The Functional Sense of Mechanism

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This article presents a distinct sense of 'mechanism', which I call the functional sense of mechanism. According to this sense, mechanisms serve functions, and this fact places substantive restrictions on the kinds of system activities 'for which' there can be a mechanism. On this view, there are no mechanisms for pathology; pathologies result from disrupting mechanisms for functions. Second, on this sense, natural selection is probably not a mechanism for evolution because it does not serve a function. After distinguishing this sense from similar explications of 'mechanism', I argue that it is ubiquitous in biology and has valuable epistemic benefits.

1. Introduction. The retina is a mechanism for transducing light into electrical impulses. The brain's medulla is a mechanism for triggering the gag reflex, among other vital activities. Male dragonflies use their cerci, appendages protruding from their anuses, as part of a mechanism for clinging to females during copulation. The electrosensors that line the goblin shark's snout are part of a mechanism for detecting prey; this mechanism, in turn, is a part of a larger mechanism for capturing prey.

One feature these mechanisms possess is that they are structurally and dynamically complex. Each has a multitude of parts that do different things, and these parts interact in fairly reliable ways to carry out various system-level activities. These mechanisms are often hierarchically nested, or chained together in a series. This observation has stimulated much of the 'new mech-

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anism' literature and is the cornerstone of various explications of 'mechanism' that this literature has produced (e.g., Bechtel and Richardson 1993; Glennan 1996, 2005; Machamer, Darden, and Craver 2000; Craver 2001; Darden 2006).

A second feature these mechanisms share is that the activities they perform are not just any old system activities. These activities are their *functions*. The dolphin's fin can act as a hook that entangles the dolphin in nets. But it is not usually described as a mechanism for doing so. That is because getting entangled in nets is not its function. Myasthenia gravis is an autoimmune disorder that leads to paralysis by disrupting acetylcholine receptors embedded in the muscle. But acetylcholine receptors are not mechanisms for paralysis. They are mechanisms for muscle contraction; paralysis results when the mechanism is prevented from doing its job.

I use this observation to explicate an alternate conception of mechanism that I believe to be widespread in biology, biomedicine, and psychology. This is the *functional* sense of mechanism. (This is not an attempt to reject or 'refute' alternate senses of 'mechanism' that have been developed in the new mechanism literature.) According to the concept of mechanism that I would like to explicate, mechanisms serve functions. Moreover, that mechanisms serve functions places substantive restrictions on the kinds of activities 'for which' there can be a mechanism. Although the heart is a 'mechanism for' circulating blood—or it is part of such a mechanism—it is not a 'mechanism for' heart disease. Heart disease is something that happens when this mechanism is disrupted.

After explicating the functional sense of mechanism, I will make two points about it: that it is *ubiquitous* and that it is *useful*. First, the functional sense of mechanism is ubiquitous in biology, biomedicine, and psychology, although I will mainly focus on biology and biomedicine. In other words, when biologists, biomedical researchers, and psychologists talk about 'mechanisms' for this or that, they are commonly using 'mechanism' in the functional sense.

The second point is that the functional sense of mechanism is useful. It yields valuable epistemic benefits for researchers. That is because, as I argue, it maximizes the inferential coherence of biology and biomedical research. Thinking about pathologies as the result of broken mechanisms, rather than as 'having' their own mechanisms, helps researchers integrate information about the etiology of disease with information about function, in such a way as to enhance the explanatory and predictive power of biomedicine.

This is to endorse neither a theoretical monism about mechanism nor a theoretical monism about function. I accept a modest pluralism with respect to both. First, although the functional sense of mechanism implies that mechanisms serve functions, it does not specify which theory or theo-

ries of function are right. My view is that many theories of function are consistent with the functional sense of mechanism, such as those that appeal to past selection, current contributions to fitness, or design, as in artifacts (see Garson [2008] for an overview). In other words, my view holds that mechanisms serve functions but allows some latitude as to which concept of function best suits our needs. In this context, I am not endorsing a selected-effects view over a forward-looking view that would define 'function' in terms of current contributions to fitness or vice versa.

