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On the Theoretical Motivation for Positing Etiological Functions.

Abstract: This paper argues that the widely accepted etiological notion of function finds no solid support in biological theory. While the notion may seem to be called for by entrenched linguistic practice, I claim that the arguments that have been raised in its favour do not succeed in providing a theoretical motivation for it. This verdict is bound to transfer to philosophical contexts where the notion is put to use, teleosemantics being the perhaps most debated application.

1. Introduction.

It is a plain fact that biology makes use of terms and expressions commonly spoken of as teleological. Biologists frequently speak of the function of biological items. They may also say that traits are "supposed to" perform some of their effects, claim that traits are "for" specific effects, or that organisms have particular traits "in order to" engage in specific interactions. There is general agreement that there must be something useful about this linguistic practice but it is controversial whether it is entirely appropriate, and if so why it is.

Many theorists have defended the use of seemingly teleological terms by appeal to an *etiological notion of function* (Wright, 1973, Millikan, 1984, 2002, Neander, 1991, Griffiths, 1993, Godfrey-Smith, 1994, and Buller, 1999). According to the etiological notion attributing a function to a trait is a matter of pointing to effects that account for why the trait has been selected for. On an alternative but related formulation function statements indicate that past tokens of the targeted type contributed to the existence of current token traits in virtue of exhibiting the effect at issue. A central feature of etiological definitions is that they come with a requirement of ancestry; there must have been type-identical tokens exhibiting the targeted effect in order for a trait to have an etiological function. Trivially, 1st generation tokens lack such ancestors as far as the novel trait is concerned and therefore lack etiological functions.

In this paper I will home in on the theoretical motivation for the requirement of ancestry, a requirement that renders the resulting functions *essentially* etiological. I will argue that the positing of etiological functions is theoretically ungrounded. Not that there aren't any tokens that meet the description, but this is far from sufficient. So,

for instance, there is a real difference between tokens with a trait T that are born 2000 years or more after the first token with T, and tokens that are born earlier. We may then add to a standard etiological definition of 'function' the requirement of being born 2000 years or more after the first mutant and let '*munction*' stand for the resulting historical property. Are there munctions? Well, if all that is required in order to answer affirmatively is that there are tokens that fit the description then the answer is clearly 'yes'. However, there is no known reason for stressing that particular difference and therefore no reason to posit munctions. Likewise, the point of this paper is that there is no theoretical reason to stress the difference between 1st generation tokens and later ones and thus no reason to conceive of biological functions as essentially etiological.

There is nothing new about voicing negative verdicts about etiological accounts of 'function' (Cummins, 1975, 2002, Enç and Adams, 1992, Walsh and Ariew, 1996, Davies, 2000a, b, McLaughlin, 2001, Lewens, 2004). I take my approach to differ from, and be a useful complement to, that of others in the following respects: I press the question of the rationale for the exclusion, by definition, of 1st generation tokens more consistently than has been done elsewhere. I attempt to run through the main considerations etilogists have brought to bear on the topic. In the process I look into the claim that the etiological notion of function is required for biological categorisation, and critically address relatively recent proposals made by etilogists on this issue. I look at Millikan's (1984) explicit motivation for her 'proper function' – that it is required to capture explanatory analogies between domains – and find that this motivation fails to suggest an etiological notion as opposed to a non-etiological one. I provide a rather thorough critical discussion of the idea, made popular by Wright (1973), that the etiological notion is vindicated by its role in explaining the existence of traits. I also discuss the possibility of finding support for the etiological account in the writings of prominent biologists and conclude that the textual evidence is not unequivocally in favour of an etiological account. All things considered, I take my account to strongly suggest that the etiological account of functions gets its support from a pre-theoretical mindset, not theoretical considerations.

The etiological notion of function is encountered in several areas of philosophy. It is invoked in defending *realism* about biological teleology (Wright, 1973, 1976, Neander, 1991). It is a central ingredient in *teleosemantics* (Millikan, 1984, Papineau, 1993, Neander, 1995), and teleosemantical views about content appear in philosophy of mind, philosophy of language, and epistemology. Furthermore, the notion appears

in philosophy of medicine/psychiatry in attempts to separate disease/disorder from health (Wakefield, 1992). These applications clearly rely on the belief that the etiological notion of function is theoretically respectable. Consequently, if it turns out that there is currently nothing apart from theoretically insignificant pre-theoretical hunches to speak for it the applications will be cast in doubt as well.

