had worried about how to define the gene in the 1950s, progress in molecular biology would have stalled” (Robinson 2010). Similarly, Evelyn Fox Keller has argued that, while meaning is highly dependent on the context, “the use of language too closely tied to particular experimental practices would, by its very specificity, render communication across difference experimental contexts effectively impossible.” (Keller 2000, p140) In linguistics, Piantadosi and colleagues (2012) argued that ambiguous words can be more efficient when contextual information enables to solve the ambiguity. This assumes, however, that disambiguation is not prohibitively costly, which we argue is the case in biology. Moreover, far from being isolated, functional and evolutionary biology are often integrated, such that context does not resolve ambiguities.

Another - considerably less discussed - family of problems arising from the ambiguity comes from assuming that all and only SEs play a BR in phenomena of interest, which can lead to ignoring anything that does not have any compelling evidence of selection. We do not claim, here, that there is currently a lack of interest in BR beyond selection (indeed historically-agnostic biomedicine receives the lion's share of research funding), but that research strategies focused on conservation, which we see as the main alternative heuristic to ENCODE-like research programmes, have this important limitation. As it has been argued before (Germain et al. 2014), there are indeed various reasons to think that many selectively neutral traits or variations among humans might play biological roles that are relevant to biomedicine. Among the most important reasons are the weakness of natural selection in (especially contemporary) humans (due to a small effective size and an increased disconnect between intrinsic features and reproductive success), the discrepancy between our lifestyle and environment and those in which we have evolved, and finally our increased longevity and concern for age-associated diseases, which fall in the so-called 'selection shadow'. Furthermore, variations might have little or no effect in normal conditions, but become a cause for concern when accumulating with other alleles and in non-normal conditions (e.g. ageing, response to drugs, etc). As Niu and Jiang (2013) write, "[i]n organisms with small effective population sizes like humans and mice, the accumulation of neutral and slightly deleterious sequences is inevitable" (p 1341). Indeed, many disease-associated variants appear to be selectively neutral (Blekhman et al. 2008), and the vast majority of transcription factor binding sites appear to be under very weak selective constraint (Vierstra et al. 2020). If a so-called loss-of-function mutation results in a slightly reduced life expectancy or increased disease risk in old age that is selectively neutral⁷, the gene would not have been said to be functional in the first place by an SE account, but a physiologist would still speak of a loss-of-function, because the gene clearly is normally contributing to the wellbeing of the organism.

There are also cases of SE that would not count as functions for physiologists. A classical example in the philosophical literature is that of flat feet, which used to exempt men from military service (on the erroneous belief that it led to more injuries) and therefore plausibly had a selective advantage in the first half of the century. Assuming it did have such an advantage, the SE account

⁷The scenario is not at all artificial: for instance, the recent sequencing or genotyping of a large fraction of Iceland's population identified a throng of (sometimes homozygous) gene knockouts in individuals that appear entirely healthy (Sulem et al. 2015), but many could turn out to have an impact for instance in old age.

would ascribe a function to flat feet, whereas both physiology and common intuition would not.⁸ Similarly, “alleles underlying blue eyes and blond hair exhibit strong signals of selection, despite the fact that no intrinsic fitness advantage exists for these two traits” (Graur 2016, p 502). This is plausibly a result of sexual selection, and physiologists would have some reluctance in saying that dark-eyed people are dysfunctional. Or that anything in the native American genomes was unfit on the ground that their fitness plummeted after the arrival of Europeans.

1.3 Dissolving the ambiguity

As we have seen, in the life sciences the term function can have, and does have in scientific practice, multiple meanings. This ambiguity is not at all productive, but rather creates problems and potential for flawed inferences. This leaves us with a number of choices.

Relying on the logical primacy of BR, Germain et al. (2014) suggested that pluralism could be accounted for by using Cummins' analysis of function, and that functional ascriptions should only be made relative to a specific high-level capacity to be understood (meaning that there are no such things as 'proper functions'). This is not a perfect solution, most importantly because functions (whether in SE, or in vernacular language outside of biology) have the connotation of explaining the presence of the function-bearer (a point to which we will return in section 4), which is certainly not the case for BRs or Cummins' analysis. Removing such an entrenched connotation from a term is no easy task, thus leading us back to risks of panadaptationism.

The risk of panadaptationism, as well as the apparent arbitrariness of BRs, have led critics of ENCODE such as Dan Graur and Ford Doolittle to argue that all functional analysis should be framed in evolutionary terms – for instance by using a SE account of functions. They often cite Dobzhansky's famous dictum that “Nothing in biology makes sense except in the Light of evolution” as a rationale (e.g. Graur et al. 2013). Doolittle et al (2014) for instance write that “we do not know what fraction of CR-traits identified are SE functional (...) [i]t seems unnecessarily misleading to assume that CR methods alone can establish ‘function’ in a meaningful sense of the word” (p 1236), where ‘meaningfulness’ is a synonym of ‘evolutionary meaningfulness’⁹.

⁸ To clarify, we are not using intuition here as an argument towards one account of function or another, but simply to point out that intuitions conflict with accounts of function, and this conflict constitutes one of our practical arguments.

⁹ In other instances the position is not that neat as to imply that only SE counts as function, but rather that inferring “a contribution to fitness (and thus selection) requires an additional and difficult-to-prove inference” (Linguist et al 2020, p 1).

Presented with counter-intuitive examples, proponents of the SE account typically bite the bullet, arguing that in biology, function is simply that. The implication, however, is that the practice of functional ascription in physiology is erroneous, which is not trivial given that, as acknowledged even by proponents of the SE account, “[t]he main home of the teleological notion of function is not evolutionary biology; it is physiological biology.” (Neander 2006, p 592) In section 3, we will discuss further problems with the SE account.

There is, however, a third avenue to solving the function conundrum: *dropping function-talk from biology altogether*. To be clear, our position is not against Wouters’ four notions (on which there is little ambiguity), but against calling any of them function, and against the use of purpose-related language in biology. Such language is associated, both within and outside of biology, with conflicting and deeply-entrenched connotations which make a reform of its use hopeless. Yet our issue with function is not merely linguistic either, for as we have argued it can have detrimental effects on scientific practice and, as we will show, lead to a distorted picture of evolution.

