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When is it Mental?

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ABSTRACT

Most philosophical debate over mental causation has been concerned with reconciling commonsense intuitions that there are causal interactions between the mental and the physical with philosophical theories of the nature of the mental that seem to suggest otherwise. My concern is with a different and more practical problem. We often confront some cognitive, affective, or bodily phenomenon, and wonder about its source – its etiology or its underlying causal basis. For instance, you might wonder whether your queasiness due to something you ate, or whether it is just nervousness, or whether your aunt’s memory loss is a neurological problem or a psychological response to trauma. Such questions attempt to localize the causes of a phenomenon at some level in the complex multi-level systems that we human animals are. In this paper I will attempt to tease out the sense of level implicit in such questions, and to show how it is related to current mechanistic accounts of levels. I will argue that the explanation of our practices of level attribution is deeply pragmatic. Such attributions are often attempts to locate the causes of problems, and to identify interventions that could solve those problems.

Keywords: levels of organization, mental causation, mechanistic explanation, mind-body problem

Most philosophical debate over mental causation has been concerned with reconciling commonsense intuitions that there are causal interactions between the mental and the physical with philosophical theories of the nature of the mental that seem to suggest otherwise. On a Cartesian view, how can mental substance interact with physical substance? On more recent views, how can mental properties have causal powers if they supervene on physical properties already sufficient for the effect?

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I shall say a few things about these issues in the course of this paper, but my main concern will be with a different and more practical problem. Very often we confront some cognitive, affective, or bodily phenomenon, and wonder about its source – its etiology or its underlying causal basis. For instance, we might ask:

- Is the problem with Tiger's golf swing mental or physical?
- Is my queasiness due to something I ate, or is it just psychological?
- Is my aunt's memory loss a neurological problem or is it a psychological response to trauma?

These are questions about where to locate causal responsibility for some phenomenon. When answers are given they typically are expressed using adjectives like 'mental', 'physical', 'neurological', 'developmental', 'cognitive', and so on. These adjectives appear to refer to different kinds, or different levels, of causes or causal processes. These questions are challenging because the phenomena we seek to understand depend upon many kinds of causes operating at multiple levels. If, for instance, psychological processes depend upon neurological and ultimately physical processes, it is *prima facie* odd to try to single out one level of causal responsibility.

My aim in this paper will be to explore the basis upon which such questions can be answered. I will begin with an overview of various conceptions of levels, and will in particular try to understand how claims about mental and physical levels are connected to what I take to be the most ontologically significant conception of levels, what I call, following (Craver, 2007), levels of mechanisms. In part II, I will review some elements of my own mechanistic account of causation, with an emphasis on how it makes sense of the kinds of inter-level causal claims that appear to be at play in judgments about levels of causal responsibility. In parts III and IV, I will return to the question of how we identify the causal basis of phenomena in systems, like human brains, which involve multi-level causal mechanisms. I will suggest that in many cases, such questions are asked within a context in which the phenomena are taken to be problems. This problem context provides constraints, which allow us to find levels at which the problem occurs (where the mechanism is "broken") as well as levels at which one could intervene to solve the problem.

1. Levels and Mechanisms

Reference to levels is commonplace in science and medicine. Consider, for example, the definition of a mental disorder from the most recent addition of the

Diagnostic and Statistical Manual of Mental Disorders (DSM-5):

A mental disorder is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning (American Psychiatric Association, 2013, sec. I).

This definition does not use the word 'level', but it does refer to three distinct kinds of processes, and these processes, I would argue, are thought to occur at some level. We see this implied in part by the notion that the psychological, biological and developmental *underlie* mental functioning. These levels are clinically important, as they will impact our understanding both of what the disorders are and how they should be treated.

But what are levels? It is common to think of levels both as features of the world and as features of the organization of scientific fields and their products (Craver, 2007, chap. 5). Often it does no harm to ignore this distinction, because of correspondences between levels in nature and levels in the disciplines that study them: psychologists study the behavior of humans and cognitively similar animals; sociology studies the behavior of societies (groups of these animals) and so on. But it does not take long for difficulties to arise. The major scientific disciplines study the natural world at a wide range of scales. Physics operates from the sub-atomic to the super-galactic; biology, from molecules to ecosystems. Even fields that appear to be focused more narrowly (organic chemistry, invertebrate biology, etc.) deal with systems with multiple levels of organization. Chemistry, for instance, operates at "the molecular level" – but molecules themselves come in vastly different sizes and degrees of organizational complexity; larger molecules are not simple aggregations of atoms, but have multiple levels of substructure that are central to understanding their behavior.

