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18 Biological Function Hierarchies and Indeterminacy of Dysfunction: Supplementary Reply to Justin Garson

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A considerable amount of Garson's chapter is taken up with issues concerning indeterminacy of biological function. The issues he raises about indeterminacy are important to address but take us into some general territory that I think is better dealt with separate from his main argument. In this supplementary section on the indeterminacy issue, I first explain why Garson raises this issue as part of his anti–harmful dysfunction analysis (HDA) developmental mismatch argument and then explain why his indeterminacy-related moves fail to save his argument from my reply. I then offer some tentative comments critiquing the solution to indeterminacy challenges that he borrows from Neander (1995). Finally, I reply to indeterminacy-based critiques Garson offers of some of examples I have put forward of dysfunctions. Note that the problem of indeterminacy of biological function tends to be discussed in relation to teleosemantic theories that attempt to derive mental contents from biological functions because in such theories, ambiguities about function can yield problems in determining mental content. However, here I am concerned only with biological functions themselves and ignore whether or how they impact on teleosemantics.

The specific form of indeterminacy Garson considers arises from the fact that selected mechanisms generally possess not just one function but a hierarchical set of functions. As Garson explains, "The problem of indeterminacy … is that there are many ways of describing a trait's function, all of which are allowed by the selected effects theory. Is the function of the heart simply to *beat*? To *circulate blood*? To *bring nutrients to cells*? To *keep the organism alive*? All of these descriptions are acceptable because they are all effects that explain why the heart evolved by natural selection." A key insight is that these functions are organized in a cause-effect hierarchy, in which the performance of one function combines with some expectable environmental circumstances to bring about the performance of another function: "In our example, the different descriptions of the heart form a certain series, that is, a hierarchy defined by cause and effect. By beating, the heart circulates blood. By circulating blood, the heart brings nutrients to cells. By bringing nutrients to cells, the heart keeps us alive. When we say that the function of the heart is simply *to beat*, we are describing the most 'proximal' member of that

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chain. When we say that the function of the heart is to *keep us alive*, we are describing its most 'distal' member. When we say that the function of the heart is to *bring nutrients to cells*, we are describing an 'intermediate' member of that chain."

This multiplicity of functions poses a question about when to say that a feature is dysfunctioning because some of its functions might be performed and others not for varying kinds of reasons. Clearly, the simple failure of a function to be performed is intuitively insufficient for dysfunction. For example, Garson observes that if a stroke prevents blood from flowing through the brain and thus causes the heart to fail in its function of circulating the blood, that is intuitively not a heart dysfunction even though a function of the heart fails to be performed. Similarly, consider the ocean bacterium that has a magnetosome mechanism that orients its motion in such a way as to guide it to the deeper deoxygenated water that it requires for survival (Dretske 1986; Wakefield 1999a). The magnetosome has a hierarchy of functions: it orients the bacterium's motion toward prevailing north in the local magnetic field; by doing so, it orients the bacterium's motion toward the earth's true magnetic north, which is almost always the same as local north under standard oceanic conditions, and by doing so, it orients the bacterium's motion toward deeper water away from deadly surface oxygen. (It works this way only for the Northern Hemisphere bacteria considered here, where the magnetic field's lines are such that magnetic north indicates deeper water; in the Southern Hemisphere, the corresponding bacteria are biologically designed to orient toward south.) Now, in the highly unlikely event that a bacterium happened to live near an enormous rock outcropping that distorted the local magnetic field so that local north was in fact true south, the magnetosome's most proximal function of detecting and orienting motion toward local magnetic north is being performed and so there is no dysfunction there, but its other two functions of orienting motion toward true north and deeper water are failing to be performed, perhaps leading the bacterium to swim dangerously close to the surface. Yet, there is no dysfunction because the failure of the bacterium's functions is entirely due to the unusual nature of its environment, not to anything that goes wrong internally with its magnetosome.