In the next sections, we bolster our case against biological functions by arguing that the notion is inextricably linked to design. This is obviously true of vernacular language, but also, as we argue in section 2, throughout the history of the discipline. As we argue in section 3, even the SE account of functions retains features of an analogy with design which leads to a distorted view of organisms and their evolution. Finally, in section 4 we show how neither evolutionary nor functional biology would actually suffer from abandoning the language of function - quite the contrary.

2. PHYSIOLOGICAL FUNCTIONS AND THE NATURALISATION OF TELEOLOGY

We begin with a very brief genealogy of the notion of biological function, arguing that it historically comes from design and that despite the theoretical transformations led by Darwin, the practice of functional ascription remained largely unchanged. We suggest that in the context of physiology, where it is born, the practice of functional ascription is best understood as investigating organisms *as if they had been designed* (the design analogy). This creates a paradox, namely that we refer to something teleologically-loaded in a context that was freed of purpose, and SE represents but one post-hoc attempt at resolving this paradox and unifying physiology's successful practice of functional ascription with evolution. However, as we have already seen, SE

cannot legitimize physiology because their functional ascriptions differ (Amundsen and Lauder 1994).

2.1 Functional ascription originates from a belief in design and persisted ‘as if’

The adaptedness of biological organisms to their environment gives the impression of design, an idea that was at the core of biology until Darwin (and in which Darwin himself was involved before the *Origin of Species*), and which has led to considerable advances in biology by asking why organisms ‘were designed the way they were’, and ‘what purpose’ a trait had. Mayr (1992) for instance notes that “When Harvey was asked what had induced him to think of the circulation of blood, he answered, I wondered why there were valves in the veins” (Mayr 1992, p 132). With its appeal to Natural selection, the *Origin* is often viewed either as the demise of this natural theology, or as its naturalised justification; in fact Darwin can be said to have reinvented teleology (Lennox 2010). Sloan (1985) notes that the evolution of Darwin’s thought from earliest notebooks on transformism (1837-1839) up to the *Origin*, seems “to place heavy reliance on the concept of nature as a selective and teleological agency endowed with wisdom and foresight” (p 133). This teleological language of final causes was however in direct tension with the introduction of Malthusian features into his thought, which emphasizes issues of survival rather than harmonious teleology. Sloan concludes by saying that

“we surely see acknowledgement of some kind of purposive character of organisms. At the same time, (...) [t]he fitting of form to function, rather than necessary evidence of intelligence planning, is more a case of the parallel tracking of one form by the other to an end that seems no higher than survival” (p 138)

What is striking, despite this major conceptual transformation, is the continuity of physiological practice: physiology continued its work largely unimpressed by the theoretical squabbles over teleology. Long after natural selection had become a core component of the standard view in the life sciences, physiologists continued understanding organisms as if they were designed. In his recognized *Outline of General Physiology* (Heilbrunn 1937), L.V. Heilbrunn compares the organism to a “living machine”; today, in the widely-used Textbook of Medical Physiology (Guyton and Hall 2000), the “purpose” of various reflexes is explained, mechanisms are described as acting “for the purpose of controlling hydrogen ion concentration in the body

fluids” (p 48), and a vessel segment is said to “functions as a separate automatic pump” (p 173). Similarly, Dan Nicholson notes that

“Machine analogies and metaphorical references to ‘locks’, ‘keys’, ‘gates’, ‘pumps’, ‘motors’, and ‘engines’ continue to pervade the technical literature (e.g. Piccolino, 2000; Frank, 2011), as does talk of the ‘machinery’ (e.g. Goodsell, 2009) and ‘circuitry’ (e.g. Alon, 2007) that underlies the cellular organization.” (Nicholson 2019, p 109)

Functional ascription in physiology has usually been tied to a roughly reductionist enterprise that is much in line with Cummins' analysis of functions, and its history could be told as a series of descending attempts at explaining some higher capacity through appeal to the action of some parts or ‘elementary phenomena’ (Duchesneau 2010), and which then turn out to be less elementary than expected. It was for instance thought that the function of organs could themselves be explained by fundamental properties of tissues (e.g. Bichat’s sensibility and contractibility) which would reduce functional talk to mere physical mechanisms. These were later replaced by the features and allegedly irreducible metabolic processes of cells (from Schwann to Pasteur), which themselves were later to be explained through macromolecular functions (Morange 2010). The early work of Lavoisier (1780), Magendie (1816) and Bernard (1853), for instance, on the importance of elementary compounds such as oxygen and nitrogen in biological phenomena such as respiration, inspired a program of chemical physiology attempting to reduce physiological processes to chemistry, especially visible in the German school (e.g. Justus von Liebig, Carl Ludwig, etc. – see in Holmes 1963) and displaying clear advances in the context of nutrition and digestion (e.g. Carl Voit’s work). Although the largely European tradition of ‘general physiology’ more or less died in the early 20th century to be later reborn in molecular biology with more detachment from medicine (Kohler 1982), in retrospect foundational works such as the Krebs cycle, Garrod’s “inborn errors of metabolism”, Linus Pauling’s molecular medicine and Beadle and Tatum’s “one gene – one enzyme” hypothesis all arguably stand in close continuity with the 19th century tradition of chemical physiology.¹⁰

10 Indeed it is not trivial that Beadle and Tatum’s famous work on nutritional deficiencies. As Morange (2021) suggested, the concept of *specificity* (exemplified by Fisher's "lock and key" metaphor) can be seen as a historical thread from the late 19th century, linking biochemistry to physiology as well as genetics (Olby 1974). The transition from physiological chemistry to macromolecular chemistry, especially following Hermann Staudinger's work (see Olby 1974),