Given that a good deal of work in both metaphysics and philosophy of science appeals to levels, the literature on levels is surprisingly sparse. Within philosophy of science, classic accounts of inter-theoretic reduction (Oppenheim & Putnam, 1958; Nagel, 1979) assume both that there is a linear ordering of levels in nature (part-whole microreduction) and that these orderings correspond to a hierarchy of scientific disciplines. In the philosophy of mind one often finds an even simpler idealization of levels and inter-level relations. Early statements of the identity theory, as well as Davidson's anomalous monism and Kim's account of the causal exclusion problem typically refer to (Kim, 1998; Davidson, 1970)

two levels – the mental (or psychological) and the physical.¹ These accounts also see a direct correspondence between levels of scientific description and levels of things in the world. They move largely without comment from discussions of mental and physical predicates or statements to discussions of mental and physical properties or events. I do not mean to suggest that these philosophers are unaware that there are more levels than just the mental and the physical, or that there is a distinction between predicates and properties; but do think these common features of philosophical discussions indicate a sense in these communities that these facts are not germane to the philosophical issues.

One of the first philosophers to think about the consequences of the messier reality of levels was Bill Wimsatt (1972; 1976). Wimsatt is a realist about levels, seeing levels as grounded in hierarchies of compositional relations between parts and wholes as they exist in the world. In this respect, his account resembles the micro-reductionist picture. But Wimsatt recognizes that compositional relations of this kind will not give the linear ordering that Oppenheim and Putnam imagined. Instead there is a vast and bushy partial ordering corresponding the various ways in which some set of fundamental entities aggregate into things as diverse as proteins, rock formations, stellar clusters and social groups (cf. Love, 2011).

Wimsatt argues that levels are intimately bound to what he calls ‘perspectives’:

Perspectives involve a set of variables that are used to characterize systems or to partition objects into parts, which together give a systematic account of a domain of phenomena, and are peculiarly salient to an observer or class of observers because of the characteristic ways in which those observers interact causally with the system or systems in question (Wimsatt, 2007, p. 227).

Wimsatt calls perspectives ‘quasi-subjective’. They are points of view on what objects or systems within a domain are doing. What makes them quasi rather than genuinely subjective is that these points of view allow scientists to selectively attend to genuine aspects of what the things they observe are doing. The screen-writer, the camera operator, the director and the diction coach are all watching the scene, and each has a different point of view on what the actors are

¹ Matters are not helped by an ambiguity in the term ‘physical’. There is the physicists’ sense of physical and the physician’s sense. In ordinary discourse about mind and body, ‘physical’ means ‘bodily’.

doing. They will break up the scene and the actors' performances in different ways, but the things they attend to (the lines, the camera angle, the blocking, the accents) are all aspects of what is really going on.

Wimsatt's views on levels anticipate and inform much of the work on hierarchically organized mechanisms that goes under the banner of "the new mechanism".² Though there is considerable debate about exactly what should or should not count as a mechanism, for purposes of this paper I will assume that any mechanism must at least satisfy a condition I call minimal mechanism:

A mechanism for a phenomenon consists of entities (or parts) whose activities and interactions are organized so as to be responsible for the phenomenon.³

I shall not try to explicate the concept of minimal mechanism here, but will highlight a few features that are relevant to our discussion.

First, mechanisms are always mechanisms *for something*. They are individuated by the phenomena for which they are responsible. Engines are mechanisms for rotating drive shafts, the cardio-pulmonary system is a mechanism for transporting oxygen and nutrients to various parts of the body, and so on. When a phenomena arises as a result of the activities of a mechanism, we can call that phenomenon mechanism dependent. The mechanism's phenomenon plays very much the role of Wimsatt's perspectives in governing decompositions. There are many ways that a system can be decomposed into parts, and is only by identifying what a mechanism is doing that one can articulate the parts by which the mechanism is doing it (Kauffman, 1970).