So, there is a question of how and when to translate failures of a feature's various functions into an attribution of dysfunction. Before presenting Garson's solution to this problem, it is worth clarifying: why does Garson raise the hierarchy-of-functions and indeterminacy-of-dysfunction issue to begin with? The answer is that he thinks he needs to address this problem in order to fill a gap in his anti-HDA argument. Garson's central argument is that when a "bona fide" disorder is due to a developmental mismatch, it is a disorder without a dysfunction and thus a counterexample to the HDA. To make this argument work, he needs to establish that there is no dysfunction in a developmental mismatch. He uses his *Daphnia* example to support the intuition that the initial predictive adaptive response in which a developmental choice is made based on sampling the environment during early development is not a dysfunction of

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the developmental mechanism that triggered it. Recall that in the example, a *Daphnia* has a mechanism, *M*, that is triggered during an early critical developmental period by its detection of predators' kairomones in the water of the *Daphnia*'s lake, and if triggered, *M* causes the irreversible development of a "helmet"-like structure that markedly increases the *Daphnia*'s chances for survival in a lake with predators but markedly decreases its survival chances if there are no predators, and it is burdened with this extra structure. So, this naturally selected developmental trigger is based on an implicit "prediction" that if predators are present in the lake during development, they are likely to be present in the same lake during adulthood. However, suppose that after *M* is triggered by detection of kairomones and the helmet develops, the *Daphnia*'s lake subsequently empties of predators and the helmet becomes seriously maladaptive, so there is a developmental mismatch (this is my naturalized version of Garson's example of his intentionally emptying a pool of all its predators after the *Daphnia* develops a helmet). Garson defends his intuition that, no matter what happens later, *M* is not dysfunctional when it triggers the helmet's development because at that point, *M* is doing precisely what it is biologically designed to do: "Here is an intuition that I have. … It seems to me that talk of 'dysfunction' is out of place when it comes to developmental mismatches … a member of *Daphnia* that chose the 'wrong' phenotype … exhibits a developmental mismatch and it takes a fitness loss as a result. In my opinion, this does not represent an inner 'dysfunction.'"

I agree with Garson's intuition that when the *Daphnia* develops a helmet in response to detected predators' kairomones, that is not a dysfunction even if the helmet is later mismatched to the environment and disadvantageous. So far, there is no dysfunction.

Nonetheless, in light of function hierarchies and indeterminacy considerations, Garson realizes that this does not quite give him the premise his anti-HDA argument requires, because he needs to be able to assert that there is *no* dysfunction in a developmental mismatch, whereas so far he can only say the initial triggering of helmet development is not a dysfunction. Garson observes that there are in fact two hierarchically organized functions of *M*, a proximal function to develop a helmet upon detection of kairomones and a distal function to increase the *Daphnia*'s later survival by triggering the helmet's development. In fact, the distal function is not performed when there are later no predators. Garson realizes that whether this failure of distal function allows one to say there is a dysfunction—and thus whether his anti-HDA argument works—depends on how one resolves the indeterminacy of dysfunction challenge:

> My viewpoint about function indeterminacy informs my intuition about the *Daphnia* case. Let's assume … a mechanism (*M*) in the *Daphnia* that instantiates the following rule: "if predators, then helmet; if no predators, then no helmet." There are many ways of describing *M*'s function. We could say that *M*'s function is to trigger a certain developmental sequence (one that yields the helmet head) in response to kairomones … by releasing a certain hormone into the bloodstream. … Alternatively, we could say that *M*'s function is to *protect the individual from*

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> *predators*. The former is a more proximal way of describing its function and the latter a more distal way. … That is because the two descriptions form a series: the mechanism typically *protects the individual from predators* by *releasing hormone H in response to kairomones*.
>
> Now, suppose we have a developmental mismatch. Is there any dysfunction? Described in the most proximal way, the answer is no. *M* fully and adequately discharged its function when it released hormone *H* in response to kairomones. Described in a more "distal" way, there could be a dysfunction. After all, if there are no predators around, *M* certainly cannot perform its function of protecting the individual from them. So, whether or not it is "dysfunctional" depends, in part, on how we describe it.

So, Garson needs a solution to the indeterminacy of function issue that blocks describing the failure of *M*'s distal function of increasing the *Daphnia*'s later survival as a dysfunction. To block such a reply, Garson cleverly adopts Neander's (1995) proposed solution to the indeterminacy of dysfunction problem, which seems tailor-made for his purposes. Neander proposes what I will call the "proximal-function thesis," that one can only attribute dysfunction to failures of the most proximal function in a mechanism's hierarchy. Garson explains, "I endorse a simple and plausible solution developed by Neander (1995). … Her view is that a trait dysfunctions only when it cannot perform its most proximal function. The heart is dysfunctional only when it cannot *beat*." If correct, the proximal-function thesis certainly does block citing the failure of *M*'s distal function of increasing later survival as a dysfunction and thus blocks the most obvious reply to Garson, and so Garson embraces the proximal-function thesis for what potentially can be described as a dysfunction: "I prefer the more proximal description for the reasons I gave above … the way we describe the *Daphnia*-type case is pivotal to my argument." To ensure the result he wants, Garson actually incorporates the proximal-function thesis into the final version of his analysis of the meaning of dysfunction: "the function of a trait is the reason it evolved by natural selection, and *a trait dysfunctions just when it cannot carry out its most proximal function, for constitutional reasons*."