This latitude, however, is not infinitely permissive. The sense of mechanism I wish to explicate is not consistent with the causal role (CR) theory of function or its more recent variants, according to which the function of a system's part consists merely in its contribution, in tandem with the system's other parts, to some phenomenon that a research community has taken an interest in (e.g., Cummins 1975; Hardcastle 1999, 2002; Craver 2001, 2013; Davies 2001). This is because, as will be amplified below, the CR theory places few, if any, substantive restrictions on the kinds of top-level system activities that can count as functions and hence few, if any, restrictions on the kinds of activities 'for which' there can be a mechanism. If a research community were interested in the pathology of myasthenia gravis, it would be entitled, on this view, to say that causing muscle paralysis is a function of the acetylcholine receptor and, therefore, that the receptor is a mechanism for doing so. Conjoining the functional sense of mechanism with some version of the CR theory is to effectively renounce the epistemic benefits of the functional sense of mechanism. Better to conjoin the functional sense of mechanism with a theory of function that places substantive restrictions on the kinds of top-level system activities that can count as that system's functions.

I also endorse a modest pluralism with respect to mechanism. The term 'mechanism' is ambiguous. Scientists do not always use it in the same way. As Moss (2012, 165) points out, sometimes 'mechanism' is used synonymously with 'physical explanation'; in this sense, 'finding a mechanism' means nothing more than discovering an intermediate causal link between a supposed cause and a more or less distal effect. This is a fairly tepid sense of mechanism that Thagard seems to invoke in his book on biomedical discovery (e.g., Thagard [1999], 122, especially his model for the mechanism of scurvy). That is what people have in mind when they say things like, "I don't believe that intercessory prayer works, because I can't imagine a mechanism for it." This is certainly what Galileo had in mind when he rejected the causal influence of the moon on the tides. My claim is merely that the functional sense of mechanism is prominent in biology and it delivers valuable epistemic benefits.

The article has five sections. In section 2, I explain the functional sense of mechanism and situate it in relation to a tradition of similar usage. In sec-

tion 3, I explain how this notion of mechanism is ubiquitous in biology. This centers around the observation that 'mechanism', as commonly used, is normative—mechanisms are the sorts of things that can break—and the normativity of mechanism is best explained by the normativity of function. In section 4, I explain why this particular construal of mechanism is useful for biology and biomedical research. In section 5, I respond to two objections. The first is that the functional sense of mechanism is actually at odds with biological usage, and the second is that it does not deliver the benefits I claim for it (or not more than other ways of thinking about mechanism).

2. The Functional Sense of Mechanism. There are two premises that characterize the functional sense of mechanism. This is not intended as a conceptual analysis, replete with necessary and sufficient conditions. The first is a necessary condition on this sense, and the second is an adequacy condition on the relevant concept of function with which the first premise can be conjoined. One might, for example, add further restrictions on the functional sense of mechanism by placing various constraints on the kinds of spatial, temporal, or organizational features that such 'mechanisms' must exhibit. This would bring the functional sense of mechanism closer to the sense at issue in the new mechanism tradition.

The first premise is that mechanisms serve functions. More precisely, where X is a system and Y is an activity of X, X is a (part of a) mechanism for Y, only if X has the function Y. The heart is a part of a mechanism for circulating blood, only if it has the function of circulating blood. The superchiasmatic nucleus is part of a mechanism for regulating circadian rhythm, only if it has the function of regulating circadian rhythm. This premise, taken alone, should be relatively uncontroversial. After all, for some theorists, 'function' means little more than a conventionally selected activity of a system. If so, the premise would border on tautology.

The second is a type of adequacy condition on the relevant sense of function with which the first premise can be conjoined. It states that the fact that mechanisms serve functions places substantive constraints on the kinds of system activities 'for which' there can be a mechanism. For example, on this view, there are no mechanisms for pathology. There is no mechanism for heart disease or Alzheimer's disease or schizophrenia because (for example) heart disease on the part of a system is not a function of that system. Rather, pathologies typically result from disrupted mechanisms. Something like heart disease, on this view, represents an explicable consequence of a broken mechanism for blood circulation.

Of course, one might hold that some things that are typically (and perhaps mistakenly) classified as 'pathological' actually do perform functions. Moreover, a certain biological phenomenon might be 'functional' with respect to one system and 'pathological' with respect to another. For example,

cancer researchers have recently elucidated a 'mechanism of therapy resistance' on the part of some tumors (see Landsberg et al. 2012). Some tumors can change their protein expression patterns in the course of immune therapy and thereby increase resistance to such therapies. This might be thought of as a counterexample to my view since it appears to be a mechanism for pathology, but it is not: mechanisms of immune therapy resistance have a function for the tumor and not for the organism. The same kind of dynamic may characterize some host-parasite interactions.[1]

In addition to the fact that there are no mechanisms for pathology, natural selection is probably not a mechanism for evolution, in the functional sense. This is because it does not serve a function, on any well-developed theory of function that places substantive constraints on the kinds of activities that can count as a system's functions. For example, on the selected-effects theory, something has a function only if it was selected for by a selection process. Natural selection itself, however, is not 'selected for'.[2] On a Boorse-style goal-contribution theory, the function of a trait consists in its statistically typical contribution to the goal of a biological system in which it is contained (that is, of which it is a component). Although natural selection can promote the existence of such goal-directed biological systems, it is not in any obvious sense a 'component' within a biological system.

