

Function, Modality, Mental Content: A Response to Kiritani

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I clarify some of the details of the modal theory of function I outlined in Nanay (2010): (a) I explicate what it means that the function of a token biological trait is fixed by modal facts; (b) I address an objection to my trait type individuation argument against etiological function; and (c) I examine the consequences of replacing the etiological theory of function with a modal theory for the prospects of using the concept of biological function to explain mental content.

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I gave an argument against the existing theories of function in Nanay (2010) and outlined a new modal theory. The argument, briefly, is that all existing theories of function define the function of a token trait in terms of some properties of some other (past, present, future) traits of the same type. They define the function of my heart in terms of some properties of other hearts (say, the hearts of my ancestors). Hence, these theories need to give an unproblematic and non-circular account of trait type individuation: of what makes hearts hearts. But, I argue, the least problematic accounts of trait type individuation appeal to the traits' function — thus, we cannot use them as part of the definition of function without running into circularity (see also Nanay, 2006). My positive account is that we need to define the function of a token trait with the help of modal claims — in terms of what the trait would do if things were somewhat different. In short, my suggestion is that we should define function in modal terms.

Kiritani (2011) raises two questions about my argument and my account. First, he points out that according to the most popular (or least unpopular) contemporary theory of function, the etiological theory, the theory according to which x has a function to F just in case X s in the past have been selected

for doing F, function-attributions carry modal force — just like according to my own account. And, second, he suggests that the etiological theory can be salvaged if we use a non-etiological theory of function for individuating trait types because then we can use this in the definition of etiological function without any risk of circularity. I clarify these (undoubtedly not sufficiently developed) aspects of my account in turn and then end with some thoughts about how this modal theory of function could help us to address the question that sparked much of the function literature in the last thirty years or so: the question about whether we can explain the content of our mental representations in terms of their function.

Modal Force

My explanatory scheme explains function in modal terms. The *explanandum* is function, the *explanans* is constituted by modal facts. This is what makes my account a “modal theory of function.” Kiritani points out that according to etiological theories of function, function attributions also have modal force. But what he means by this claim is very different from what I do. For Kiritani, it is not the *explanans*, but the *explanandum* of the explanation of function that has modal force. The way Kiritani argues for this claim is to point out that function attributions have normative force: they tell us what a trait should or ought to do. And he then states that “the auxiliary ‘should’ or ‘ought’ can be regarded as expressing modal force” (Kiritani, 2011, p. 2).

The first thing to note is that there is a lot of recent discussion on how “should” and “ought” should not be regarded as expressing modal force (see Cariani [in press] for a good summary). Second, and more importantly, even if Kiritani’s analysis of the auxiliary “should” and “ought” were correct, this would imply that according to the etiological theories, the *explanandum* of function explanations carries modal force. But as the *explanans* of these theories (*Xs* have been selected in the past for doing F) does not carry modal force, the proponents of these theories would need to have an account of how modal facts can be explained in terms of simple events in the past — which could, in itself, be thought to be a way of pointing out that the etiological theories are problematic.

I do think that there is an interesting question in the vicinity about the explanation etiological theories give. As I say in a footnote in Nanay (2010), “It has to be noted that at least some versions of the etiological notion of function could also be interpreted as carrying modal force — whether they do depends on how we interpret the concept of ‘contribution’ in the definition of function” (p. 421, footnote 24). Focusing on the modal force of “contribution” would give a better support for Kiritani’s claim about the modal character of etiological explanations. But I fail to see why this constitutes a problem for my account. The reason I give for why etiological theories are problematic is not

that they fail to attribute modal force to the *explanans* of functional explanations, but that they define the function of a token trait in terms of some properties of past instances of the same trait type and this, as we have seen, presupposes an unproblematic and non-circular way of individuating trait types. If this argument is correct, then etiological theories of function fail regardless of whether their *explanans* has modal force.

Cummins Functions

Kiritani's (2011, p. 3) second question is about a way of salvaging the etiological theories of function, or, rather, defending them against the trait type individuation objection. The suggestion is this: we could use Cummins functions to individuate trait types and then plug this way of individuating trait types into the definition of etiological function. Here is Robert Cummins's definition of function that Kiritani proposes for the purposes of individuating trait types: "x functions as a γ in s (or: the function of x in s is to γ) relative to an analytical account A of s's capacity to ψ just in case x is capable of γ -ing in s and A appropriately and adequately accounts for s's capacity to ψ by, in part, appealing to the capacity of x to γ in s" (1975, p. 762).

This is a possibility that I only addressed in a footnote, and quite briefly, so I need to say more about it. I agree that using Cummins functions to individuate trait types and then using this way of individuating trait types to define etiological function is not circular. But as I noted in the original article (Nanay, 2010, p. 416, footnote 10), this is not a very promising way of individuating trait types. Note that in Cummins's definition of function, x's being capable of γ -ing is a necessary condition for x's having a function to γ . In other words, if we used Cummins function to individuate trait types, only those hearts would be hearts that are capable of pumping blood. But this is blatantly false: malformed and defective hearts are still hearts, but they may not be capable of pumping blood.

Function and Content

Finally, I want to address a question that I did not have space to address in Nanay (2010) and one that provides the subtext of Kiritani's article. It is also presumably the reason why this discussion is of interest to the readership of this journal. The question that triggered much of the research about the concept of biological function is whether we can explain the content of mental states in terms of the function of these states. When I think about a cat, my thought is *about* this cat: it *refers to* this cat. Cat is the *content* of this thought. One grand question in philosophy of mind is how to explain this mysterious relation between a mental state and what it is about. And an influential suggestion (Millikan, 1984; Neander, 1995; Papineau, 1993) is that we can explain the content of our repre-

sentations in terms of their function: the content of a representation is X just in case its function is to indicate X s. The project of explaining mental content in terms of function is sometimes called “teleosemantics.”

Teleosemantics uses the implicit assumption that function is etiological function. So my attack on the concept of etiological function could be taken (and this is, I believe the way Kiritani took it; see p. 1) as an attack on teleosemantics. I don't think this follows. In fact, I think teleosemantics may be quite promising as a way of explaining mental content, if we take function to be fixed not by history, as the etiological theory suggests, but by modal facts, as I argued. Many arguments against teleosemantics are in fact arguments against the etiological theory of function — the most famous example being the “Swampman objection.” Thus, it may be a good idea to look for ways of formulating teleosemantics without relying on the etiological theory of function. If we replace the etiological theory of function with a modal one, this does not need to mean that we need to dispose of teleosemantics. It could also usher in a way of resurrecting it.

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