The rest of the paper is structured as follows: In Section 2 I will present and contrast etiological and non-etiological accounts of 'function', and introduce the basic considerations that motivate the adoption of the etiological version. Throughout 3 I address what has been said in favour of etiological functions and argue that none of these considerations points to the need of positing such functions: 3.1 addresses the role of function in biological categorisation, 3.2 discusses Millikan's explicit motivation for 'proper function', and 3.3 deals with the claim that function ascription amounts to explaining the existence of traits. Finally, in 3.4 I discuss seemingly supportive attitudes among prominent biologists and argue that the textual evidence cannot be taken to indicate a commitment to an etiological definition.

A terminological note: I will use '*T-term*' to denote terms commonly spoken of as teleological, wishing to indicate non-commitment as to whether they are to be understood as truly teleological or not in the context.

2. *The Promise of the Etiological Notion of Function.*

As is well known, several proposals have been made through the years to the effect that T-terms within biology should be defined in terms of selection. The approach is recommended by George C. Williams in *Adaptation and Natural Selection* (1996/1966, in which he mentions Muller, 1948, Pittendrigh 1958, and Simpson, 1963, as predecessors), and it has been proposed by a large number of philosophers since then.

The category of selectionist proposals can be split into two main sub-groups: *etiological* accounts and *non-etiological* ones. What is distinctive about etiological, or *backward-looking*, definitions of 'function' is the requirement of ancestry; a trait's function-hood requires the trait to have a history of selection, or at least a history of contributing to survival and reproduction.¹ A 1st generation trait doesn't have such a history and so it follows that it lacks functions.

¹ I acknowledge Buller's (1998) distinction among etiological theories between "strong" ones that

It is clear that if we use 'function' as a placeholder for appeals to selection, these appeals will frequently invoke past events. It is equally clear that, frequently, several generations may have to pass before we will be in a position to support a hypothesis about selective advantage. However, etiological definitions do not allow 1st generation traits to have functions even with the benefit of hindsight; the exclusion is a matter of metaphysics, not epistemology. Proposals of this kind include Wright (1973, 1976), Millikan (1984), Neander (1991), Griffiths (1993), Godfrey-Smith (1994), and Mitchell (1995).

Non-etiological (and yet selectionist) accounts, on the other hand, accept that there may be functions in the very first generation. Proposals of this kind are found in Bigelow and Pargetter (1987), Horan (1989), Walsh (1996), Walsh and Ariew (1996), and Lewens (2004). Such accounts can be viewed as applying a *causal role* account of 'function' (Cummins, 1975, Amundson and Lauder, 1994) to contexts where the relevant role is contributing to fitness (Buller, 1998).

It is important to see that the distinction between etiological and non-etiological notions of 'function' is a distinction based on definitions, not on whether the notions may be used in etiological explanation or not. 'Etiological function' is to be read as implying, by definition, the exclusion of 1st generation traits, whereas 'non-etiological account' applies to views that allow the ascription of functions to 1st generation traits. The non-etiological notion can of course be used in etiological explanation. The fact that the expression 'fitness advantage' may be used in an explanation of how a currently fixed variety became fixed doesn't render the expression essentially etiological. Our demand for etiological explanation doesn't require organisms of the kind to have type-identical ancestors in order to count as having the relevant fitness advantage.

Why should anyone opt for the etiological version then, given its "aversion" against 1st generation traits?

There clearly is a pre-theoretical reason to be found. It doesn't sound quite right to say about a 1st generation trait, such as an ecologically useful preference, that it is supposed to achieve anything. However, once the trait is common it may well pertain to the pre-theoretical extension of T-terms. Then, prevailing linguistic practice may

demand selection to have occurred and "weak" ones that merely require a contribution to survival and reproduction to have occurred. Both varieties are implicated here since my account targets the etiological requirement as such.

seem to call out for a distinction between 1st generation tokens that are "accidental", and type identical later tokens that are "teleologically oriented" towards the relevant consequence. This is the distinction the etiological notion of function promises to capture. In this manner we may accommodate the perhaps very intuitive idea that applications of T-terms within biology are of a kind with applications within the intentional domain.

As for the intentional context, if there is any theoretical point to expressions like 'purpose' and 'supposed to', then they shouldn't apply to cases where useful effects appear accidentally. We already have ways of speaking of mere consequences, good or bad, so such usage would be unneeded. Restricting the expressions to occasions when the consequence is due to the right kind of history, involving a fitting intention, is advisable. Then, in order to connect to what is considered teleological in the domain of human activity we need biological functions to be etiological rather than non-etiological. Since intentions are out of the question (for non-creationists) something else must be invoked instead as the relevant etiological factor. An appeal to selection clearly brings about the wanted elimination of 1st generation tokens as there hasn't been selection for the targeted trait at that stage.