Many of the phenomena for which mechanisms are responsible may be thought of as functions of the mechanism, but the minimalist conception of mechanism places few constraints on what should count as functions. From the minimalist point of view, a car may be a mechanism for getting to the grocery, but it is just as much a mechanism for melting the ice cream that you left in the back seat. We can think of there being a functional relation between mechanisms and their phenomena, but it is in the weak sense of a causal role function, rather than in terms of any account of etiological or proper function.

² Well-known accounts of mechanisms include (Glennan, 1996; Glennan, 2002a; Machamer, Darden, & Craver, 2000; Bechtel & Richardson, 1993; Bechtel & Abrahamsen, 2005; Craver, 2007). For introductions to the literature and a survey of some debates among new mechanists see (Glennan, 2015).

³ This conception of minimal mechanism is defended in (Glennan, forthcoming; cf. Illari & Williamson, 2012). Illari and Williamson and I argue new mechanists should accept it as a minimal condition, and that disagreements are mostly about whether minimal mechanism is too permissive.

Second, mechanisms are spatiotemporally localized. The phenomena for which mechanisms are responsible are produced or maintained by the causally organized activities and interactions of mechanism's parts, and the spatial and temporal organization that gives rise to this causal organization. The engine's capacity to rotate the crankshaft, for instance, depends upon the precise arrangement of parts (e.g., rods, pistons, cam shafts, spark plugs, fuel), and the various sorts of activities and interactions they engage in (e.g., sparking, burning, rotating).

Third, mechanisms can be both systems and processes. In the systematic sense, mechanisms are complex objects that do things. A heart, for instance, is a mechanism for pumping blood. Mechanical processes, on the other hand, are ordered and orchestrated sequences of activities and interactions of entities that typically lead from a start state to an end state, or perhaps in a cycle of states. Metabolic processes like photosynthesis or the ATP cycle are mechanisms in this sense. These two senses are interrelated, because the capacities of mechanical systems arise from processes involving their parts.⁴

Fourth, mechanisms are hierarchical in the sense that the parts and their activities and interactions can be broken up into further parts and their activities and interactions. This feature of mechanisms has particularly been emphasized by Carl Craver, who represents it schematically in figures like Figure 1.

Here the *x*s represent the parts of the mechanisms, the φ s, their activities and interactions. Reading from left to right we see a mechanical process, by which some start up condition (represented by the left-most arrow, triggers a sequence (including possible cycles) leading to a termination condition (the right-most arrow). The *x*s are parts of a system *S*, and their collective activities and interactions are what constitute the system's phenomenon – i.e., the activities in which the system as a whole engages (*S* ψ -ing).

Mechanisms are hierarchical. A mechanism (*S* ψ -ing) may be embedded in a larger mechanism, and the parts of a mechanism (the *x*s φ -ing) may themselves be mechanical systems whose behavior will be produced by the activities and interactions of their parts. Activities and interactions, like entities, are decomposable. The interactions represented by the arrows typically involve complex mechanistic processes, which are constituted by further entities whose activities are responsible for the larger activity.

⁴ Given the minimal conception of mechanism it will not always be the case that mechanical processes involve the operation of mechanical systems. Such processes are instances of what I call ephemeral mechanisms (Glennan, 2002b; Glennan, 2010b).

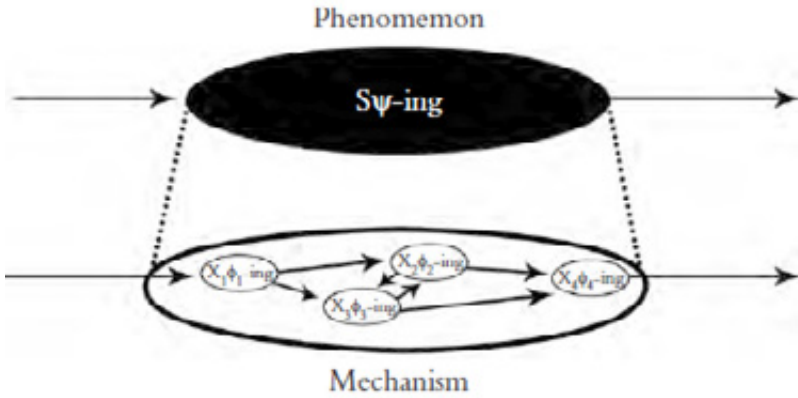


Figure 1: A schematic view of a mechanism (Craver, 2007, p. 7)