The language of function permeated this tradition, without necessarily being tied either to a theological worldview nor to natural selection. Indeed, teleological thinking before Darwin was not all theological (nor even vitalistic): as Timothy Lenoir (2011) has shown, the very important school of German teleomechanists simply felt compelled, as good empiricists, to follow Kant into accepting organic purposiveness (Naturzwecke) as “an objective fact of experience” (Lenoir p 25), but did not postulate an organizing agent, instead remaining largely agnostic as to the basis of this organization. Some more epistemic threads of the school argued for the necessity of a teleological framework on practical grounds, due to the extreme (irreducible, some would say) complexity of biological causation. They did not deny that chemistry was driving biological processes, but argued that this approach “will probably require much more time, and completely new methods of research will have to be invented before anything satisfactory can be achieved in this area.” (Bergmann and Leuckart 1852, cited in Lenoir p 176) This general view was long-lasting, and as Lenoir writes “biologists have learned to live with a kind of schizophrenic language” (ix): “In effect they seemed to be saying that physiology cannot operate in actual practice without the postulation of an intelligent universe, but that all mention of this postulate must be carefully excluded from the explanatory framework itself.” (p 236). Even Helmholtz, a fierce opponent of the teleomechanist programme, accepted “[t]he wonderful - and, through the growth of science, the more and more evident - purposiveness in the structure and function of living beings” (Helmholtz 1896, cited in Lenoir p.237), but explained it in Darwinian terms. Natural selection was not the reason for teleological concepts in biology, but rather the *post-hoc rationalization of existing (and successful) practices*.

2.2 Disconnect between functions and fitness

Although natural selection offered an abstract justification for teleology, in practice function worked largely in a disconnect from organism fitness. Duchesneau for instance (2010) notes that, in Theodor Schwann’s cell theory, the activities of cells were to be entirely emancipated from any idea of causal significance tied to their organism-level integration. This view was by-and-large realized, and indeed a good argument can be made as to the value of a relative isolation of (pairs

offered a concrete vessel for this specificity (and prime function bearers), eventually leading to the enzymatic theory of life.

of) organizational layers in understanding biological phenomena (see for instance Morange's 1997 analysis of cell biology as intermediate phenomena). According to Morange (2010), it is in the 1930's that functions traditionally attributed to organs or cells began to be transferred to macromolecules, a program unified with genetics through the one-gene-one-enzyme hypothesis of Beadle and Tatum (1941), which 'justified the idea that any gene (and its product) bears an elementary function'. If in the original work in *Neurospora* the link between molecular function and organism fitness was relatively straightforward, it became increasingly abstract and vague as the program was extended to more complex pathways and organisms. A further step, developing especially in the decades after the 1970's, was to understand protein function through the identification of functional domains, the genetic counterpart of which was to become ENCODE's 'functional element'. As with Schwann's program, it is important to note that these 'elementary functions' are only remotely connected to their organismic meaning: that a given protein can catalyze a biochemical reaction does suggest that this is important for some broader capacity and ultimately for the survival and reproduction of the organism, however this link is not needed for biology to characterize and study this activity. A whole biology was to be grounded in the characterization of these activities, opening the door for instance to a broad scanning of various predicted proteins for so-called 'functional domains'. Indeed, most of the 'molecular functions' assigned to gene products in the Gene Ontology are based on mere similarity to proteins with known functions, and while this is fairly predictive (e.g. of binding domains, catalytic activities, etc), it could hardly be more remote from functions as selected effects. Of course, the interest in these activities is not random: biologists investigate enzymatic functions because they expect them to be relevant to the organism, but this link was long not at the core of the research programme. It would therefore be erroneous to think that ENCODE and the likes departed in an unorthodox fashion from an established biological tradition unified around natural selection: it is instead the legacy of a different, considerably older tradition, aimed at understanding biological complexity through a decomposition into parts whose properties and actions contribute to higher level phenomena – in other words, Cummins' functional analysis.

2.3 Revisiting the ENCODE controversy

The ENCODE project, along with a series of similar projects (e.g. Roadmap epigenomics, FANTOM, Blueprint epigenomics, etc.), are best seen as representing recent steps in this tradition. It is not our aim here to discuss in any detail the ENCODE project or the controversy attached to

it (see Germain et al. 2014; Eddy 2012, 2013; Doolittle 2013; Graur et al 2013; Niu and Jiang 2013; Pennisi 2012; Birney 2012; Brunet and Doolittle 2014; Doolittle and Brunet 2017; Doolittle et al 2014). However, since it has become an important locus of discussion about functions in biology, we believe it is important, especially in light of the present discussion, to address an important misconception regarding it.

To recall the context, the ENCODE (Encyclopedia of DNA Elements) project was launched in 2003 with the objective of characterizing the regulatory elements of the human genome (dubbed ‘functional elements’), as well as stimulating the development and standardization of genomics technology related to this task. Genes are taken as developmental resources to be expressed depending on the cellular context, and this differential regulation is mediated by (non-coding) regulatory elements in the genome (such as, for instance, the binding sites of transcription factors), whose identification and characterization is therefore paramount to biological understanding. However, these are much less straightforward to identify from the genome sequence than protein-coding regions, and until their mechanisms of action are experimentally validated, one can only rely on a variety of signatures for their identification. One such signature is sequence conservation: if a given region of the genome is important to organismic functioning, then mutations to this region that disrupt this contribution should be deleterious to the organisms and be selected against over time, and as a result, the region’s sequence should be constrained across evolution. In contrast, a region which plays no relevant role in the organism should, with time, accumulate mutations. We therefore see a physiological endeavour, namely the mapping of regions that modulate gene expression, integrating knowledge of evolutionary biology to narrow down the most important regions (estimates of conservation in the human genome today vary within a 3-15% range, with most around 8-9% - see Graur 2016, p. 503).