Many have assumed that the pre-theoretical range of application of T-terms, which includes biological traits as well as intentional action and artefacts, can be given a unified theoretical justification. Larry Wright (1973) presents an account of 'function' according to which function ascription is a matter of explaining the existence of traits by appeal to consequences of such traits (this proposal will be the topic of **3.3**). Karen Neander claims that the etiological notion of function is (1991, 467) " ...the 'conceptual glue' of biology...", and she speaks of the discipline as genuinely teleological. The use of 'genuinely' indicates that a connection to paradigmatic cases is assumed. Ruth Millikan (1984) introduces a technical, etiological definition of 'proper function' and holds that this notion is especially interesting because, she takes it, it promises to provide a unified account of purpose across domains. She claims that a benefit of her analysis is that "...a connection between human purposes and the natural purposes of bodily organs and instinctive behaviors will be established that does not rest on mere metaphor." (1984, 4). Millikan then hopes to be able to naturalise intentionality by appeal to proper functions. The possibility of misrepresentation has been a major problem for such attempts, and it has struck many as *prima facie* plausible to take this possibility to be secured by the possibility of malfunction. It is

commonly held that this requires a notion of function that is in some sense normative, and the etiological account of function has appeared promising in this respect (Neander, 1995, 112, Macdonald and Papineau, 2006, 11-12).

Still, however intuitive and promising the etiological account may seem, I will now turn to argue that we simply haven't been given any theoretically sound reason to posit essentially etiological functions.

3. The Quest for Support for the Exclusion of 1st Generation Traits.

Again, the aspect of the etiological notion of function I will concentrate on is the "aversion" against 1st generation traits. Given the lack of causally relevant differences between 1st generation traits and later ones, positing etiological functions makes *prima facie* too much of location in the lineage (Enç and Adams, 1992, Walsh and Ariew, 1996, Davies, 2000a). Consider such functions from the point of view of fitness. 1st generation tokens may be causally indistinguishable from later, post-selection tokens in relevant respects. If fitness supervenes on causally relevant properties it follows that the survival and reproduction of biological items is insensitive to whether history bestows functions on them or not. What matters is that they have effects that are useful for survival and reproduction, but these clearly do not require type-identical ancestors in order to be. The same argument applies, *mutatis mutandis*, to the variety of content teleosemantics reckons with. Since a 1st generation individual may have everything later individuals have as far as biological utility is concerned and yet lack the content the descendants have in virtue of history, it would seem that such content makes no biological difference. I think that this *prima facie* oddness about etiological function and its derivatives warrants some suspicion.

Evolution deals with descent with modification and biologists need to take the relevant kinds to begin with the relevant modifications, prior to selection, that is. Novel types appear through modifications and if they are fitter than prevailing varieties they may spread. This kind of explanation does not demand function categories that postpone membership until the right kind of post-modification consequences have occurred.

Now, selective regimes may change such that what was once adaptive no longer is. That is, effects that once made for selection may no longer do so. This means that we need ways of indicating whether we are discussing a currently adaptive feature or one

that once was adaptive. Thus, if 'function' is neither essentially etiological nor essentially forward-looking, function statements may be ambiguous with respect to time. However, we clearly don't have to invent an essentially etiological definition of function (nor indeed an essentially forward-looking definition) in order to disambiguate the situation. There are other means of doing that. This is a trivial and quite general point. The properties that made an athlete rise to the top may very recently have ceased to be efficient due to changes in the rules of the game and/or tougher competition. So, there may be contexts where we need to indicate the time dimension explicitly. However, we presumably wouldn't do that by introducing a definition of the relevant property term according to which causally similar prior instantiations are required in order for the term to apply.

We also need *ontogenetic* explanation that, roughly, deals with how traits develop from the fertilised egg onwards. Now, since such explanation is causal, it needs conceptual tools that are useful from this point of view. The problem with 'etiological function' is, again, that the notion distinguishes between causally indistinguishable tokens; a 1st generation trait may be like a later one in all causally relevant respects and yet lack the function of its descendant. It is hard to see how ontogenetic explanation could demand such a concept.

3.1. Etiological Functions and the Forming of Categories.

Neither evolutionary nor ontogenetic explanations call out for a notion of function-hood that excludes 1st generation tokens. Now, I don't think this will be disputed by etilogists. It is likely, however, that some will insist that the etiological notion is required for the purposes of categorisation (Millikan, 1989, Neander 1995, 2002).