As an illustration, consider the mechanism of the action potential. An action potential is a process by which the membrane of a neuron depolarizes and repolarizes – the characteristic spiking by which neurons “fire”. The firing of the neuron is an activity (ψ) of the cell as a whole (S), but the cell has the capacity to generate an action potential only in virtue of the organization of its parts. The cell, for instance, has a membrane, and this membrane has various sites (the ion channels) which allow passage of charged ions across the membrane, leading to the membrane’s depolarization. The opening and closing of these channels are examples of components of the mechanisms and their activities. Again, the parts of the mechanisms have parts (membranes and ion channels are complex structures which themselves have parts), and the mechanism itself is part of larger mechanisms and its activities will be stages in these larger activities. One neuron’s firing is part of the vast patterns of neural firings that are essential for the central nervous system as a whole to engage in its activities.

The hierarchical account of mechanisms sketched here leads naturally to an account of what Craver (Craver, 2007, chap. 5; Craver & Bechtel, 2007) calls mechanistic levels. Two entities are on the same mechanistic level just in case they are both parts of a mechanism, and a mechanical system is one level up from the parts that constitute it. In terms of the figure, the x s are at one level, and S is at the level above. Dotted lines represent the constitution relation.⁵ Mechanis-

⁵ Here and throughout this paper I will use the term ‘constitution’ in the proprietary sense of mechanistic constitution – the relationship between a mechanism and the entities, activities and interactions that make it up. This use of the term ‘constitution’ was introduced by Craver and is now well-established in

tic levels are a kind of compositional level, where parts are at a lower level than the wholes which they make up, but the parthood relation is determined by the activities of the parts, and whether these parts contribute to the activities of the whole.⁶ Entities, activities or interactions can only be judged to be at the same or different level when they are involved in the same mechanisms. There is no answer to the question about whether a rock on a distant planet and a microbe in our ocean are on the same or different level, because they aren't part of any common mechanism. Since mechanistic levels are only defined within a mechanism, mechanistic levels are essentially local (cf. Love, 2011).

Though mechanistic levels describe the fundamental hierarchical organization of complex systems in the natural world, most levels talks refers to these structures only loosely. Adjectives like 'neurological', 'mental', 'cognitive' and 'developmental' are best understood as referring to what I will call "heuristic levels". These levels (and you could call them kinds, so long as you didn't think of kinds from an essentialist perspective) clump together a somewhat heterogeneous set of entities, activities, systems and processes. For instance, neurological processes and mechanisms include the activities of single neurons and their parts, as well as synapses and larger structures involving, bundles of nerve fibers, tissues and brain regions. Moreover, a mechanism that fits within a heuristic level will typically span multiple mechanistic levels. For instance, the visual mechanism for shape recognition would typically count as a cognitive mechanism, but the processes underlying the phenomenon of shape recognition operate at mechanistic levels ranging from those inside single cells to higher levels involving the organized activities of various tissues within the eye, or various regions of the visual cortex.⁷

Although they are loose, heuristic levels help localize the causes of phenomena. If we learn the problem with Tiger's swing is mental, we really have said

the mechanisms literature. The term 'constitution' also has a history in recent metaphysical debates (e.g., Baker, 1997), where constitution is a "made up of" relation, but not one between mechanisms and their parts. It is instead a relation between two whole things – like a statue and the marble of which it is made or a person and her body. I shall not try here to unpack the relation between these two senses.

⁶ Space prevents me from considering when a set of parts constitutes a mechanical system, and how (or if) one can separate a mechanism from its environment. Many have been skeptical of Craver's attempt to mark this boundary, and Craver himself (2013) argues that such boundaries do not exist except from the perspective given taking some event or state as the phenomenon for which the mechanism is responsible.

⁷ Heuristic levels are not local in the way that mechanistic levels are, because they provide a way of classifying widely distributed sets of particulars. One might for example, find instances of cognitive processes in systems found in distant parts of the galaxy.

something about where things are going wrong. We shall return to these levels in the last two sections of the paper. But first we need to show how to make sense of inter-level causal claims on the mechanistic account.