Garson needn't have gone to all this trouble. In my reply above, I did not mount the kind of reply that he anticipated in which one tries to find some distal level of dysfunction of *M* to counter his claimed nondysfunction-disorder counterexample. My response was based not on the claim that developmental mismatches involve distal dysfunctions of developmental mechanisms but rather on the claim that *developmental mismatches are not disorders to begin with*. A careful examination of the Developmental Origins of Health and Disease (DOHaD) texts that Garson cited as support for his approach revealed that those very DOHaD texts maintain that a developmental mismatch is not a disorder. His own sources indicate that, if a condition that is considered a disorder turns out to be a developmental mismatch, then it is not really a disorder. By showing that the disorder ascription is incorrect, I rendered Garson's indeterminacy of dysfunction analysis moot because his point about lack of distal dysfunctions is only

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relevant to his anti-HDA argument in the context of a disorder. My counterargument negated Garson's hypothesis that developmental mismatches can be disorders without dysfunctions and did so in a way that arguments about indeterminacy of dysfunction can't fix.

I now want to examine further the proximal-function thesis and dispute Garson's embrace of the thesis. I will argue that at a minimum, the thesis has explanatory limits and is subject to exceptions; it works for a wide range of examples of a certain kind, but it fails under other conditions.

To prepare the way to some of these counterexamples to the proximal-function thesis, let me first back up for a moment and clarify a point about the definition of "dysfunction." Before he incorporated the proximal-function thesis into the definition of dysfunction, Garson offered a simpler definition that "something is dysfunctional just when it cannot perform its function, for 'inner' or 'constitutional' reasons, rather than because it's in an unsuitable environment." A useful tweak to this definition is to explicitly specify that a feature is dysfunctional only if it is incapable of performing a function *under the conditions for which it was selected to perform that function*. For example, as Christopher Boorse (2002) points out, it is not a dysfunction of your blood-clotting mechanisms if they never actually perform the function of causing your blood to clot, if the reason is that you never are injured. Moreover, it is the internal structure of the clotting mechanisms that prevents them from performing their function of causing clotting when there is no injury; that is part of their design. Nonetheless, there is no dysfunction as long as the clotting mechanisms are capable of performing their function should the appropriate conditions occur for which the clotting capacity was selected, namely, an injury. Or, for example, erectile dysfunction is not the lack of an erection, and it is not the incapability of having an erection under current conditions but the incapability of having an erection when confronted with the standard appropriate environment in which erection is the biologically designed response—say, a sexually desired and responsive partner (for how Masters and Johnson managed to go wrong on this very elementary point, see Wakefield 1988). So, we might say: *a dysfunction is the inability of a mechanism to perform its function for internal reasons even under the appropriate circumstances for which it was selected to perform that function*.

Now, I agree with Garson that the distal failure to perform the function of greater fitness in the case of the *Daphnia*'s helmet's developmental mismatch with its later environment is not a dysfunction, and most such developmental mismatches are not dysfunctions. However, the reason is actually quite simple and can be stated without any need to invoke the proximal-function thesis: to have a dysfunction, a mechanism must be incapable for internal reasons of performing its function under the appropriate circumstances, but the *Daphnia* suffers from no such incapacity and thus no distal dysfunction from developmental mismatch. When the *Daphnia* is grown, although the helmet is highly maladaptive in the actual circumstances in which the *Daphnia* finds

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itself, the helmet is perfectly capable *under the appropriate circumstances* of performing its function of protecting against predators and increasing the *Daphnia*'s survival—namely, the (predicted) environment for which the helmet was selected, a lake with predators. So, there is no reason to attribute a dysfunction, quite independently of any indeterminacy considerations, because the cause of the failure of *M*'s function is in the environment, not in anything internal to *M*. The fact that the distal fitness function of *M* fails to be performed sheerly because of the unsuitable environment lacking predators in which the *Daphnia* finds itself is no more a developmental dysfunction than, say, it is a sexual dysfunction to find oneself in an environment without any potential sexual partners.