An uncontroversial feature of biological categories is that they include various "deviants" as well as those we deem representative. In Karen Neander's terms, the categories are *abnormality inclusive* (Neander, 2002, 392). Furthermore, the morphological differences within kinds may be substantial. So, it is claimed, we cannot categorise on the basis of structure and/or actual capacities. True as this is there is a basic problem with the idea that appeals to function will help us include deviants. Stating the function of a trait does nothing *per se* to suggest which tokens belong to the type. If we point to a thing that doesn't pump blood, stating that hearts were selected for pumping blood doesn't tell us whether the targeted thing is in fact a heart (see Davies 2000b for discussion).

An intuition that appears in this context is that it wouldn't make sense to ask of a *swamp creature* whether it is normal or deviant (Millikan, 1996, Neander, 1996). Bringing this intuition over to realistic cases involving novel mutants will then make for the verdict that the idea of being deviant is senseless without a history of selection to establish the relevant "norm". However, one can argue that this sentiment simply reflects the pre-theoretical assumption that a biological trait's being deviant is somewhat like an outcome of an intention that is not as intended, which very sentiment is at stake here. It may well be true that we won't be, when first facing the mutant, in a position to say much of categorical value about this novel trait. This doesn't rule out the possibility of claiming with the benefit of hindsight (should the first mutant be well documented) that this first token was actually a quite unusual and non-adaptive version of a trait that has since spread due to its beneficial effects. That is, given enough information we should be in a position to relate this token to later tokens in the same manner we relate current deviant tokens to earlier adaptive ones. We might of course seldom or never have enough information but, again, on the etiological definition the etiological character is a matter of metaphysics, not epistemology.

Now, etiologists probably deem it inappropriate to use 'abnormal' about 1st generation tokens, but on my view this merely reflects their commitment to the pre-theoretical ring of T-terms. If we spoke instead of variants that don't exhibit the effect that accounts for the spread and/or persistence of the relevant type, which is all there is to being "abnormal" in the context of relating function to selection, there should be no reason to discriminate on the basis of location in the lineage.

We can illustrate the uselessness of appeals to function in forming abnormality inclusive categories by considering something as familiar as our thumbs. There are humans whose thumbs lack the capacity to oppose the other fingers, which is considered a crucial matter when it comes to the selection of opposable thumbs. Assume that someone doubts that such "things" are thumbs at all. Let's assume further that the kinds of comparisons we could make as regards placement, form, tissue, cells, genes, and development do not suffice to claim that we are dealing with thumbs. Now, the functional (*qua* selectional) perspective will only give us the following conditional claim: If the targeted items are thumbs then they have the function of opposing the other fingers in grasping. But clearly, what we need is sufficient reason to think that the antecedent is true. If the other considerations fail to link the targeted items to

paradigmatic thumbs then the link cannot be made at all. Thus, we seem to be facing a *reductio ad absurdum* of the view that the inclusion of deviants require appeals to selectionist function, at least if we believe that we indeed have sufficient reason to accept our entrenched categorisation. We must then conclude that the comparative evidence suffices. But then we have not been given any reason to deny that it would suffice whatever the time-order between the compared tokens.

Since appeals to function won't serve to relate deviants to non-deviants, the need for abnormality inclusion will not speak in favour of the essentially etiological account of function and against the non-etiological one.

Leaving the issue of abnormality inclusion behind, it is clear that there are other things for appeals to function to do. Biology clearly trades in wide functional categories that span traits of different origin and thus are not *homologous*. Such categories are formed by appeal to similarities in selective respects (*analogies*) and are frequently invoked in general approaches to animal behaviour (e.g. Ridley, 1995). Also, Karen Neander (2002) has argued that function has a further role to play even within homologous kinds, since these may include sub-kinds that have diverged markedly since different sub-populations have been exposed to very different environmental challenges. Neander claims that such categorisation "...is a matter of drawing conceptual lines at those places where there is a significant change in what there is or was selection for" (2002, 413). In a recent paper Rosenberg and Neander (2009) argue for an even more fundamental role for function; they claim that the very concept of homology often relies on functional considerations.

Neander is clearly right that there are categories that are formed by appeal to analogy, both across and within homologous kinds. I am less convinced by what is stated about the relation between function and homology in her collaboration with Rosenberg. Whatever one thinks of this, what matters here is that there is nothing in these accounts that indicate the need for a definition of 'function' that renders functions essentially etiological.