However, as we have seen in section 1 and argued before (Germain et al. 2014), there are a number of reasons to believe that, especially in our species, non-conserved regions can influence gene expression in a way that matters to biomedicine. Moreover, a major limitation of the conservation signature is that while it offers extremely good indications that a region is relevant to the organism, it offers no clue as to how it might be. ENCODE therefore sought an alternative approach, building on the observation that the chromatin of regulatory elements tends to be

associated with certain sets of biochemical characteristics.¹¹ The consortium therefore reasoned that genome-wide profiling of an array of such characteristics would yield not only a list of putative regulatory elements, but their classification into various types, leading to more specific hypotheses as to how they can be expected to impact on the regulation of the genome - the biochemical signature strategy (Stamatoyannopoulos 2012).

Reporting in 2012 the application of this strategy to 147 cell types (to various degrees), the consortium noted that 80.4% of the genome was found to be covered with at least one potential functional element. ENCODE was hailed in mass media with a recurrent theme: that although it was long thought that most of the genome is junk DNA (i.e. DNA not benefiting its bearer), thanks to ENCODE we now know that most of it is functional.¹² Criticisms of ENCODE's claims by leading biologists in several academic articles shortly followed (Eddy 2012, 2013; Doolittle 2013; Graur et al 2013; Niu and Jiang 2013). The consortium was accused, among other things¹³, of using the wrong notion of function. i.e. something between BR and biological activity¹⁴, instead of SE. Another criticism, coming in particular from Doolittle (Doolittle 2013) was that ENCODE have wrongly conflated SE and BR, whereby the 'mere existence' "of a structure or the occurrence of a process or detectable interaction, is taken as adequate evidence for its being under selection" (pp 5296-5297).

An important point to understand is that, as a physiological endeavor of the tradition just described, ENCODE was not about junk DNA - an essentially evolutionary concept. Its findings hardly have any bearing at all on the question of whether most of DNA is junk (Germain et al.

11For example, regions bound by transcription factors show a distinct accessibility profiles, and active enhancers show a distinct set of histone modifications (H3K4me1 and H3K27ac), while active transcription start sites exhibit other histone modifications (H3K4me3).

12For instance, USA Today says that ENCODE "have junked the notion of junk DNA", and the Guardian, focusing also on the concept of junk DNA, adds that "this concept will now, with ENCODE's work, be consigned to the history books".

13For instance, it was accused of inflating the proportion of the genome deemed functional in various technical means (e.g. counting everything transcribed, even though the vast majority is spliced out and to our knowledge has no impact whatsoever), and of overselling the results of ENCODE to the public.

14Graur et al. (2013), despite quoting Cummins (1975), appear to misunderstand his notion and conflate biological activity and role (see Germain et al. 2014). ENCODE did however employ a notion of function which is halfway between biological activity and role: while not completely unconstrained as the biological activity, it is not either strictly related to specific capacities to be analyzed. ENCODE looked at a set of biochemical activities that are (more or less strongly) associated with gene regulation, leaving the burden to researchers to establish biological roles on a case-by-case basis.

2014), and in fact the expression does not appear even a single time in the publication. It is really unfortunate that ENCODE's PIs did not try right away to carefully shape the press-release of their publications in order to avoid misunderstandings around the scope of the project itself, and arguably even contributed to the misconstrual of the project as 're-writing evolutionary biology textbooks'. What emerged from other ENCODE publications (for instance Kellis et al 2014) aimed at de-escalating the controversy is that 'biochemical activities' are merely proxies, and that there is no necessary connection to functional ascriptions understood in an evolutionary sense. In other words, ENCODE ought to be interpreted as a means to understanding the inner workings of an organism, i.e. functional decomposition à la Cummins, and while conservation is an important good guide to picking out important parts, it is not equivalent to it. We want to stress that this succinct reinterpretation of the ENCODE controversy is by no means a defence of ENCODE itself, let alone of its usage of functions. Our aim is merely to point out that the entire controversy is based on flawed communication and a misunderstanding of the scope of the project.

Anticipating these observations when reporting the results of the pilot phase in 2007, the consortium had indeed noted the discrepancy between regions identified through conservation or through the biochemical signature strategy, in particular the "apparent excess" of the latter over the former, offering a clear hypothesis: "We believe there is a considerable proportion of neutral biochemically active elements that do not confer a selective advantage or disadvantage to the organism." (ENCODE Consortium 2007, p 812) Such a statement makes it clear that ENCODE is not conflating BR or activities with selected effects, but rather acknowledging the limits of both conservation analysis and natural selection. As with physiological functions, ENCODE's idea of function is not a proxy for selected effects, but simply the product of an activity of functional decomposition.

2.4 Functions as design analogy

As we have seen in section 1, the existence of two traditions with distinct practical usages of the term 'function' poses a problem of ambiguity which needs to be addressed, and somewhat of a challenge to the unity of biology. A firm evolutionary framing, declaring SEs to be the only *bona fide* functions, provides the illusion of a unification but does not solve discrepancies between the traditions unless physiologists agreed to reform their practices. In the next section, we will see why, even barring the pragmatic difficulties, SEs might bring trouble of their own. But we first

want to suggest a reading of functional ascription in biology which is rooted to its history, remains descriptively accurate, and is arguably more honest.

A direct appeal to natural selection having of itself ascribed functions, so to speak, is not the only approach to naturalizing its inherent teleology. Ernst Mayr, for instance, attempted to do so indirectly through an appeal to the goal-directedness of programs (see Mayr 1974, and Nagel 1977 for a criticism). Born in an era marked by cybernetics, Mayr's notion of program, although difficult to reconcile with contemporary developmental biology, was explicitly continuous with man-made machines, the chief example being the goal-directedness of servomechanisms. We suggest that the analogy to design, if properly grounded, can also be understood as constituting such an indirect approach (if somewhat deflationary) to biological functions. Michael Ruse for instance suggested that function talk is appropriate in biology because natural selection produces design-like objects:

”At the heart of modern evolutionary biology is the metaphor of design, and for this reason function-talk is appropriate. [...] Natural selection produces artifact-like features, not by chance but because if they were not artifact-like they would not work and serve their possessors' needs.” (Ruse 2004, p 273)

Under this view, one is justified to say that it is the function of the heart to circulate blood not because the heart was selected to this effect (which, although very likely, would be rather difficult to show), but because organisms tend, thanks to natural selection, to have parts whose action appear directed at contributing to their survival and reproduction, and the heart's circulating blood clearly does this. Others have followed a similar approach (see for instance Matthen 1997), in particular Tim Lewens (2004) who analyzed in depth the relationship between organisms and designed artifacts.