2. Causation in Multi-level Mechanisms

Both the definition of minimal mechanism and examples we have discussed imply that mechanical processes and systems are causal structures. The sense in which they are responsible for phenomena is a causal one. While some (e.g. Woodward, 2013) argue that mechanisms are simply certain kinds of causal structures, and see causal relations as being more basic than mechanisms, I have argued (Glennan, 1996; 2011; 2010a; forthcoming) that mechanisms underlie causation, in the sense that mechanisms are truthmakers for causal claims. Specifically, causal claims are existential claims about mechanisms (cf. Waskan, 2011): an event a causes an event b is true just in case there is a mechanism whereby a contributes to the production of b .

Many recent discussions of causation suggest that there are two concepts of cause, or two kinds of causal relations – difference making (or dependence or causal relevance) and production (Glennan, 2010a; Hall, 2004; Menzies, 1999; Woodward, 2011). Theories of causation can be classified according to which sort of causal relationship is taken as primary, and mechanistic approach fits within the production tradition.

Production accounts emphasize the continuity between causes and effects and give primacy to singular causes. On the other hand, difference-making accounts are comparative or contrastive: causes are things that make a difference relative to actual or counterfactual circumstances in which the cause does not occur. For instance, one might claim that a mutant allele is the cause of a disease because organisms with the mutant allele develop the disease, while those without it do not. The mechanistic theory takes production as basic, because causal relations are not essentially contrastive, and require the existence of productive causal processes connecting cause to effect. What makes a a cause of b are facts about mechanical processes actually connecting a and b and not anything about what does or would have happened in other circumstances.

Even so, difference-making is a central feature of causal and explanatory claims, and a mechanical theory must make sense of them. To see how, consider a canonical form of singular causal claim which references both relationships (Glennan, 2010a; forthcoming):

Event e **produced** event e in virtue of **relevant feature** p .

For instance:

Elliot's throw of the bowling ball produced a strike in virtue of its speed and launch angle.

On a mechanistic theory of causation, this claim would be true if there were a mechanical process whereby the throw is connected to the knocking down of the pins. That process would involve multiple stages, the roll of the ball down the lane, the striking of the first pins, and chain reactions as those pins knock down others. The point of the relevance claim is to allow one to express the fact that certain features of the entities and activities involved in the process make a difference to the event, while others do not. Clearly the speed and direction of the toss would make a difference, as would the weight of the ball, but the color of the ball or the sound it makes as it rolls down the alley would not.

So far we have focused on a simple process operating at a single mechanistic level. Let's consider how the mechanistic account works for inter-level causal relations. Craver and Bechtel (2007) have argued that the correct way to understand such cause-effect relationships is in terms of what they call a "mechanistically mediated effect" – a hybrid between intra-level causal relations and inter-level constitutive relations. To illustrate their approach consider this example: as my fingers type on the keyboard, their motions are controlled by the contraction of muscle fibers attached by tendons to bones in my finger. How exactly do these muscle contractions work? There are quite a number of levels just within the muscle. Muscle tissue (1) consists of bundles of cells called myocytes (2) which contain with them large quantities fibers called sarcomeres (3). Sarcomeres in turn are composed of collections of filaments (4) made of the proteins myosin and actin (5), and the contraction of sarcomeres, arises, as actin bands are pulled along the fixed myosin bands, powered by ATP (Krans, 2010). It is tempting to see the contraction of muscle fibers as an example of bottom-up causation – the muscle contraction is caused by the contraction of the myocytes, which is caused by the contraction of the sarcomeres which is caused by the sliding filaments within the sarcomeres. But this, for Craver and Bechtel, is a mechanistically mediated effect. All the causation is actually within level – for instance, the pulling of the actin along the myosin bands, or the pulling of the bone by the contracting muscle. But the contraction, say, of the sarcomere isn't caused by the sliding of

the filaments that make up the sarcomere. The contraction of the sarcomere *just* is the sliding of those filaments. Schematically, the contraction of the sarcomere is $S\psi$ -ing, and the sliding of the bands within the sarcomere are the $xs\varphi$ -ing. They don't cause the contraction; they constitute it.