To begin with, kinds formed on the basis of analogy merely require that traits get categorised with respect to effects that account for selection and to this end we do not need to drive a wedge between 1st generation effects and later ones. If a mutation produces, say, a novel and ecologically useful aversion then there is no reason not to include the first mutant in the novel functional category.

Secondly and similarly, as for functional sub-kinds within homologous kinds, people who use 'function' in accordance with a non-etiological account are perfectly capable of acknowledging the changes and differences in "selective regimes" that Neander is appealing to. That is, one is not incapable of granting Neander's point merely because one takes novel functions to appear at the very moment of novel mutations or sudden environmental changes that redistribute fitnesses.

As for the relation between function and homology, even if Rosenberg and Neander (2009) are correct in what they say about this they haven't thereby vindicated the etiological notion of function. What they argue for is that the concept of homology depends on tacit appeals to selection. They hold that homology "...is a property of traits which are often individuated by their functions in the [selected effect] sense, and selected effect traits are traits with a certain type of ancestry..." (Rosenberg and Neander, 2009, 309). However, as a footnote (p. 309) as well as the very title of their paper² indicates, they do not claim to argue for the etiological account in this context. By their own explicit remarks they do not take themselves to be providing any reason to prefer an essentially etiological notion of function to a non-etiological alternative.

More substantially, it would be very odd if the concept of homology came with the exclusion of 1st generation tokens. Descendant tokens of some type T are trivially connected by descent to the ancestral mutant that introduced T. So, for instance, my opposable thumbs are trivially homologous to the ones possessed by my paternal grandfather and they would have been so even if the trait had begun with him.³ Then, whether or not homology requires shared functions there is reason against thinking that it requires shared etiological functions since these exclude the 1st generation.

A non-etiological notion of function is sufficient to do the crucial work when it comes to categorisation. This work involves allowing categories based on analogy, making functional distinctions within categories, and, if Neander and Rosenberg are correct, helping out to establish homologous kinds to begin with.

3.2. Millikan's Motivation for Introducing 'Proper function'.

² The title is "Are Homologies (Selected Effect or Causal Role) Function Free?"

³ Not that I think that our kind of opposable thumbs arose through a single mutational event from clearly non-opposable thumbs. Hopefully, this lack of realism won't obscure the point made about the relation between etiological function and homology.

Ruth Millikan is presumably the philosopher who has devoted most time to defining an etiological notion of function: 'proper function'. The motivation she presents when introducing the term is that she wants it as a tool to make clear explanatory analogies between language devices and biological devices in general (Millikan, 1984, 4). However, there is really nothing in this motivation that calls out for 'proper function' as a conceptual tool. If the same kind of explanation of persistence applies within both domains this can be acknowledged without introducing a notion that excludes 1st generation tokens. We could, for instance, simply speak of 'persistence effects' and use our ordinary manners of dealing with tense to express whether we are dealing with persistence in the past, current persistence, or predicted future persistence. There is no reason to believe that such a conceptual scheme would be less powerful when it comes to providing a domain-general account of utility and persistence.

It is very plausible to assume that Millikan's choice of the essentially etiological 'proper function' rather than some non-etiological alternative like 'persistence effect' is due to her wanting the proposed term to have the desired relation to 'purpose' and 'supposed to', which latter terms do not, pre-theoretically, sit well with 1st generation tokens. However, in the context of introducing 'proper function' Millikan does nothing to argue for the tenability of this desideratum. She claims that in virtue of 'proper function' "...a connection between human purposes and the natural purposes of bodily organs and instinctive behaviors will be established that does not rest on mere metaphor." (1984, 4). This is merely assuming that her account will be theoretically sound as regards "natural purposes", which is not to provide a reason for that assumption.

I think we need to be aware that 'proper function' may be quite misleading in the context of accounting for meaning and intentionality in that it has us express clearly descriptive historical claims about spread and persistence in terms with normative connotations. Peter McLaughlin gives clear expression to what we don't want to happen (2001, 10):

[T]here is a very real danger that the vested interests of philosophy of mind in intentionality lead it to foist more teleology upon biology than the biologists need or want by providing a self-indulgent analysis of functional explanation.

An account of the proliferation of some feature in terms of its effects in specified circumstances does not as such seem to invite the idea that, for the very reason of proliferation, current tokens are supposed to achieve anything. However, if we choose to discuss proliferation in terms of proper functions this is more likely to prime normative sentiments. So, perhaps 'proper function' does help to close the gap between selection and intentionality, but in a way it shouldn't. We shouldn't have the ring of words achieve what it ought to take arguments to do. It seems an open, perhaps testable, issue whether something of the kind is at play here.