Before I go on to describe why the functional sense of mechanism is ubiquitous and useful, I outline a tradition of similar usage, and I distinguish my view from other, related, views about the relation between mechanism and function. Perhaps the first person to explicitly use the term 'mechanism' in this sense was G. C. Williams (1966) in his celebrated and contentious book *Adaptation and Natural Selection*. As is well known, Williams defines 'function' in terms of selected effects, thus prefiguring much of the philosophical functions literature of the 1970s and beyond. As is less well known,

1. As one anonymous reviewer suggested, this conception of function and mechanism can be incorporated into a hierarchical context. Buss (1987), e.g., argued that the evolution of multicellularity involved mechanisms for inhibiting subversion on the part of individual cells. Here, the mechanism for inhibiting subversion has a function relative to, or 'for', the multicellular individual, and it performs that function by preventing the cell (or cellular components) from performing certain of its functions. However, each cell has mechanisms that have the function of contributing to its own replication, even if doing so entails subverting a mechanism on the part of the multicellular organism. Hence, when we characterize mechanisms by the functions they serve, we have to be careful in each case to specify precisely the entity for which the function is performed.

2. One could hold that natural selection in the evolutionary sense was not selected for but that it gave rise to other kinds of 'selection processes' broadly construed, such as antibody selection or operant conditioning (see Garson 2011, 2012). There is no contradiction in assuming that some selection processes were selected for by natural selection because of some fitness-related benefit they delivered and, hence, that some selection processes have functions.

however, he defines 'mechanism' in terms of function: "The designation of something as a *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" (9).

Moreover, this passage cannot be dismissed as an afterthought since Williams relies on this stipulation with admirable consistency. For example, his short book is punctuated by rhetorical questions about the use of 'mechanism', such as these: "Should we therefore regard the paws of a fox as a mechanism for constructing paths through snow? Clearly we should not"; "Should we therefore call the causal activities of the earthworm a soil-improvement mechanism?" (1966, 13, 18). Williams thinks there is something deeply counterintuitive about such usage. Williams's usage has also been adopted in large measure by evolutionary psychologists (e.g., Buss 2008, 69; also see Tooby and Cosmides 2006, 185).

Some philosophers of biology have also indicated a special connection between mechanism and function and, in particular, that judgments about mechanism presuppose judgments about function.[3] Two recent developments are by Craver (2001, 2013; also see Piccinini and Craver 2011) and Moss (2012). Although I disagree with the precise way each construes this relation, I agree with the general proposition that judgments about mechanism presuppose judgments about function. I briefly sketch their views in order to clarify the content of my own.

Craver (2013) holds that the age-old opposition between 'mechanism' and 'teleology' is fictitious. Instead, he proposes that judgments about mechanism presuppose judgments about function. This is because whether a system counts as a 'mechanism for' something depends on which capacity is selected as the 'end', 'end-state', or 'function' of that system: "This teleological feature of mechanistic description is also implicit in the fact that mechanisms such as the NMDA receptor are bounded: a judgment has been made about which entities, activities, and organizational features are in the mechanisms and which are not" (140). In short, in order to carry out a mechanistic analysis of a system, we have to make a decision about what counts as the system's functions (also see Craver 2001).

One logical implication of his view is that if a certain activity is not a function of any system, there cannot be a mechanism for it. Consequently, Craver would seem to be committed to the claim, which I hold, that there are no mechanisms for pathology since pathologies are almost universally held to be dysfunctional or nonfunctional states of a system. However, Craver argues that there are 'mechanisms for' pathologies such as heart disease or

3. Piccinini gives a particularly clear statement of this dependency relation: "Different notions of mechanism may be generated by employing different notions of function" (2010, 286).

drug addiction and that much of biomedical research is devoted to discovering them (e.g., Craver 2001, 67).