Consider the heart example. The heart beats, thereby pumps the blood, and thereby nurtures the cells throughout the body, and here again the distal functions are accomplished by the proximal function in combination with certain environmental conditions. If the environmental conditions do not occur—for example, if there is a blockage of blood vessels that keeps the blood from reaching cells throughout the body—the distal function of nurturing may not be performed, but that is not a heart dysfunction because the failure is due to the environment rather than to something internal to the heart, and in the right environmental circumstances (i.e., should the vessels be unblocked), the heart is still perfectly capable of performing the distal function. So, again, the proximal-function solution is not needed to explain our intuition that the heart does not have a dysfunction in virtue of having a stroke. These examples suggest that for a class of cases, the modified definition of dysfunction has sufficient explanatory power to render the addition of the proximal-function thesis superfluous.

Another problem with the proximal-function thesis is that it is contradicted by a class of cases in which there is a clear dysfunction at the proximal level that by itself and without further environmental vicissitudes makes failures to perform further distal functions inevitable. In such cases, we often feel perfectly comfortable attributing distal dysfunctions as well, contrary to the proximal-function thesis. For example, if the heart ceases beating within normal range, then it is not only a dysfunction that the heart is not capable of beating adequately but also a dysfunction that the heart is failing to propel the blood with vigor through the blood vessels, as well as a dysfunction that the heart is not adequately causing nurturance to reach the cells throughout the body. This is presumably because once the heart has a proximal beating dysfunction, it becomes incapable of performing those distal functions under standard conditions as well.

Similarly, imagine a (Northern Hemisphere) bacterium with a magnetosome that is malformed so that when it detects the local magnetic field, it orients the bacterium's motion toward local south, thus failing in its function of orienting the motion toward local magnetic north. That is surely a dysfunction, and it is just as surely a dysfunction that the magnetosome fails to perform its distal functions of orienting motion toward true north and toward the safe deeper water. This is so because the malformation in

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the magnetosome makes it incapable under standard oceanic conditions of performing any of these functions.

Despite these problems, the proximal-function solution does reflect the reality of a certain kind of case. Generally, if a mechanism performs its most proximal function, it will not have a dysfunction at any level because if it fails to perform a distal function, the failure will be due to an unexpectable environment, thus not a dysfunction. This is because in the causal "by means of" relations that create the hierarchy of functions, the most common situation is that each "by means of" link involves just two factors: the previous function being fulfilled and the presence of standard environmental circumstances. If the prior function is performed, it is only the expectable environmental facts that are left to go wrong and cause a failure, but that means that under the standard circumstances, the mechanism would be capable of performing the distal function, so there is no dysfunction. Thus, the proximal-function thesis reflects the reality that generally speaking, when there is no proximal dysfunction, there will be no distal dysfunction.

However, I now want to claim that there are cases that falsify the proximal-function thesis even when the proximal function is successfully performed. I believe that Garson pointed the way to understanding how this can occur by his emphasis on predictive adaptive responses (PARs) and developmental mismatches, for such cases do have properties that open the way to a persuasive domain of counterexamples to the proximal-function thesis. We saw above that DOHaD theorists have the concept of a developmental disruption, in which the input to a developmental mechanism is outside of the range for which it biologically designed to be adaptively responsive. Disruption is at the core of my hypothesis.

My hypothesis is the following: when there is irreversible developmental disruption in the performance of a proximal function that makes it impossible for internal mechanisms to accomplish downstream distal functions even under appropriate environmental circumstances, those failures of distal functions can be said to truly be dysfunctions. First, developmental disruption can be thought of as an input to developmental mechanisms that is outside of the range for which the developmental mechanisms were biologically designed, meaning roughly that it is outside the range of inputs that exerted the selective pressures that led to the mechanism's selection. If such a disruption in some developmental process occurs during a critical developmental window, it may permanently and irreversibly alter the subsequent trajectory of development in a way that was never selected for. This notion surely has many ambiguities and obscurities that warrant examination, but developmental disruption is a notion that is relied on throughout developmental theory and, as we saw, is amply discussed in the DOHaD literature, so I accept it as a working concept of adequate credentials for the sake of this analysis. Should such a disruption occur that permanently alters the mechanism's responses, at that point there may be internal reasons why the mechanism is incapable

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of performing subsequent distal functions even in the appropriate environment and thus dysfunctional. None of the examples used by Garson satisfy these criteria, and that is why it seems to him that mechanisms can never have distal dysfunctions, but in fact that appearance is due to an accidental feature of the examples rather than a correct general principle. The best way to test my hypothesis that true distal dysfunctions due to irreversible developmental disruption falsify the proximal-function thesis is by examples, to which I now turn.