This example also gives us an example of top-down, and mental causation. What is ultimately causing the many highly orchestrated muscular actions that lead to my fingers striking the keys is my intent to put certain words on the virtual page. That basic intent is about all that is within my conscious control. I am not aware of the intent to type certain letters, or certainly to move my fingers in any certain way. As I am fluent in English and a touch typist, all of this is automatic. Nonetheless, it is the high level conscious intent that seems to cause all lower level events, from the firing of motor neurons to the expenditure of ATP in actin-myosin reactions. All of these though are mechanistically mediated effects. My intention to type the word 'myosin' activates certain motor neurons that control muscles in my fingers, but my intention is constituted by the activities of neurons distributed across various regions of my brain. And it is the intra-level interactions of those neurons with motor neurons and ultimately muscle cells that cause the muscle contractions which move my fingers across the keyboard.

When applied to cases of mental causation, Craver and Bechtel's account of mechanistically mediated effects leads very naturally to a version of identity theory. My intent to type the word 'myosin' is a mental event, but that event is identical to some neurological event, and hence there is no difficulty in this neurological event causing other neurological events, like the activation of motor neurons, since the events are all at the same level.

This identity theory is, however, a token identity theory, and a mechanistically mediated effect is a token effect. The theory does not identify psychological and neurological events as types. As such, the theory can honor intuitions that in cases of mental causation, it is high-level mental properties rather than neurological properties that are really the difference makers. Take again the case of typing. What is the common difference-making property in virtue of which I type the letter 'm' on the many occasions that I do so? It can be nothing other than my intent to type an 'm'. Any given token of this intent is identical to some transient neurological state, but because the neurological context changes, one should expect that further tokens will have somewhat different neurological properties. And of course the tokenings will differ more substantially across different brains. What binds these neurologically diverse states together is their

occupying the same causal role, and the different tokenings have some higher level similarities that allow them to do this. For this reason we can say that high level mental properties, like intentions are difference-making causes.⁸

Craver and Bechtel's account of mechanistically mediated effects strictly speaking bans inter-level causation, but it does explain the sense of claims of top-down and bottom up causation, and as I've construed it, it saves the causal relevance of the mental. It thus begins to answer the question 'When is it mental?' A phenomenon is mental when it is in virtue of mental properties of events that it occurs. But this is only the beginning of an answer, because in general properties at multiple levels can make a difference to a phenomenon, and we need some account of why some levels are chosen as primary or central.

3. Causes and Problems

When we assert that phenomenon is of some (heuristic) level, we often mean that the phenomenon itself occurs at that level. A sensation or a belief is mental in this sense, while a swollen knee is physical. But my concern in this essay are assertions not about the level of the phenomenon itself, but about its causes, i.e., about the mechanisms responsible for it. For instance, we might say of an LSD induced hallucination that it is a chemical phenomenon, meaning that the event that caused it was the ingestion of a chemical. Had the hallucination been initiated by an ecstatic experience in a tent revival meeting, we might instead have called it a psycho-social phenomenon. In either case, the hallucination itself would be mental, but our causal explanation of the hallucination would appeal to events and mechanisms at different levels.

In light of the multi-level character of mechanistic processes, it is far from clear what principles are at work in identifying "the level" of the cause. Psychological processes are neurological processes, and neurological processes biochemical ones, so why favor one level over the other? I cannot hope to explore all of the constraints that explain level attributions, so I will focus on a particu-

⁸ There are other ways to interpret this situation. John Heil and David Robb (Heil, 2013; Heil & Robb, 2003) have argued that strictly speaking there are no higher level properties at all. The only real properties are the properties that belong to basic substances, perhaps some elementary physical properties. All other properties are just compounds of these basic properties of the basic substances. What I am calling higher level properties they treat simply as abstract descriptions of complex objects that are in truth nothing but modification of the basic substances and their properties. I have sympathy for this position, but for me the fact that there are objective similarities that account for objectively similar causal powers across these complex objects is enough to get genuine higher-level properties – but I will not argue the point here.

lar class of cases that I will call *problems*. Classifying phenomena as problems is a normative exercise that involves distinguishing the problematic phenomena from alternative phenomena that are taken to be normal.

For instance, when we ask about what's wrong with Tiger's swing, we are suggesting his current swing has problems, and we are in so doing implicitly contrasting it with other swings. The question then becomes, what is it that is different about the mechanisms responsible for Tiger's swings now that distinguishes them from those responsible for his earlier unproblematic swings? Is it mental – perhaps induced by anxieties about bad media coverage – or is it really about a physical injury to his knee?