This apparent contradiction can be resolved by describing the concept of function he subscribes to. Over the last decade, Craver (2001, 2013) has developed a version of the CR theory of function that he calls the 'mechanistic causal role' view. In this view, the function of a system, considered as a whole, is relative to the research community that investigates it. The function of each part of the system consists in the contribution that it makes, in tandem with the other parts, to yielding the function of the system as a whole. A central pillar of his view, that the analytical decomposition of a system and the subsequent identification of part functions relies on adopting a conventional standpoint or perspective regarding "what the system is doing," is based on the work of Kauffman (1970, 259), Wimsatt (1972, 69–72), and Cummins (1975). In fact, this twofold characterization of function is also endorsed with little ado in one of the major works of the new mechanism tradition (Machamer et al. 2000, 6). Furthermore, the CR theory seems to be the default theory accepted by many proponents of the new mechanism tradition to the extent that that tradition engages with the literature on function (see, e.g., Machamer et al. 2000, 6; Craver 2001, 2013; Glennan 2002, 127 n. 6; 2005, 456).[4]

One consequence of his view is that, although mechanisms serve functions, this fact does not impose any substantive restrictions on the kinds of top-level biological activities 'for which' there can be a mechanism.[5] (Of course, once a research community has assigned a function to the system in toto, there are substantive constraints on the functions that can be assigned to the components of the system.) This conventionalist aspect of the CR theory lets Craver recognize 'mechanisms for' pathologies such as drug addiction or Alzheimer's disease. This is because the only context in which a researcher would talk of a 'mechanism for' drug addiction is a context in which drug addiction is the system's conventionally chosen function. For this reason, Craver describes his view of function as 'perspectivalism' (2013; also see Craver 2001, 71).

4. Also see Bechtel and Richardson (1993, 17), where the 'function' of a part is characterized in terms of its CR—i.e., its contribution, in tandem with the other parts, to the 'behavior' of the system as a whole. Glennan (2005, 456) seems to use 'function' and 'causal role' more or less interchangeably.

5. There may be some constraints that stem from the fact that, in order for a system's activity to be the object of a functional explanation, the system must have the right kind of internal complexity, the system-level activity must be sufficiently distinct in kind from the activities of its parts, etc. (see, e.g., Cummins 1975, 764; also Davies 2001, 79). However, although these constraints might explain why, e.g., a dropped vase does not have the function of falling to the ground, they will not explain why the neural tube does not have the function of producing anencephaly, which is what I am more concerned to rule out.

As a consequence, although I label my view 'the functional sense of mechanism', this does not mean that other explications of mechanism make no assumptions about function. The difference between my view and (for example) Craver's is that the sense of function I appeal to places substantive restrictions on the kinds of systems that can be mechanisms. The label, then, merely signals that I bring the concept of function to the foreground and give it additional work to do in identifying mechanisms.[6]

Moss (2012, 165) also explores the concept of mechanism. In his view, there are at least two main senses of 'mechanism', a strong and a weak sense.[7] According to the weak sense of mechanism, which I call mechanism as *physical explanation*, to claim there is a 'mechanism' for a phenomenon is simply to claim there is a physical explanation for it, and to ask for this mechanism is simply to request such an explanation. According to the strong sense of mechanism, a mechanism for a biological phenomenon refers to the goals, ends, or purposes of the biological system in question. As Moss puts it, "the very idea of a mechanism in biology begins with a holistic pre-conception of a living system as a functional end-in-itself that sustains itself through functional-physical means" (166). When we construe a part of a system as being a 'mechanism' for something, we tacitly (or explicitly) conceive of the system itself as having goals or ends, and we suppose that the mechanism is somehow relevant to the attainment of those goals.

On a superficial reading of Moss, one might think that he accepts that mechanisms serve functions; that is, he seems to suggest that something counts as a mechanism only if it promotes the system's goals, which, according to one prominent tradition of thinking about functions, would imply that something counts as a mechanism only if it serves a function. However, Moss does not think that mechanisms must serve functions. Rather, he accepts a weaker claim, namely, that mechanisms are in some sense relevant to the goals of the organism, even if they do not support those goals. On this construal, Moss could accept that there are mechanisms for pathologies. The reason one can talk about a mechanism for (for example) diabetes is because, on my understanding, diabetes is a pathology that makes a difference to the goals of a biological system, namely, the goal of self-sustenance.