To say that biological functions are metaphorical, or ascribed in analogy to artifact functions, is not to dismiss such ascriptions. Indeed, the heuristic value of design metaphors has been highlighted for instance by Griffiths (1996), who distinguishes two ways in which “artifact thinking” helps biological investigation: 1) *reverse-engineering*, which seeks to use observed traits to infer environmental pressures (and developmental constraints), and 2) *adaptive thinking*, which instead tries to predict traits on the basis of environmental problems. Both have led to important discoveries, and are problematic only when they are taken as valid inferences, instead of merely useful heuristics. By emphasizing that functions are used in analogy with designed artefacts, we

are also emphasizing that it is a simplified, perhaps even distorted and dangerous, and yet potentially useful way of talking about biology -- so long as one recognizes it as such.

In response to Lewens' metaphor account, Neander (2006) argued that if biological functions are indeed a metaphor to design, then "it is a dead one, for function talk in biology has an independent life of its own" (Neander 2006). We instead believe the design analogy to be descriptively accurate, with alternative approaches to simply attempt to "cover up" or rationalize in a post-hoc and inadequate fashion a practice based on analogy. Indeed, as we will argue in a moment, the design metaphor even permeates the SE account of functions, leading to problems that are closely tied to the disanalogies between organisms and artifacts.

To sum up, we have seen that functional ascription in biology originally comes from physiology, and that it persisted, and very successfully so, as if organisms had been designed, independently of the demise of such an idea. This, we suggested, is because the heuristic of considering organisms as analogous to artifacts is useful most of the time. On the other hand, we believe that the genealogy of functions helps understand why the concept is so problematic today. ENCODE, we argued, is best understood as within this tradition, discussing functionality in a way that remains agnostic about the evolution of the function bearer. While this discrepancy between SEs and physiological functions became more prominent in genomics, where the distance to the phenotype leads to a reduced overlap between the two ascriptions, the discrepancy has been there all along the history of physiology. Rather than trying to dissolve it by pretending SEs to ground physiological ascriptions, we might as well acknowledge the whole idea of biological teleology to be a metaphor, i.e. a (perhaps evolutionarily warranted) heuristic, a powerful explanatory simplification, but not an ontological feature of biology. To be clear, we do not mean that ENCODE's usage of functions was right or wrong: it was a fruitful heuristic, which however becomes dangerous when we forget that it is just that. There is nothing wrong with metaphors - they are productive tools - so long as one does not mistake them for something real.

3. THE DANGERS OF SELECTED EFFECTS

Given the problems related to ambiguity with respect to biological functions (section 1) and the observation that functional ascriptions originate from a physiological practice impregnated from ideas of design (section 2), a natural conclusion is to want to reform functional ascription. Because they are both firmly grounded in evolutionary theory and are likely to overlap with a considerable

proportion of physiological functions, SEs appear as a natural candidate for a new, reformed understanding of biological function. We have already hinted at some problems of this approach, such as the fact that its functional ascriptions depart (both by inclusions and exclusions) from physiological ascriptions, to which SE proponents could answer by saying that physiology was wrong and needs to move it, or use different words. In this section, we want to go deeper into the SE account and show why, beyond the arguments already raised, a SE monism might not be such a desirable thing. Not only SE functions fall short of what they promise, but furthermore these shortcomings are mostly because, we argue, the account remains within an unacknowledged analogy to design.

The advantages typically touted of the SE account is that it is 1) objective (in the sense of independent of our question/interest), 2) explanatory (it explains the presence of the trait, just like we take it to do in designed artifacts), 3) normative (giving a more or less clear notion of dysfunction) and 4) non-accidental (thereby fulfilling some idea of teleology). We believe many of these to be in fact problematic, but want to concentrate especially one aspect, namely the pretense that selected effects explain the presence of the trait¹⁵, which we believe to be simplistic and to promote a distorted view of biology.

3.1 To what extent do SE explain traits?

The intuitive idea is that SE are explanatory is best captured by Wright's account as "the fact that when we say 'A in order that B' the relationship between A and B plays a role in bringing about A" (Wright 1996, p 21). In the biological context, the rationale is often expressed as follows: if the organism's ancestors hadn't had the trait, their progeny would have been unlikely to survive. That is, however, an extremely simplistic view, and the first problem it encounters is that of functional equivalents, raised long ago by Nagel: the function of a trait fails to explain why this solution arose rather than another. But even this presupposes that traits are solutions to selective pressures conceived as design pre-existing problems, in close analogy to designed artifacts. This assumption

¹⁵This is emphasized by the prominent evolutionary biologist and critic of ENCODE's use of function Dan Graur, who for instance writes that the function "explains the origin, the cause (etiology), and the subsequent evolution of the trait" (Graur 2016, p 492), and that "distinguishing what a genomic element does (its causal-role activity) from why it exists (its selected-effect function) is very important in biology" (p 493).

was famously criticized by Lewontin and colleagues (1984, 1985), who argued that rather than adapting to pre-defined problems, organisms both choose and shape their environment. This is critical because it invalidates the claim that if the organism's ancestors did not have the trait, their progeny would not have survived – indeed, in many examples of speciation both organisms with and without a distinguishing trait have survived, but one has, perhaps enabled by a trait, colonized a new niche.

There are also other plausible explanations, as Gould and Lewontin famously showed, such as developmental constraints, which might play a much bigger role than adaptationism leads us to believe. Explaining traits from SE privileges one (very insufficient) part of the explanation over many others, such as which variants arose, drift, etc., when it is unclear why one should privilege one cause over another. The reason we think natural selection is so explanatory is the cumulative effect, namely that each variation leads to increased chances of survival, making a very unlikely series of variations considerably more likely (See Neander 1995; Godfrey-Smith 1999; Godfrey-Smith 2009; Germain 2012). But if functional attribution depended on the explanatory role of natural selection, then this means that traits that arose as a one-off change would not be functional, which is clearly not in accordance with biological practice – even in evolutionary biology.