3.3. Etiological Function and Explaining the Existence of Traits.

Etiological theorists commonly endorse the view that teleology involves a specific kind of explanation where one appeals to consequences of traits to account for their presence (Ayala, 1970, Wright, 1973, Brandon, 1981, Millikan, 1984, Neander, 1991, Godfrey-Smith, 1994, Mitchell, 1995, Buller, 1999). The proposed distinguishing mark of teleology is that effects of a feature F are relevant in accounting for the existence of Fs.

The classical locus for the idea that teleology is about explaining the existence of traits is Wright (1973). Wright suggests that the statement 'The function of X is Z' is true iff (1973, 161):

a) X is there because it does Z, and b) Z is a consequence (or result) of X's being there.

It is uncontroversial that, in order to avoid positing backwards causation, the first clause cannot be read as saying that a token X is there because that token does Z. Rather, the common view is that, in standard biological cases, later token Xs are there because ancestral type-identical tokens did Z (Millikan, 1984, Neander, 1991, Griffiths, 1993, Godfrey-Smith, 1994). Consequently, this variety of explanation cannot, in biological cases, apply to 1st generation tokens, or at least not to the very first manifestation of the effect at issue (Wright, 1976, 114). So there will be a distinction within biological categories between (later) tokens that have teleological explanations and type-identical (earlier) tokens that are merely "accidental".

There is certainly something to be said for this. It seems clear that while we cannot invoke fitness to explain a 1st generation token there is some connection between fitness and later tokens. If a trait lacks fitness then there will very likely not be many

descendants with the trait. It seems then that when traits have become widespread due to superior fitness the existence of later tokens is probabilistically related to the fitness of earlier tokens. I think this must be granted. However, if we bring out the general features of this line of reasoning we stumble on a problematic matter; it involves us with an arbitrarily restricted explanatory mode.

No doubt, many current token features do depend for their existence on the fitness-generating activities of ancestral type-identical ones. However, this dependence is not confined to hold between type-identical tokens. It holds for biological features in general that the existence of a current token *F* typically depends on prior *F*s, but also on a myriad of interactions executed by other traits in the lineage from the beginning of life onwards. A token *F* will stand in the same explanatory relation to all traits of its ancestors that made a contribution to survival and reproduction. For brevity, I will simply refer to the non-*F* traits among them as *con-F*s. The account of the explanatory role of T-terms proposed by Wright and others does not allow that we explain the existence of *F* by stating the function/purpose of any *con-F* since this explanatory scheme demands that it is effects of type-identical tokens that figure in the explanation. An explanatory mode with such a constraint seems quite arbitrary. Current opposable thumbs are surely there due to activities of past thumbs, but also due to the activities of past hearts and lungs (see Davies, 2000b, 28-29, and Lewens, 2004, 98-99, for related discussion). However, by citing the function of the heart we can, according to the explanatory account of T-terms at issue, explain why there are hearts but not why there are opposable thumbs. But effects of past hearts help explain the existence of any current trait the existence of which depends on effects of past hearts.

Explanations of spread and maintenance of traits, on the one hand, and explanations of the existence of traits, on the other, fare radically differently as regards arbitrariness. If we want to explain the spread and maintenance of *F* then obviously we will have to focus on *F*s; looking at just any *con-F* cannot be expected to reveal the superiority of *F* over the alternatives. If, on the other hand, we wish to turn the selectionist account into one that explains the existence of current *F*s we will get an arbitrarily restricted explanation; the activities of prior *F*s are not different in kind from the activities of prior *con-F*s *qua* conditions for the existence of current *F*s. Then, are we to believe that while we needn't invoke essentially etiological properties when

we account for how the existence of current thumbs depends on past heartbeats, when we account for how it depends on the grasping of past thumbs we do?

It should be obvious that the explanatory "potential" at issue here cannot be something that renders the positing of essentially etiological functions important for biology. It simply cannot be important to use a different manner of explaining existence, one that utilises T-terms, when type-identical tokens are involved than when they're not. Thus, we are not, by turning to explanations of existence, given any reason to insist on an essentially etiological notion of function. Most likely, if it weren't for the fact that T-terms are pre-theoretically associated with intentional cases, and that the latter involve a special kind of etiology, no one would suggest such a thing.