One reason that Moss rejects the claim that mechanisms serve functions is that, like Craver, he believes it to be a basic datum of biological usage that biologists talk about 'mechanisms for' pathology. Any view that disallows this would be prima facie inconsistent with the way biologists talk. I come back to this point in section 5, where I suggest that although biologists do use the locution 'mechanism for pathology', this is often elliptical.

6. I thank an anonymous reviewer for prompting me to clarify this point.

7. Moss indicates a third sense that I will not discuss here, as it seems to me more an analysis of the cognitive psychology of the use of 'mechanism' rather than an additional sense of the term.

3. The Ubiquity of Functional Mechanisms. The functional sense of mechanism is ubiquitous in biology, biomedicine, and psychology. I think it is implicit in the way that scientists often talk about mechanisms. One indication of this is that 'mechanism', as commonly used, has a normative dimension, and this normativity is best explained by the normativity of function. To say that mechanisms are normative simply means it is possible for a token system, *X*, to be a mechanism for an activity, *Y*, despite the fact that *X* cannot perform *Y*.[8] The toilet in my home is a mechanism for disposing of human waste products, despite the fact that, due to a corroded plug, it cannot do that. In other words, to say that mechanisms are normative is to say that they are the sorts of things that can break.

The idea that mechanisms can break is pervasive in biology. Biologists and biomedical researchers have a rich and colorful lexicon to describe the ways that mechanisms can break. A mechanism can 'breakdown'; it can be 'usurped', 'co-opted', or 'hijacked' by another mechanism or biological process; it can be 'interfered with', 'impaired', 'disrupted', or 'disabled'; it can 'fail to function'. A handful of citations exemplify this usage; one need not look hard to find them: "drugs of abuse can *hijack* synaptic plasticity mechanisms in key brain circuits" (Kauer and Malenka 2007, 844; emphasis mine). "Only by understanding these core synaptic mechanisms can we hope to understand how drugs of abuse *usurp* or *modify* them" (845; emphasis mine). "It is argued here that potentially irreversible *impairments* of synaptic memory mechanisms in these brain regions are likely to precede neurodegenerative changes that are characteristic of clinical [Alzheimer's disease]" (Rowan et al. 2003, 821; emphasis mine). "However, it is possible that a *disruption* of synaptic plasticity-related mechanisms by soluble Aβ also contributes to clinical symptoms" (826; emphasis mine). Additionally, philosophers of the new mechanism tradition have recognized the fact that mechanisms can break. Some of its proponents have described this fact as being significant for understanding causation, identifying the components of mechanisms, and treating disease (e.g., Bechtel and Richardson 1993, 19; Craver 2001, 72; Glennan 2005, 448; Darden 2006, 259).

In my view, the normativity of function explains the normativity of mechanism. To say a mechanism is broken entails that it cannot perform its function. Being unable to perform a function is only necessary for a mechanism's being broken, but it is not sufficient: typically, to say that a system is broken also implies that it cannot perform its function for 'constitutional' reasons and not just because it is in an unsuitable environment (e.g., Dretske 1986). In some sense, an unplugged blender 'cannot' perform its function, but that does not mean it is broken. Glennan (2005), in a discussion of broken

8. See Neander (2008, 385–86), for a careful explication of the sense in which functions are 'normative'. In particular, 'normativity' in this sense has nothing to do with ethics, values, or personal preferences.

mechanisms, seems to accept the connection between a mechanism's being broken and its having a function: "the concept of a mechanism's behavior generally presupposes a concept of normal functioning. When one describes the behavior of a mechanism, one describes how it will behave if it is not broken" (448). However, he asserts that such functions need not involve selection or design (449).

Not only does the normativity of function explain the normativity of mechanism; it is a very good explanation for it. This is because it exhibits conceptual economy. Philosophers have a clear understanding of how functions can be normative. As a consequence, philosophers have availed themselves of the concept of function in explaining the normativity of other biological categories, such as the normativity of biological information and the normativity of biological trait classification (e.g., Dretske 1986; Neander 1991; Rosenberg and Neander 2009). This is neither to endorse these approaches nor to say that these attempts are free from controversy (e.g., Griffiths 2006). However, the fact that appeals to the normativity of function are plausible and compelling in other contexts suggests that it is a reasonable approach here, too.

4. The Utility of Functional Mechanisms. The functional sense of mechanism is not only ubiquitous but useful. In other words, biologists should construe mechanism in terms of function. This is because doing so maximizes the inferential coherence of biology and biomedicine. Using the functional sense of mechanism makes for better biology because it makes for better generalizations.

