



Functional explanation and the problem of functional equivalence



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ARTICLE INFO

Article history:

Received 9 December 2016
Received in revised form
22 June 2017
Available online 8 July 2017

Keywords:

Functional explanation
Functional equivalence
Etiological function
Teleology
Adaptation
Design explanation

ABSTRACT

The legitimacy of functional explanations in biology is threatened by a problem first identified by Hempel: the problem of functional equivalence. In order for the prevalence of a trait to be explained by its function, the function would have to explain why that very trait is prevalent and not some other functionally equivalent trait. But functions alone cannot meet this explanatory demand. I argue that this is a problem not only for Nagelian deductive-nomological models but also for etiological models of functional explanation. I contrast these models with a dual model of *adaptive* explanation and *design* explanation. This dual model largely circumvents the problem of functional equivalence, but divests functions of much explanatory power.

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1. Introduction

The history of biological thought is replete with differing attitudes about the status of biological functions: are they eliminable from science, or are they epistemically useful fictions, or even robust features of the architecture of the world? Different views on the matter have often played a determinative role for more general commitments about reductionism and the unity of science among philosophers of science and scientists alike.

An important factor for evaluating whether functions are indispensable to biology is whether they have any distinctive explanatory value. If they did, then efforts to reduce or eliminate function-talk entirely would face a principled obstacle. In a *functional explanation*, roughly, the prevalence or maintenance of a trait is explained (in part) by its having a given function. The heart has the function of circulating blood, and it is fixed in humans in some sense *because* it circulates blood. Many philosophers think this form of explanation is defensible, though they differ on the details (Griffiths, 1993; McLaughlin, 2001; Mossio, Saborido, & Moreno, 2009; Nagel, 1961; Wright, 1973, 1976). But any defense of functional explanation needs to contend with the problem that originally motivated Hempel's eliminativist attitude toward functions—namely, the problem of functional equivalence. It seems to be an empirical fact that distinct traits can have the same function: hemoglobin transports oxygen in the blood of vertebrates, whereas hemocyanin plays this role in some invertebrates such as

arthropods and mollusks. This creates a problem for functional explanations: one cannot explain why a given organism has hemoglobin from the fact that it needs to transport oxygen in its blood, because as far as the function is concerned it might have had a functionally equivalent trait like hemocyanin instead. If functions cannot explain this, however, then it is unclear what their explanatory value consists in.

The problem of functional equivalence featured prominently in Hempel's (1959) and Nagel's (1961) early analyses of functional explanation in terms of the deductive-nomological (DN) model of scientific explanation. A consensus subsequently formed that functional explanations could not be conformed to their frameworks due to the problem of functional equivalence, and instead should be interpreted along the lines of the etiological account of functions. Since then, theorists have tended to either ignore functional equivalence or to claim that it poses no problem for etiological accounts (Wright, 1976, p. 101; Salmon, 1998, p. 63; Craver, 2013, p. 146).¹ I aim to show that, in fact, functional equivalence *does* pose a problem for etiological functional explanations, and not only for DN functional explanations. In finally taking this neglected

¹ Evidence that the functional equivalence problem has fallen out of the discussion of biological functions can be seen in the absence of any treatments in recent articles or volumes on functions (e.g., Huneman, 2013a; Krohs and Kroes 2009). Although the phenomenon of functional equivalence seems to be widely recognized, it is generally not viewed as creating a problem for etiological functional explanations. I hypothesize why this has been the case in discussing etiological functional explanations in Section 4.

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problem into account, moreover, we can see that functions are less explanatory than is commonly assumed.

After first introducing the generic relation between functional explanation and function ascription, I define a criterion of functional equivalence. I then show why both Nagel's DN functional explanations and etiological functional explanations are vulnerable to the objection from functional equivalence. This leads to formulating an alternative model comprising *adaptive* explanation and *design* explanation. I argue that these fare better on the problem than etiological and DN functional explanations, respectively. However, we end up with deflationary consequences for the explanatory value of functions.

2. Function ascription, functional explanation, and functional equivalence

To get clear on how functional explanations work, it is first necessary to establish a working understanding of function ascriptions. The following is a generic formula:

Trait T has the function F in individual x under conditions C_n if T 's performing F in x under C_n contributes to attaining the goal G .