3.4. *But What about the Biologists?*

Still, one may complain that my dismissive attitude flies in the face of what has been asserted by many biologists. The etiological account does seem to be supported by many of the discipline's most prominent contributors, and they should know what they need, shouldn't they? Then, we could perhaps obtain an indirect argument from authority that has some weight.

Now, while it is true that many biologists have endorsed the view that T-terms can be appropriately used in providing etiological explanations it is less clear that they have thereby endorsed an etiological definition of 'function' along the lines proposed by Wright, Millikan, Neander, and others. The thing is that biologists tend to focus on established traits, when tokens do meet the etiological requirement, and it is in this context some of them have expressed their views on teleological language.

George C. Williams seemingly endorsed an etiological account of T-terms (1996/1966, 9, emphasis is original):

The designation of something as the *means* or *mechanism* for a certain *goal* or *function* or *purpose* will imply that the machinery involved was fashioned by selection for the goal attributed to it. When I do not believe that such a relationship exists I will avoid such terms and use words appropriate to fortuitous relationships such as *cause* and *effect*.

As it stands, the passage clearly suggests that Williams would speak in terms of cause and effect, and not function, when dealing with 1st generation tokens, since these weren't "fashioned by selection" for their effects. However, Williams is not addressing the relation between 1st generation tokens and later ones in the context. He wants to distinguish the effects that matter from the point of view of selection from the effects, however fortuitous, that don't. When he says that a "...benefit can be the result of chance instead of design" (1996/1966, 12) he is not out to recommend anything about what to say about fitter mutants, but to remind us that beneficial effects need not have spread in virtue of providing the benefits. For instance, the feeding activities of earthworms have accumulating long-term benefits for plant growth. Williams criticises W. C. Allee for calling this activity a soil-improvement mechanism because he holds that it can be "...adequately explained on the assumption of design for individual nutrition [for earthworms]." (1996/1966, 18). Given that Williams's focus lies elsewhere we cannot infer from his discussion that he would consider attributing selectionist functions from the 1st generation a problem.

Williams mentions Pittendrigh (1958) as a predecessor as regards the interpretation of T-terms, and both men are listed by Colin Allen (2002) as representing the etiological approach. Pittendrigh's account focuses, not of 'function', but on 'adaptation'. I assume then that Allen takes what Pittendrigh says concerning adaptation to transfer to function as it is the latter that constitutes the subject matter of Allen's account. It is clear that Pittendrigh returns, throughout his paper, to a historical perspective on adaptations. It is trivial that established adaptations will have histories of selection. It is also trivial that adaptations are typically accumulated structures and that they thereby have histories. However, can we really take Pittendrigh to be saying that a novel trait cannot count as a novel adaptation until there has been selection for it, which is the verdict we get if adaptations are essentially etiological? I think not.

Pittendrigh takes there to be a "...close relationship—if not identity—of organization and adaptation..."(1958, 394). Furthermore, he suggests that we understand organisation in terms of information (1958, 395). As for genetic information, Pittendrigh holds that "...novelty is thrust into the inherited message by mutation and recombination..." (1958, 397). It seems plausible to interpret Pittendrigh as saying that novelty of information, which is introduced by mutation and recombination, brings about novelty of organisation, and then due to the tight relation between organisation and adaptation we may thereby have novelty of adaptation at the very first generation.

Thus, he doesn't come out sounding as one who takes adaptations to be essentially historical.

Other biologists, like Simpson (1963) and Lorenz (1963), have spoken of the necessity for biology to pose the question "What for?". In doing this it is evident that they have established traits in mind. The question would seem odd if asked about a novel feature. Thus, they are indeed considering an etiological explanation. Still, neither author seems to conceive of the question such that it requires essentially historical properties. Simpson, who like Pittendrigh is a terminological predecessor mentioned by Williams (1996/1966), takes the answer to "What for?" to be "...in terms of the adaptive usefulness of structures and processes..." (1963, 87), and 'adaptive usefulness' does not require an etiological definition. Lorenz provides the following example (1963, 9):

If we ask 'What does a cat have sharp, curved claws for?' and answer simply by saying, 'To catch mice with', this does not imply a profession of any mythical teleology, but the plain statement that catching mice is the function whose survival value, by the process of natural selection, has bred cats with this particular form of claw.