This argument proceeds in two steps. First, there are many more states of an organ or organ system compatible with disease than with health. As Neander puts the point, in a twist on Tolstoy, "Healthy bodies are all alike; all unhealthy bodies are unhealthy in their own way" (Neander, forthcoming). The same point can be made about function. There are many more states of an organ or organ system compatible with its failing to perform its function than with its performing its function. The ways the body can go wrong are bewilderingly diverse; the ways it can go right are relatively few and predictable. This does not mean there is only one way for the body to function well, and this is not to deny the plasticity of our nature: as we know from Darwin, variation is the rule of life (Amundson 2000).

Second, the former kinds of states (those in which it fails to perform its function) typically can be explained as resulting from the disruption of mechanisms for the latter kinds of states (those in which it performs its function). These two points suggest it is economical—it is a wise piece of biomedical strategy—to try to identify the relatively smaller number of mechanisms for functional states and to make sense of the diversity of pathological states as explicable consequences of the disruption of mechanisms for functions.

Moghaddam-Taaheri (2011, 608–10) makes a similar point in her argument that biomedical researchers should attempt to use information about mechanisms for functions to explain the diversity of pathological states. She does not, however, situate this important insight within a general theory of mechanism. This point is also suggested by Neander (forthcoming), who argues that biomedical research is best conducted when pathologies are characterized as deviations from proper function. While she does not specifically discuss mechanism, I believe the same point can be made with regard to mechanism: pathologies are most efficiently described as resulting from breakdowns in mechanisms for functions.

A simple example illustrates the epistemic benefits of the functional sense of mechanism. There are several devastating disorders of neurulation, or neural tube defects, including anencephaly, spina bifida, and craniorachischisis. These all result from incomplete or disrupted neural tube folding. There are two methods one might envision for tackling the etiology of, say, anencephaly. One method is to look for something like a 'mechanism' for anencephaly, replete with spatial, temporal, organizational, and perhaps hierarchical and serial constraints. Then, if one were feeling ambitious, one might go about seeking a 'mechanism' for spina bifida, and one for craniorachischisis as well. This would represent a 'piecemeal' approach, which is perhaps consistent with the progressive but unfortunate trend toward increasing medical specialization and fragmentation.

The second method would be to construe anencephaly not as 'having' a mechanism but as the explicable result of a broken mechanism (in this case, the mechanism for neural tube folding). This latter method has two main virtues. First, it forces the researcher to integrate information about the etiology of anencephaly with information about the mechanism for the corresponding function, in this case, neural tube folding. The first approach does not require this informational integration. In principle, in the first approach, one could have an exhaustive account of the 'mechanism for' anencephaly without knowing how neural tube folding is normally brought to completion or what its purpose is.

Second, the latter method could potentially be used to explain, or even predict, other neural tube defects—other than those that were the target of the initial investigation. This is because thinking of, say, anencephaly as the result of a broken mechanism encourages researchers to imagine other ways that the same mechanism could break and to consider its likely results. "If anencephaly results from preventing neural folding at the anterior neuropore, what happens if folding is disrupted at the posterior neuropore instead?" Of course, this is intended not as an actual reconstruction of the discovery of the etiology of spina bifida but merely a way to illustrate how seeing a given pathology as the result of broken mechanisms could potentially lead to the discovery or explanation of others.

Before moving to objections, there are four points of clarification to head off possible misunderstandings. First, I am not claiming that biomedicine cannot possibly progress on understanding a given pathology without a prior grasp of the mechanisms for functional states that underlie it. For example, prion-related diseases were believed to be caused by proteins before anybody knew what mechanism or mechanisms they disrupt.[9] (As it turned out, the prion co-opts the folding pattern of other proteins, which disrupts the ability of the latter to carry out their functions.) In such cases, researchers should just say that the pathology is likely the result of the breakdown of an unknown mechanism, or of the breakdown of a mechanism for an unknown function, rather than that there is a 'mechanism for' the pathology. In this way, the notion of a broken mechanism serves as a placeholder to indicate that kind of information that would be required for an ideally satisfying explanation.