In biological contexts, the "goal" G is usually identified with fitness. But G can also be read neutrally as any specific end-state or capacity of interest, which results from T 's performing F and which distinguishes F from other effects of T . C_n denotes the normal or typical set of internal and external conditions under which T does F . A function F is a type of effect—e.g., circulating blood—and a functional trait T is the item causally responsible for the functional effect—i.e., hearts. Finally, a trait must be part of an encompassing individual or reference system, such as an organism. This is the individual that is taken to have a goal G , so that an effect of a trait counts as a function if it positively contributes to G in x . Different accounts of biological function will stipulate stricter conditions for something to count as a function, for example, that tokens of the trait type T must have been actually selected for performing F in the past, but these nuances are screened off from the present context.

In a functional explanation, the explanandum is the fact that T is instantiated in x , and the explanans is the fact that T has function F . For example: hearts (T) exist in humans (x) because hearts (T) circulate blood (F). The objection from functional equivalence is that this sort of explanation will not explain why T_1 is present in x whenever there are other functionally equivalent traits such as T_2 or T_3 that might also have been present in x . This objection needs to be made more precise by defining a criterion of functional equivalence.

Functional equivalents are traditionally understood as distinct traits that have the same function. Otherwise expressed, two traits are functional equivalents iff:

- (1) they do not belong to the same trait type, and
- (2) they have the same function.

The reason for invoking types in (1) is that functional explanations typically do not purport to explain differences between trait tokens that belong to the same type. So, for example, two human hearts differing solely in mass should not be considered functional equivalents with respect to the function of circulating blood. Functions are not expected to explain this sort of physical difference, and the difference is also not relevant to the main biological factors that define the trait type "hearts." In different explanatory contexts, however, trait types can be individuated in different ways. In evolutionary biology, trait types are often individuated by *homology*, or shared descriptive properties due to common descent. I suggest that homological trait distinctness is the relevant factor for evaluating condition (1) in the context of most evolutionary

functional explanations, though we will see that other trait types operate in other kinds of functional explanations.

To be homologically distinct here just means that the traits in question do not belong to all of the same homology trait types. Two things can be noted about this qualification. First, since traits can be individuated at different levels of homology, traits can belong to different types at one level and to the same type at another level. For example, fish hearts and mammal hearts are both animal hearts. That is why traits that are homologically the same should belong to *all* of the same types; otherwise they have some relevant degree of homological distinctness. Second, the qualification is negative so that functional equivalents can include evolutionary novelties, which belong to no homology types at all (Müller & Wagner, 1991, p. 243).

Next, to evaluate condition (2) that two traits have the same function, we need some idea about how functions are individuated. This difficult issue has received surprisingly little attention in the extensive literature on biological functions (although see Wimsatt, 2002). Given that functions are effects of the activity of traits, a causal description of the activity of a trait should include a causal description of the function. However, well-known issues concerning the granularity of causation enter the picture at this point, for causes and effects can be individuated with more or less specificity. The function of the heart can be described as "circulating blood," or "circulating blood at a certain rate," or "circulating blood at a certain rate and pressure," and so on to include even highly specific properties. Although there may not be a uniquely correct description in such cases, the granularity of the causal description does get constrained by the aims set by the background account of function. In those accounts in which functions are supposed to explain something about their bearers, the function cannot be described in terms that are so general that it is no longer explanatory. At the same time, if functions are supposed to ground normative claims about what a trait is "supposed" to do, they cannot be described in terms that are so specific that any small deviation constitutes a dysfunction. Finally, those accounts that make some claim of descriptive adequacy cannot depart too far from biological practice in how they individuate functions. These factors will become important for evaluating etiological functional explanations later on.

Additional restrictions on which traits count as functional equivalents might arise from different forms of functional explanation. When functional explanations are grounded in natural selection, as in etiological explanations, this imposes certain historical restrictions on which kinds of functional equivalence can be taken to undermine the explanation. Because selection can only act on actually present heritable variations if it is to result in evolution, the relevant class of functional equivalents should include only those heritable variations that have been available to an evolving population at the same time. It is therefore no objection to an etiological functional explanation of why humans have hearts that it fails to explain why humans do not have fish hearts, because fish hearts were never a competing variant in an evolving population of humans. Such contrasts lie outside the scope of what etiological explanations purport to explain. As we will see, because Nagel's DN functional explanations are framed in terms of taxic laws rather than selection, they do not have the same historical restrictions on their explanatory claims, and so are more vulnerable to the objection from functional equivalence.

3. Nagel and Hempel on functional explanation

According to the deductive-nomological model of scientific explanation, a legitimate scientific explanation consists in the subsumption of the explanandum under one or more general laws

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