Lorenz clearly takes the question "What for?" to be dealing with history. Paul Griffiths (1993) takes the quote to illustrate that Lorenz explicitly endorses an etiological interpretation of function statements. I find that reading quite hard to reconcile with the textual evidence. On the page before the quote Lorenz says the following (1963, 8, emphasis is original):

We know that it is the *function* of an organ that alters its form, in the sense of functional improvement; and when, owing to a small, in itself fortuitous, hereditary change, an organ becomes a little better and more efficient, the bearer of this character, and his descendants, will set a standard with which other, less talented members of his species cannot compete; thus in the course of time those less fit to survive will disappear from the earth's surface.

Now, I cannot say that I find the claim about function altering form entirely transparent. However, it appears quite reasonable to take Lorenz to identify

"functional improvement" with "fortuitous, hereditary change", and thus to take him to operate with a notion of function that is not essentially historical.

As long as biologists deal with the interpretation of T-terms in the normal context of discussing established traits, at least two readings of seeming endorsements of etiological definitions are *prima facie* available: 1) They are indeed supporting an essentially etiological definition of 'function'. 2) They are supporting the (tacitly) restricted convention that when established traits are addressed function ascription implies a history of selection. The latter interpretation doesn't rule out the ascription of functions to fitter 1st generation tokens. It is just that those cases will go unmentioned until we move from the (tacit) constraint of discussing established traits of interest to discussing the general case that includes all tokens that biology takes to be type-identical, thus including the 1st generation.

Now, I clearly haven't presented a comprehensive survey of the biological literature as regards the use of T-terms. Nevertheless, the cases I present are ones that have been taken to illustrate that biologists are committed to an etiological interpretation. If that verdict is questionable in these cases then chances are that it is harder than some think to obtain unambiguous evidence for an etiological commitment among biologists. If we really want to find out we can of course poll them on the matter.

If we turn from the question whether biologists actually endorse the etiological notion of function to the question whether they provide good arguments for it, the answer is, as judged by the philosophical debate, negative. It should come as no surprise to those familiar with the debate that supportive arguments stemming from biologists form no part of it.

4. Conclusion.

There is no doubt that we have a pre-theoretical tendency to apply T-terms widely, to non-intentional biological traits as well as to intentional action and artefacts. Furthermore, it is no mystery that the etiological notion of function has been considered promising when it comes to making sense of this practice. However, this notion comes with a *prima facie* awkward distinction within biological categories due to its exclusion of 1st generation tokens. This should make us press the question about the theoretical rationale for it. I have argued that while the notion may seem to be required when we look at prevailing linguistic practice there are no theoretical considerations that support it.

There is nothing about functional categorisation that calls out for an etiological rather than a non-etiological definition of 'function'. It is clear enough that we couldn't have abnormality inclusive categories if we required a high degree of structural similarity within kinds. However, an appeal to function, whether understood etilogically or non-etilogically, does nothing to relate tokens that can display the crucial effect to tokens that cannot. Appeals to effects that account for selection are required to form analogous kinds as well as functional sub-kinds within homologous kinds, but we do not need an essentially etiological notion of function for this purpose. Even if Rosenberg and Neander should be correct in claiming that homology depends on function we have reason to reject invoking essentially etiological functions in that context.

I have also argued that there is no support for Millikan's claim that the etiological notion is needed to press explanatory analogies between biological traits and linguistic devices on the topic of persistence. The introduction of 'proper function' can rather be expected to bias the kind of investigation teleosemanticists have in mind.

Furthermore, the idea that positing etiological functions is required as a manner of explaining the existence of traits by citing effects of such traits is unconvincing. There is no reason why dependence on past effects of type-identical ancestors should be put in a completely different explanatory form than dependence on past effects of non-type-identical ones when it comes to accounting for the existence of current tokens.

Perhaps an indirect argument from authority could be had and have some impact if we saw that the relevant experts endorse the etiological notion. However, cases that have been presented as indicating such endorsement do not seem convincing on closer look.

So, it appears that we are facing a tension between pre-theoretical habit and science. It is true that biologists, like people in general, are in the habit of ascribing teleological terms and expression to biological traits, terms that seem unfit for use about 1st generation tokens. Still, as far as science is concerned, we do not need a "technical definition" that accommodates that particular ring of T-terms. If 'function' is to be defined in terms of selection in a theoretically defensible manner, it will have to be an account on which functions are not essentially etiological.⁴

⁴ I suspect that terms like 'purpose', 'supposed to', and even 'proper', have too much of a biologically unfortunate pre-theoretical (i.e. mentalistic) load for them to be successfully implemented as technical

If the essentially etiological notion of function is theoretically unwarranted then, of course, the enterprises that are heavily predicated on its appropriateness are bound to suffer accordingly.

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