Additionally, as environments change, so do the ways a trait contributes to fitness. The SE account is therefore faced with three choices: i) if the function of the trait is the “first” one, or the union of all contributions to fitness it has made in its evolutionary history, then a trait might have a function that it never performs anymore; ii) if the function is the most recent one, or the one with respect to which it is still selectively constrained (Neander 2006), then it fails to explain the trait, because the trait would have been entirely different had there not been a previous selection pressure; iii) if the function is relative to a specific time period, then some functions are lost, and the present trait is not explained by the present function.

Millikan's position presented itself as ‘historical’, but we argue that it was not historical enough (see also Griffiths 1996). SE accounts mislead us into thinking that intuitions we attach to design and (vernacular) functions are largely warranted in the case of biological organisms. It leads us to underestimate the historical contingency of evolution, the role of constraints and accidents, and the interconnectedness of traits. It privileges a perspective (certain causal factors above other, certain levels of selection), which would in itself be unproblematic were it not to pretend to some absoluteness, claiming that “proper” functions are such independently of the questions we ask.

In order to solve these issues, Linquist et al (2020) distinguish origin functions (resulting from positive selection) from *maintenance functions* (resulting from purifying selection). Origin functions are always adaptations, while maintenance functions can affect adaptedness (e.g. their deletions cause a decrease in fitness) but do not imply any positive selection. In other words, we can surely infer purifying selection from conservation of sequences, but this tells us nothing about why or how the traits arose. The same applies to the dependencies produced by constructive neutral evolution, so that Linquist et al can sense of the historical contingency of evolution without necessarily ascribing positive selection to all seemingly SE-ascriptions, thereby avoiding a kind of pan-adaptationism “where all traits at a level above that of neutral or nearly neutral variations in nucleotide sequence are assumed to be adaptations which have a ‘function’ create by positive or directional selection” (p 4)¹⁶. An apparent perk of this account is that both functions partly explain the presence of the trait. However, we think this proposal does not solve the core problems. First, setting aside the fact that the distinction between positive and purifying selection is often very difficult to establish empirically, the origin/maintenance distinction and its mapping onto positive/purifying selection is too simplistic. A trait or genomic region can have, over time, more than two ways in which it contributed to fitness, e.g. can be maintained or positively selected over time for different reasons. Secondly, it can be argued that, especially in small effective populations, long-term maintenance also requires positive selection to re-establish deleterious variations that drifted to fixation (see Brunet, Doolittle and Bielawski 2021). Finally, in cases of traits which have lost their origin function, either the trait will be ascribed the lost function (running counter the intuition that a trait F-ing is necessary for it to have function F), or it will only be ascribed to ancestors, in which case the current function does not, as etiological accounts purports, explain the trait.

3.2 Disanalogies in the design metaphor

¹⁶ We interpret the proposal of the weak etiological monism by Brzovic and Sustar (2020) in a similar fashion. Weak etiological monism ascribes functions to traits if they contribute to the current fitness of the organism, and to the fitness of the organism’s ancestor, regardless of how exactly they arose. Fitness is defined as any contribution to complex causal processes involved in viability, fertility, fecundity, and mating ability. In this way, the authors cover even those traits that have been neutrally fixated, but then they have been maintained by purifying selection, and no claim of positive selection has to be made.

Many of the limitations of the SE account just discussed, and its simplistic claim to explain traits, rest on a wrong understanding of organisms as if they were designed artifacts. Indeed, most of the disanalogies (noted for instance by Lewens 2004) between artifacts and the products of natural selection directly underpin some of the aforementioned limitations of SEs. Here, we will highlight the four most critical to our discussion.

First, natural selection relies on the existence of variants, whereas intentional selection does not (or only in a very abstract sense); this means that the design analogy does not consider the causal role, in the origin of a trait, of which variants did or did not arise. There are many examples of initially neutral but nonetheless selectively maintained complex traits (e.g. Starr et al. 2018; Brunet, Doolittle and Bielawski 2021), and characterizing them solely on the basis of their maintenance is losing a fascinating part of their highly contingent history. As we have seen, this is one of the problematic suggestions of the adaptationist paradigm of the SE account's claim to explain traits.

Second, while in artifacts parts are the primary function-bearers, natural selection acts on variants, and as such variants are the paradigmatic function-bearers in SE accounts. Typical exemplars of SE-functions, such as tropotaxis being the function of serpents' forked tongue, suggest that the forked tongue was selected versus a non-forked tongue, which is a gross oversimplification of evolutionary history. As variants and their (SE-)functions are relative to alternatives, it is not so straightforward to go from these functions to the functions of traits – or, for that matter, to the functions of DNA regions.

Third, although we often speak of selection as a force, fitness is essentially a population-level phenomenon and cannot be translated into forces acting on individuals (nor token traits). As Lewens writes, “Discussions of drift often proceed as though drift is caused by things like lightning strikes, while selection is caused by things like predation. There are no grounds for this claim. The forces that explain the individual events in drifty series of births, deaths, and reproductions can be the same forces that explain selective series of births, deaths, and reproductions.” (p.39; see also Walsh et al. 2002; Walsh 2007). The distinction between drift and selection is not in the causes of specific births and deaths, but becomes visible only at the level of populations over time periods (and, for a gray area that is fairly critical for humans, only in a probabilistic fashion). Instead, the distinction is clear in the context of artifacts, where intentions can be appealed to (it is not only hammers in general which have been made with the intention to drive nails, but token hammers as

well). SE accounts suggest a clear distinction between these effects, which leads to the somewhat odd conclusion that of two new variants with the exact same effects increasing in frequency, one in the context of a large population and the other of a small one, one has a function and the other has not, as it is indistinguishable from the effects of drift.