Consider a somewhat different kind of magnetosome example than those above, more along the lines of Garson's PARs and developmental-mismatch focus. Consider a cousin species of the above bacterium species that evolved so that, rather than the magnetosome continually orienting the bacterium's movement, the magnetosome samples the environment once during an early critical developmental period and, based on its detection of local magnetic north during that period, permanently and irreversibly fixes the bacterium's direction of motion toward local north. There is still a hierarchy of functions during the critical period: the magnetosome performs the function of permanently and irreversibly fixing motion toward local north and, by doing so, performs the function of permanently and irreversibly fixing motion toward true north and, by doing so, performs the function of permanently and irreversibly fixing motion away from surface water. (This is not wholly implausible given that these three functions almost always coincide and this bacterium saves considerable energy costs of continual magnetosome sampling of the environment.)

Now, suppose that this bacterium happens to be near the magnetic rock outcropping described earlier at the very time of the magnetosome's crucial developmental window in which the bacterium's orientation of motion becomes permanently and irreversibly fixed. The magnetosome perfectly performs its proximal function of detecting local magnetic north and accordingly fixing motion. However, in doing so, it fails to perform its distal functions of fixing motion toward true north and away from the surface. But this time, there is a crucial difference from the earlier bacterium that happened to be near the rock outcropping. If the other bacterium just moved away from the rock outcropping that was distorting the magnetic field, in the standard environment, it would be fine. In contrast, the present bacterium, once its critical period has occurred, has a fixed internal structure such that even if it is placed away from the outcropping and into a standard oceanic magnetic environment—the appropriate environment for which its mechanisms were naturally selected—the bacterium will still swim relentlessly toward the surface and its death.

My intuition is that there is now something wrong with this bacterium and that there is a clear dysfunction of the magnetosome. That dysfunction of the magnetosome is not a failure of its proximal function, which is to direct the bacterium toward whatever was local north at the time of the critical period. The dysfunction is due to the failure of the magnetosome's distal functions of directing the bacterium to true

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north and away from the surface, which it is now incapable of doing even under ideal conditions due to the internal fixation of its motion orientation. This example of a case with no proximal dysfunction but clear distal dysfunction falsifies the proximal-function thesis. Developmental plasticity, especially critical periods of the sort studied by DOHaD, change the nature of the dysfunction picture quite dramatically in a way that leads to conclusions in conflict with Neander's analysis.

The above analysis in which a developmental disruption during a critical developmental window causes a distal dysfunction also explains a second example, which is an example of mine that Garson criticized as a violation of the proximal-function thesis. Richters and Hinshaw (1999) present an example of an abused child who gets a fixed idea about the threat posed by the environment and responds aggressively and so becomes conduct disordered. This can be construed as an early developmental mismatch type example, based to some degree on the findings of Dodge (1990) that internalized cognitions of threat seem to be the vehicle by which abuse is carried from childhood to adulthood. Richters and Hinshaw's point, parallel to Garson's *Daphnia* argument, was that this youth has a disorder—a "bona fide" disorder of conduct disorder, one might say—but there is no dysfunction because the child's learning was a calibrated response to its early environment, and thus this is a counterexample to the HDA. Garson notes that I provided two possible answers to this proposed counterexample. One possibility is that the described processes are within the expectable range that shaped the natural selection of personality traits and thus part of normal learning and personality formation, and then there is no dysfunction and no disorder even though the outcome is socially undesirable. Indeed, *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*) backs me up on the claim that even where the conduct disorder symptoms are present, there is not necessarily a disorder if normal learning or normal personality formation took place in a high-threat general environment: "Conduct disorder diagnosis may at times be potentially misapplied to individuals in settings where patterns of disruptive behavior are viewed as near-normative (e.g., in very threatening, high-crime areas or war zones). Therefore, the context in which the undesirable behaviors have occurred should be considered" (American Psychiatric Association 2013, 474).