Second, this does not mean we cannot use information about pathology to illuminate or discover the mechanisms underlying normal function. Neuroscience since the mid-nineteenth century has systematically exploited this procedure via lesion and ablation experiments, starting with the work of investigators like Pierre Flourens in France. But this is a point about the order of discovery, rather than the logic of explanation. Inducing pathologies in the brain can help us discover the mechanisms that are disrupted and to formulate hypotheses about what functions they serve and how they serve them. Equipped with this information, we then explain (in the causal sense) the pathology as a result of disrupting a mechanism for a function.

Third, I am not claiming that any given pathology results from disrupting a single mechanism. Some pathologies may require the simultaneous or successive breakdowns of multiple mechanisms in order to arise; I take this to be consistent with my view.[10] Finally, one might think that in some limited circumstances, the appeal to economy would actually justify talk of 'mechanisms for pathology'.[11] For example, there are different prion-related diseases; one might seek a certain inferential coherence by attempting to identify a 'mechanism for pathology' common to them all (i.e., a mechanism for protein misfolding). But the same biological facts can be described, with equal convenience, in terms of the way prions disrupt mechanisms for functions (the way they disrupt mechanisms for protein folding). The question then becomes, which of these two frameworks, on the whole, is more useful and valuable for biology and biomedicine? It seems to me that the general appeal

9. I thank Lindley Darden for this observation.

10. I thank Anya Plutynski for this point.

11. I thank an anonymous reviewer for this point.

to economy given at the beginning of this section adequately justifies the latter mode of description.

5. Two Objections. There are two objections I would like to consider. The first objection is that when biologists use the word 'mechanism', they are typically not appealing to the functional sense of mechanism. In other words, the term 'mechanism' is often used without any functional or normative connotations, even in biomedicine. This is suggested by the fact that biologists and psychologists sometimes talk about 'mechanisms for' pathologies, as seen by doing a search for 'mechanism for' in any biological or biomedical journal. However, it seems to me that often, when biologists use the phrase 'mechanism for pathology', they may be using it elliptically. In other words, where *Z* is a pathology, to say that *X* is a 'mechanism for' *Z* simply means *X* is a mechanism for some function *Y*, and *Z* results from its disruption.

For example, two recent popular presentations of scientific articles seem to recognize mechanisms for pathology (Marino 2005; Long 2010).[12] However, a careful reading of the articles on which they are based shows them to actually support the view that mechanisms serve functions. For example, in the scientific article on cancer metastasis, the mechanism identified, and described as a 'mechanism', is merely a mechanism for cell elasticity (a 'migration mechanism'). This property has functional significance but can be co-opted in such a way as to facilitate metastasis (Rolli et al. 2010). In the article on bone destruction, the mechanism identified, and described as a 'mechanism', is a mechanism for bone resorption, which, along with bone formation, performs the function of maintaining bone structure (Lynch et al. 2005, 489). It explains bone destruction in terms of the dysregulation of the balance between formation and resorption.

These articles suggest that when biologists talk of a 'mechanism for' pathology, the mechanism in question should often be understood not as a 'mechanism for' the pathology but a 'mechanism for' a lower-level component within a pathological system, which when considered on its own may have functional significance but which may be co-opted to produce pathology. One need not recognize mechanisms for pathology in order to accommodate this usage.

Despite the fact that many apparent counterexamples are not actual counterexamples, actual counterexamples probably exist. However, the actual counterexamples do not discredit or marginalize the functional sense of mechanism, as long as they are infrequent. Along the same lines, some of the founding documents of the new mechanism tradition emphasize that the various definitions of 'mechanism' offered are intended not as necessary and

12. I thank Stuart Newman for these references.

sufficient conditions for use but as characterizations that emerge from philosophical reflection on biological usage (e.g., Darden 2006, 273). The proposal offered here should be taken in a similar spirit.

A second objection can be formulated as a question. Why can't we accept that mechanisms serve functions and accept a liberal, Cummins-type theory of function that makes function relative to the interests and goals of observers? I have stated above that my view of mechanism helps to explain the normativity of mechanism (how mechanisms break) and that it is good for biomedicine. But could advocates of the CR theory not enjoy the same benefits, despite their view that what counts as a (system-level) function is relative to our goals and interests?

Let's examine each of my two main points in turn—that the functional sense of mechanism explains how mechanisms can break and that it is good for biomedicine—and consider how a CR theorist could lay claim to those same virtues. In my view, a broken mechanism is just one that is not performing its function. Now suppose we maintain that the function of a system (considered in toto) is a more or less arbitrarily chosen activity that the system, or others like it, occasionally produces. Then, a 'broken' mechanism is simply one that cannot perform that activity. If we append the claim that mechanisms serve functions with the CR theory of function, we can still make sense of the distinction between broken and working mechanisms. The distinction is simply relativized to the goals and interests of research communities; there would be no mind-independent fact about the matter. One could say that a function of the thyroid is to regulate blood pressure and that hypertension results from its disruption; one could also say that a function of the thyroid is to cause hypertension and that normal blood pressure results from its disruption.