When we identify some phenomenon as a problem, we seek not just to explain it, but also to correct it. A *solution* to a problem is an intervention that eliminates or at least mitigates the problem. Often the level of the problem and the level of the solution coincide – a knee problem requires a physical therapist, a mental problem a psychotherapist – but this is not always the case.

To explore the considerations that lead us to identify a problem or its solution as mental, physical or otherwise, I want to focus on problems of human health. The problem of impotence – in contemporary clinical parlance, erectile dysfunction (ED) – will provide a good initial example that can help us understand the various factors which lead to ascribing the causes of phenomena to some level. Often, I shall argue, the levels we choose say as much about our technology, our economics, our moral attitudes and our politics, as about the nature of the phenomenon itself.

Failures of sexual performance are paradigms of physical problems with mental causes. Writing in the late 16th century, Michel de Montaigne recognized this in his remarkable essay “On the Force of the Imagination”. Of penises he complains:

The indocile liberty of this member is very remarkable, so importunately unruly in its tumidity and impatience, when we do not require it, and so unseasonably disobedient, when we stand most in need of it: so imperiously contesting in authority with the will, and with so much haughty obstinacy denying all solicitation, both of hand and mind (Montaigne, 1877).

For Montaigne, impotence is an example of ways in which mental imaginings can cause physical failings. He recounts stories of sexual failures of men who believing themselves to be in enchanted, or simply in remembering theirs or others' past disasters, are unable to get erections and have sex with their part-

ners. In modern parlance, we might say “they psych themselves out”. These causes clearly are at a mental level, in the sense that the mechanism which produces the effect involves perceptual and cognitive processes. Stories of curses must be heard, believed and remembered to have an effect.

Montaigne also recognized that there were mental solutions to the problem of impotence, in the sense that psychological interventions could alleviate the problematic behavior. He recounts a story in which he sought to aid a friend who, on his wedding night, thought himself to be cursed by a jealous former suitor of his new wife. In case of troubles, Montaigne gave his friend detailed instructions on a set of elaborate rituals involving, among other things, a medallion engraved with «celestial figures» that he assured him would cure him, and the treatment did the trick. Montaigne remarks:

These ape’s tricks are the main of the effect, our fancy being so far seduced as to believe that such strange means must, of necessity, proceed from some abstruse science: their very inanity gives them weight and reverence (ibid)

Writing in the 16th century, Montaigne has already recognized the remarkable power of the placebo effect.

Montaigne’s essay supports intuitions that the causes of impotence (ED) can be psychological, but ED can have physical causes as well. The Mayo Clinic’s website guide to ED in fact explicitly divides its list of potential causes between the physical and the mental. Among the physical causes it mentions heart disease, clogged blood vessels, obesity, diabetes, alcoholism, and «surgeries or injuries that affect the pelvic area or spinal cord» (Mayo Clinic Staff, 2015). Psychological causes include stress, anxiety, depression and relationship problems. ED, like many other disorders of mind and body, is a syndrome – a cluster of symptoms with different causes in different cases. ED in one patient may be a result of blood flow problems due to obesity, whereas in another it may be due stress diminishing hormonal response in sexual situations. Syndromes are not natural kinds. There are no laws describing their operation, and no universally effective prescriptions for treatment. From a mechanistic perspective, none of this is surprising. The mechanistic account of causation stresses that causes and effects are singular events and states of affairs that are connected by localized processes. Causal generalizations are approximate descriptions of the behavior of idealized types, but the reality varies from case to case (Glennan, 2011; Glennan forthcoming).

Focusing on the single case constrains the levels question, but it does not resolve it. Even in the single case, causal processes are multi-level, so we need an explanation of how to single out particular levels of those processes as causally relevant to the phenomena. The key to offering such an account is to recall that causal relevance (and with it the relevant level of causes) is a comparative notion. The causally relevant level is the level at which causes make a difference. But if difference-making is comparative, what is the comparison to? Here is where focus on problems is helpful. By contrasting problematic phenomena with normal phenomena (or pathological phenomena with healthy phenomena), one can look for the components or activities that are functioning abnormally. In other words, one can find at what location and level the mechanism is broken.