Fourth, Lewens notes that in the design of artifacts, functional specialization precedes the process of ‘selection of modifications’, whereas it is the (potential) outcome in natural selection. This means that while we have good grounds to expect functional isolation in artifacts (due to the way they were designed), we do not for organisms. Indeed, the selective history of traits is not independent, because it is whole organisms, rather than traits, that are selected. As a result, we can expect considerably more interconnected causal contributions than in artifacts, and much fewer cases where a given trait might be given a single more or less autonomous function. As Lewens writes, “the illusion of design does provide us with a way of rationalizing organisms and dividing them up into specific functional traits, even when in reality we should say that all traits are highly multifunctional.” (p 175) Indeed, at the level of genes, contributions to fitness are probably most often so indirect, mingled and non-sufficient that the quest for functions (in terms of either selected effect or individuated contribution to fitness) might be hopeless.

This last point does not apply only to SEs, but to functional decomposition *per se*, and becomes critical in the context of molecular biology where the extent of functional promiscuity begins to be recognized (Nobeli et al. 2009). Dan Nicholson (2013, 2014, 2019) has thoroughly argued that most of contemporary biology, and in particular cell and molecular biology, have considered organisms and cells as machines.¹⁷ While such an approach was very useful in the past, Nicholson argues that it is now hampering progress in biology, and that we need to recognize that

“The cell is not a machine, but something altogether different—something more interesting yet also more unruly. It is a bounded, self-maintaining, steady-state organization of interconnected and interdependent processes; an integrated, dynamically stable, multi-scale system of conjugated fluxes collectively displaced from thermodynamic equilibrium. (Nicholson 2019, p 123)

¹⁷“It is almost ‘natural’ for us”, he writes, “to interpret everything in mechanical or engineering terms because such interpretations accord well with our experience of the familiar macroscopic physical world that we (and our machines) inhabit.” (Nicholson 2019, p 123)

In sum, disanalogies between design and biological evolution are likely to lead us to a misunderstanding of selective process, to ignore the role of developmental constraints, the role of drift and chance occurrence of certain variations versus others, and finally to “underestimate the functional interconnectedness of organic, as opposed to artificial, design.” (Lewens 2004, p 31). While adaptive explanations are powerful explanatory devices, when taken to identify ‘proper functions’ the SE account reiterates these mistakes through a far too simplistic rendering of evolutionary history, which is most visible in its pretension to explain the presence of the traits. While more refined etiological accounts can address some of these problems, we contend that it cannot address all of them while maintaining the main claims of etiological approaches, for the simple reason that these very desiderata come from an erroneous tradition of design.

4 AN ELIMINATIVE PROPOSAL

Given the difficulties discussed throughout the previous sections (i.e. the problems on the one hand of pluralism and ambiguity, and on the other hand of monism in whatever flavour, as well as the connotations bound to be attached to functions because of its vernacular usage), our proposal is that, in biology, we should get rid of the notion altogether (with a proviso to be specified later). This might sound like an extreme and unrealistic proposal, but in fact, despite the prevalence of such language in biology, we argue that it could easily do without it. In this section, we show how our proposal would work in the two biological fields where the controversy about function has been central - namely evolutionary biology and molecular biology. Although challenging in practice, we show that the elimination of the word does not create any substantial challenge in the work of either discipline. If we anyway have to reform the language of function, then the additional costs of getting rid of it are so low, and the epistemic payoffs (in terms of avoiding problems and distortions) so high, that our proposal strikes us as not only sound, but obvious.

4.1 Eliminating functions from evolutionary biology

In his manual on Molecular and Genome Evolution (Graur 2016), Dan Graur gives some nice examples of human variants having recently been under positive selection, and never explicitly mentions their ‘function’. One of the cases is the impressive work by Lamason et al. (2005), who

discovered that a given light-skinned phenotype in zebrafish was caused by a single nucleotide substitution in a gene whose human homolog (*SLC24A5*) shows evidence of strong positive selection in European populations: an allele very rare in African or East Asian populations is present in nearly all individuals of European descent. A number of lines of evidence suggest that the gene impacts melanogenesis (the production of the melanin pigment) through a regulation of calcium exchange (Lamason et al. 2005; Ginger et al. 2008). Pigmentation has a number of effects, positive, negative, or neutral for the organism: it affects how we look like, light scatter in the retina, and the amount of UV making it through the skin and as a consequence both DNA damage due to exposure to the sun and vitamin D synthesis. From this, an educated guess can be made at the evolutionary history: since there is less sun at higher latitudes (and diets were poor in vitamin D), the positive impact of lower pigmentation on vitamin D synthesis outweighed DNA damage, and individuals that carried the allele leading to less pigmentation had a higher fitness, spreading the variation. In brief, a mutation in the *SLC24A5* gene caused lower pigmentation, and rapidly spread in the European population because, most likely, its bearers suffered less from lack of vitamin D and consequently tended to have more descendants.

This brief summary mentions descriptive and historical facts (such as the prevalence of genotypes, environmental pressures, etc.), as well as causal relationships (from genetic to phenotypic differences, and from phenotypic differences to fitness differences). It is a very good case for adaptation by natural selection. It explains (still very roughly) how a variant influences a phenotype, and why it was to be expected that a variant would spread in a given environment. We could venture further and say that it shows the function of the *SLC24A5* gene, or of the variant (already a first major confusion), but what would this further claim bring? Nothing, except misleading suggestions, i.e. that this is *the* function of the gene and that it does nothing else of relevance (unlikely); that the gene exists to synthesize vitamin D (plainly wrong); that the variant was placed there to enable Europeans to produce vitamin D (ludicrous); that East Asians are dysfunctional (dangerously off); etc. Of course, a thoughtful evolutionary biologist will resist these suggestions (though would unconsciously certainly be influenced by them), but the less wise will fall prey to them -- a risk we run for no benefit whatsoever.

4.2 Eliminating functions from molecular biology

Evolutionary biology is not the only context where our eliminative proposal works well. Even the so-called ‘molecular biology’, or ‘functional biology’ can – despite the name – do very well without the word ‘function’. Given that the word ‘function’ in this broad discipline is used with either the concept of ‘biological role’ or of ‘biological advantage’ in mind, we could just simply use these concepts, without even mentioning the word ‘function’.