The other possibility is that the familial abuse took the early environment outside of the expectable range and, in DOHaD language, was a developmental disruption. Familial abuse is plausibly an evolutionarily unexpectable environment that interferes with the evolutionary point of the developmental mechanism, which is to make a reliable PAR based on detection of the likely level of threat in the general environment. In this case, given the disruption and irreversible fixation of threat sensitivity outside of the selected range, there is a developmental dysfunction and so the subsequent traits are a disorder.

My either/or response to Richters and Hinshaw was in fact strictly in keeping with the later DOHaD view documented above. We saw that the DOHaD literature admitted that it is often difficult to tell whether an early environmental input is within the range

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for which responses have been naturally selected or is a disruption. This is especially the case when there is a continuous variable (such as threat sensitivity) that at some extremes is no longer within the evolution-relevant range.

Another example of mine that Garson disputes on the basis of Neander's proximal-function thesis is my gosling example. I argue that a gosling who accidentally imprints on a passing fox (or, in earlier renditions, a porcupine) has both a dysfunction and a disorder. In this example, the claimed dysfunction is the failure not of the most proximal function, which is presumably imprinting on whatever creature is first observed upon hatching. I agree with Garson's comment that if the gosling did not imprint on the fox, that would be a dysfunction. However, this is not an either/or situation; each function's status must be evaluated on its merits. I think in this case there is a failure and dysfunction of a distal function, namely, imprinting on the mother. (Interestingly, as I argue elsewhere, there is nothing wrong with the gosling at the brain-descriptive level, for the problem is a matter not of brain functioning per se but of the reference or meaning of the image stored in its brain, a psychological-level mental-content construct, so that we have here an example of a psychological dysfunction that is not a brain dysfunction.)

Here is Garson's full critique of my gosling example:

> There are cases where I think Wakefield is potentially inconsistent in his approach to function indeterminacy, and the way we describe the *Daphnia*-type case is pivotal to my argument. On the one hand, Wakefield's (1999a, 386) explicit comments about indeterminacy seem to agree with my own, namely, that we should prefer the most "proximal" description of an item's function (as in the bacterium case). On the other hand, some of his specific examples seem to run contrary to that point. He discusses an example of filial imprinting gone awry, that is, where a gosling imprints on a passing porcupine (Wakefield 1999b, 468; 2000, 263). (Imprinting refers to a developmental "window" of time in which a juvenile organism forms a strong, lifelong preference. The function of filial imprinting in goslings is to cause them to form an attachment to their own mothers. The mechanism by which this works is that they form an attachment to the first large, suitably moving object that they encounter. Imprinting goes awry when the mechanism causes a gosling to imprint on an object that is not its mother.) The gosling now has an enduring inner disposition to follow around a porcupine. Wakefield says that this disposition is a dysfunction. I do not consider it a dysfunction (Murphy and Woolfolk [2000b, 279] have similar reservations about Wakefield's imprinting case). I think there would be a dysfunction if the gosling failed to imprint on a passing porcupine, so long as that porcupine moved about in the right sort of way and if that porcupine entered the gosling's visual field at just the right time. The gosling's disposition would be chronically maladaptive but not dysfunctional.
>
> I suspect that the difference of opinion between Wakefield and myself traces back to the problem of function indeterminacy. For there are two ways of describing the function of the imprinting mechanism in the gosling's brain, and one is more proximal than the next. The first, most proximal description is to say *M*'s function is to cause the gosling to form a strong

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attachment to the first large, suitably moving object it sees. The second, more distal description is to say *M*'s function is to cause the gosling to have a disposition to follow its mother. The first is more proximal than the second because the mechanism typically causes the gosling to follow its mother *by* causing the gosling to form a strong attachment to the first large, suitably moving object it sees. If we stick with the first description in the porcupine case, we see there is no dysfunction. *M* has performed its job admirably. If we stick to the second description, we have some evidence of dysfunction (after all, *M* cannot discharge its function). I have given reasons for my preference for the more proximal description.

It will be seen from the above passages that Garson rests his case against my gosling example completely on his prior acceptance of the proximal-function thesis. However, we have seen that that thesis cannot be given full confidence to reflect our intuitions in various kinds of cases. In fact, the structure of the example fits my "developmental disruption" schema, and the example seems to succeed in illustrating my point and falsifying the proximal-function thesis.