I have misgivings about whether the CR theorist really has a clear notion of what it means for a trait to possess a function yet to be unable to perform it. This is because, at least for Cummins himself, a function is a disposition (Cummins 1975, 758); if a system does not have the disposition to perform an activity, then that activity cannot be its function. But I will suppose for the sake of argument that the CR theorist could find some way to explain what it means for a mechanism to break.

The second virtue I claim for the functional sense of mechanism is that it maximizes the inferential coherence of biomedicine because it makes for better generalizations. This benefit requires that we sharply distinguish between the claim that *X* is a mechanism for *Y* and the claim that *Y* results from a broken *X*. But a CR theorist could accept the distinction: like the distinction between a broken and working mechanism, he or she would just relativize it to the interests and goals of the research community (e.g., Hardcastle 2002, 153). Fortunately, biomedical researchers have the goal of making people well; given their characteristic interests, they tend to construe the

functional organization of the body in terms of health-related norms. Given their disciplinary aims, the CR view would allow them to say that the function of neurulation is to support the formation of the brain and spinal cord, and diseases such as anencephaly result from its disruption.

I admit that there is nothing intrinsically inconsistent about holding both that mechanisms serve functions and that there is no mind-independent fact about what counts as the function of a trait. My main problem is with the CR theory itself. The way the CR view licenses function ascriptions seems irreconcilable with the way scientists actually talk. This is a version of the classic problem of overbreadth (e.g., Millikan 1989, 294; Kitcher 1993, 390). Suppose, for example, an insidious alien species wishes to destroy human life and concludes, after long deliberation and painstaking experimentation, that the most effective way to do so would be to shroud the earth in a toxic gas that is readily absorbed by the lungs and deposited in the bloodstream. On the CR theory, they would be correct, relative to those goals and interests, to say that the function of human lungs is to deposit a toxic gas into the bloodstream. That strikes me as counterintuitive. It seems to me that the function of the lungs is to distribute oxygen and to remove waste and that this proposition is not falsified just because someone has other plans for it.

However, I need not rest my case on a fictitious example. There are scientific disciplines devoted to promoting pathology; pest toxicology is one of them. If functions are relative to the goals and interests of observers, it would seem to follow that pest toxicologists have a different construal of the functional organization of pests than do, say, evolutionary biologists. Pest bodies should be seen as little more than effective mechanisms for absorbing poisons or transferring them to their conspecifics. However, a casual perusal of articles that appear in pest toxicology journals, such as *Pesticide Biochemistry and Physiology* or *Chemical Research in Toxicology*, suggests that they do not. Instead, pest toxicologists seem to think that the purpose of the toxins they manufacture is to disrupt or co-opt normal cellular or other physiological functions on the part of undesirable living things. For example, one study describes how "exposure of rats to a single pesticide . . . may result in the *impairment of antioxidant mechanisms*" (Aydin 2011, 169; emphasis mine); another holds that fungicides work by "disrupt[ing] basic cellular functions" (Casida 2009, 609). This implies that toxicologists tend to construe the functional organization of the pest in much the same terms that biomedical researchers construe the functional organization of the human body. At the very least, the kind of discipline relativity of function that one would expect on the CR theory does not exist.

So far I have said nothing about the validity or legitimacy of the concepts of mechanism that have emerged from the new mechanism literature, concepts that tend to emphasize structural and dynamic features of complex systems. I have presented the functional sense of mechanism as an alternate

explication that accurately characterizes many paradigm mechanisms in biology. However, to the extent that this is a plausible theory, one cannot help but wonder how it relates to other theories of mechanism. Should functional mechanisms be seen as a subset of mechanisms 'proper'? Do they coexist harmoniously? Or do functional mechanisms somehow supplant the other kind? Moss, for example, does not find room for the 'new mechanism sense' in his taxonomy of mechanism: he has something like functional mechanisms, and he has dull chains of cause and effect that make few assumptions about spatial, temporal, or hierarchical structure. He does not seem to find use for additional theories of mechanism. This seems to me, broadly speaking, to be an empirical question.

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