Much of the literature on mechanistic philosophy, when it refers to molecular biology, could contribute to this attempt to eliminate the word ‘function’. The strategies of decomposition/localization described by Bechtel and Richardson (2010) can be understood and framed in terms of a BR-analysis. We mean that the identification of the locus of control (i.e. where the biological phenomenon is supposed to happen) by segmenting a system from the environment, the decomposition of this locus into components that may in principle causally contribute to producing/maintaining the phenomenon under investigation, and the identification of actual entities which are causally relevant, are all relevant to a ‘functional’ ascription procedure in Cummins’ sense. The same analysis applies to other mechanistic frameworks; for instance, Craver and Darden’s (2013) description of the characterization of phenomena under investigation, construction of how-possibly models that have to be turned into how-actually models *via* modular subassembly, forward/backward chaining etc can be interpreted along the line of Cummins’ functional analysis. In all these cases, ‘functional ascription’ is the activity of identifying an entity and/or a process that is causally relevant to the production of a biological phenomenon – by following a strategy *à la* Cummins, you identify a system to which the entity ascribed a function will contribute to. There is no need to use the word ‘function’, because causal role, biological/physiological role, causal connectivity, causal organization, mechanistic components etc are all terms widely used in the molecular field.

Finally, many biologists are attached to the concept of “functional validation”, which endows a finding with a higher status indicating that it is not merely a description of the properties and activities of molecules, but of how these act together to impact physiology. While the same idea can be expressed without function-language, it is necessarily less succinct. However, it is telling that what constitutes a functional validation very much depends on the scientist: to a molecular biologist, showing the causal impact of a molecule on, say, the level of a gene or the electrophysiological properties of a cell constitutes such a validation, while for others it implies

the observation of a physiological change in a whole organism. It hardly ever translates in fitness, and most of the time represents a mere BR which we might benefit in spelling out.

4.3 Function eliminativism

Searle (1995) has argued, in a more sociological context, that function-language is subjective and eliminable. Recently, Weber (2017) has questioned this claim, particularly on the ground of the multiple realizability of functions: in a nutshell, since the same function can be realized in very different ways, functions are not eliminable because they group different similar contributions, a job that could not be accomplished by their causal (or any other) underpinnings.

Our answer to Weber's objection is twofold. First, we wish to point out that declaring a set of features across species as fulfilling the same function is generally used either as explanatory simplification or heuristic, for instance when the analogy of function is used to hypothesize common descent or selective pressures. As such, however, functions need not be understood as a feature of the world (a view which Weber also seems to resist). Second, admitting the scientific utility of such grouping, we would argue that the same conceptual work can be achieved without function-talk, in particular using Wouters' four concepts: we can for instance say of analogous features that they have similar biological roles or advantages.

It might be argued that, if scientists are anyway always precise as to which of Wouters' four notions they are using, attaching the additional qualificative of 'function' to any of them is harmless enough. However, we think that the word function carries with it a heavy load of implicit intuitions which could go awry even when authors do their best to be clear, and that nothing is truly gained by doing so. There seems to be little ground other than conservatism for insisting on the word.¹⁸

Finally, one reviewer expressed the fear that, in abandoning functions, one would exclusively focus on the question of 'How does it work', while it is the question 'What is it for?'

¹⁸ It might be objected that the elimination of functions from biology should lead to its elimination also in psychology, i.e. that, when ascribing a function to a hammer, we are not doing something qualitatively different than ascribing a function to, say, the behavior of an animal. While arguments could be made for intentionality in sentient vs non-sentient creatures, and for the irreducibility of psychological phenomena, our stance on this question is more pragmatic. Just like we are convinced that the concept of 'free will', though seriously called in question by philosophy and neuroscience, is extremely useful (perhaps necessary) to social life, we think that the concepts of function and intention are extremely useful (and cost-free) to daily human activities. In contrast, we argue, the same concepts are both unnecessary and costly in the biological sciences.

which gives the public much of its interest in biology. First, elimination would still mean having to answer the historical (rather than ontological) question ‘how did it come to be this way?’, and although we agree that there is a funding disequilibrium between the two questions, this has little to do with language or the concept of function, and incomparably more with a society obsessed with growth and the mastery of nature. Second, if we are right in our analysis, then a good part of the interest in ‘What is it for?’ stems from a theological quest for meaning which we think is both erroneous and dangerous, and this is not the sort of public support we should be striving for. We believe there is also a genuine grandeur in our view of life, with its complexity and contingency, and that this is what we should help people recognize.

In sum, we believe that actual scientific practice already has, in contrast to scientific discourse, gotten rid of teleology and replaced it with more precise concepts (such as Wouters’). Its usages of function are either superfluous (i.e. purely linguistic or redundant) or heuristic. Biology has simply not yet acknowledged this in its language, which should now catch up with scientific practice.

CONCLUSION

We began from the observation that there are multiple notions of function in biology, and that ambiguity between them can lead to important mistakes and misunderstandings. We've further argued that the different meanings are difficult to reconcile because selected effects cannot track physiological ascriptions, which have their roots in design and are the fruit of functional decomposition. Furthermore, we've argued that functions, even when understood as selected effects, are far from being exempt from the taint of design and still convey a distorted understanding of organisms and evolution by considering organisms as if they were machines. In face of these difficulties, we proposed that function-talk should be discarded from biology altogether, or if retained then explicitly as a figure of speech. We further argued that this elimination and replacement by more precise notions (such as Wouters’) would actually amount to little change other than a change of language. In light of their history, functions should not simply be assumed necessary or fundamental to biology, but should be demonstrated to be or dropped.

Biological functions are a relic of a time when organisms were understood as engineered machines. Darwin's tour de force was to use the metaphor of Natural selection as an agent to get

rid of the engineer, but we have stuck with the metaphor and the function-talk. Maybe it's time to complete the process.

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