Garson's commentary misses the crucial fact that the imprinting process permanently and irreversibly locates an image of the target of the imprinting in the brain, and so once that occurs, the subsequent failures of function are due not to events in the environment but to the internal state of the gosling. Once the imprinting on the fox takes place, the gosling is incapable even under ideal circumstances—for example, in the presence of its mother—of performing basic functions that are developmentally essential and for which the mother alone is primed to provide a complementary interactor.

In a true developmental disruption, the subsequent trajectory is influenced in ways that are not consistent with selective pressures. This is what happens in the developmental sequence that befalls the gosling. The gosling following its mother triggers many other developmental programs and expectable inputs as the gosling watches its mother hunt for and share food, warm and shelter the gosling with her feathers, protect the gosling from predators, and help the gosling learn and recognize species-specific behaviors and vocalizations, as well as recognize conspecifics so that the gosling can eventually select an appropriate mate. Of course, all this could fail due to a deviant environment where the mother is not available. However, in the misimprinting example, it is not the environment that makes all these later developmental performances impossible. Rather, it is an internal structure in the gosling that makes the gosling incapable of responding to these various stimuli, namely, the permanent and irreversible developmental disruption that occurred when, in the critical window for imprinting, the gosling imprinted on a passing fox rather than its mother. My intuition is that this is a clear dysfunction of the imprinting process despite not being the most proximal function.

Lastly, while my hypothesis does not depend on it, it would be of interest if the above analysis predicted a way that our intuitions about dysfunction in Garson's own *Daphnia* example might be flipped. Here is a possibility: imagine that in a lake without

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predators, there is pesticide runoff pollution, and it happens that the pollutants have the same chemical signature used by the *Daphnia* to detect the presence of the predators' kairomones. (Note that in actuality, contrary to Garson's characterization, it is not detection of the kairomones that is the most proximal function of the *Daphnia*'s mechanism *M* but detection of the chemical signature by means of which the *Daphnia* detects the kairomones.) So, the most proximal function—detecting a certain chemical signature—is successfully performed. Yet, these *Daphnia* then develop helmets despite the lack of predators during the developmental period and are then severely disadvantaged by the helmets in surviving in the predator-free lake in adulthood. My intuition is that in this case, something has gone wrong and the mechanism *M* suffers a dysfunction in mistaking the pollutant for kairomone. In this case, a distal function—detecting kairomone—fails to be performed and is a dysfunction.

I conclude, first, that Garson's and Neander's proximal-function hypothesis does not fit common intuitions about dysfunction. And, second, the examples I have used in which there are distal dysfunctions despite successful proximal functions, despite being inconsistent with the proximal-function thesis, are entirely in keeping with common intuitions given the special situations of irreversible disruption on which they are based. It is the proximal-function thesis and not my examples that needs fixing.

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Defining Mental Disorder

Jerome Wakefield and His Critics

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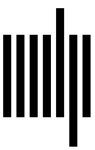
DOI: 10.7551/mitpress/9949.001.0001
ISBN (electronic): 9780262362931
Publisher: The MIT Press
Published: 2021

The open access edition of this book was made possible by generous funding and support from Arcadia – a charitable fund of Lisbet Rausing and Peter Baldwin



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The open access edition of this book was made possible by generous funding from Arcadia—a charitable fund of Lisbet Rausing and Peter Baldwin.

This book was set in Stone Serif and Stone Sans by Westchester Publishing Services.

Library of Congress Cataloging-in-Publication Data

Names: Faucher, Luc, 1963– editor. | Forest, Denis, editor.
Title: Defining mental disorder : Jerome Wakefield and his critics / edited by Luc Faucher and Denis Forest.
Description: Cambridge, Massachusetts : The MIT Press, [2021] | Series: Philosophical psychopathology | Includes bibliographical references and index.
Identifiers: LCCN 2020016671 | ISBN 9780262045643 (hardcover)
Subjects: LCSH: Wakefield, Jerome C. | Psychiatry--Philosophy. | Mental illness--Philosophy. | Mental illness--Diagnosis. | Mental illness--Classification.
Classification: LCC RC437.5 .D434 2021 | DDC 616.89--dc23
LC record available at https://lccn.loc.gov/2020016671

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