The minimal conception of mechanism does not distinguish between properly functioning and broken mechanisms. Mechanisms are just the causal structures that are responsible for phenomena – problematic or not. But the minimal conception can be augmented by placing a normative constraint on the relationship between a mechanism and the phenomenon for which it is responsible. Such a conception is provided by Justin Garson’s (2013) «functional sense of mechanism». On Garson’s view, the crucial thing about mechanisms in the functional sense is that they may malfunction: «“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» (2013, p. 320). Garson does not insist on any particular theory of normative functions, but he does require that function is more than causal role. There are not, in this sense, pathological mechanisms. Pathologies are instead breakings of functional mechanisms.⁹

Garson argues that the functional sense of mechanism is useful «because... [t]hinking 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» (*ibid.*, p. 318). We

⁹ While I agree with Garson that thinking of pathologies as breakdowns of mechanisms is a useful strategy, in some biomedical contexts researchers speak of disease mechanisms. This conceptualization can also be clinically useful. Instead of trying to figure out how to fix the broken “healthy” mechanism, one instead considers as “normal” the mechanical processes by which the disease progresses, and figures out how to disrupt that mechanism. So, for instance, in some hormonally fed cancers (as some forms of breast or prostate cancer), one disrupts the growth by suppressing production of estrogens or androgens.

can see just how such a strategy works in the diagnosis and treatment of ED. If a patient suffers from ED, the aim is to isolate which of the many mechanisms involved in normal sexual function is broken, or is being interfered with. For instance, the problem may be a vascular problem – blood vessels supplying blood to the penis may be constricted. On the other hand, it may be stress related. Stress responses in the autonomic nervous system suppress sexual arousal. The system of stress response is not what is broken. The problem lies in whatever psychological conditions and mechanisms are triggering the stress response at a time when sexual arousal is a normal response.

While attending to breakdown helps us assign a level of causal responsibility, there is nothing to say that breakdowns will be localized to one mechanism or one level. Very often interaction effects will make the phenomena truly multilevel. The Mayo Clinic website makes this point about ED: «...[A] minor physical condition that slows your sexual response might cause anxiety about maintaining an erection. The resulting anxiety can lead to or worsen erectile dysfunction». In cases like this, difference-making causes (and breakdowns) are both mental and physical.

Thinking about problems helps to identify contrasts and to find relevant difference makers, but it brings with it important and ineliminable pragmatic elements. In the case of human health and behavior, to identify some phenomena as problems and others as not is to make social and moral judgments about the acceptable range of behavioral norms. What is a «clinically significant disturbance», as DSM-5 puts it, is a consequence of social norms. In the contemporary medical community variations in sexual orientation are taken to be acceptable, but this was not always so. Homosexuality used to be a disorder. If a gay man failed to be aroused in a sexual encounter with a women, the failure would be due to a defect of sexual orientation. Such a view may strike us as unenlightened or even bigoted, but it is not hard to find cases in which things that we now accept as real problems involve appeal to norms that are historically contingent and potentially problematic. Consider again ED. ED is a widely diagnosed and treated disorder in large part because of cultural norms, supported in part by the advertising budget of the pharmaceutical industry, which create the expectation that men should continue to be sexually active well past middle age. But decreased capacity for sex (and fertility) is a common accompaniment of the aging process. The use of limited medical resources to treat this as a curable disease may be a consequence of economic and gendered power differentials that are justifiably cause for scrutiny.

Here is a different example to consider, which will highlight some other features of level ascription. Obesity research suggests that the feelings of hunger and satiety which trigger our eating and cessation of eating are regulated in part by two hormones, ghrelin and leptin. Ghrelin is produced in the stomach and gut when the stomach is empty, and appears to stimulate feelings of hunger by binding receptors in the hypothalamus. Leptin, mainly secreted in adipose tissue (body fat), is released as energy stores increase, and triggering feelings of satiety, also through a pathway in the hypothalamus (Klok, Jakobsdottir, & Drent, 2007).

If this were the whole story, it would be appropriate to classify hunger as a gastro-intestinal and hormonal phenomena – that is to say, as mental phenomena whose underlying causes are gastro-intestinal and hormonal. Hunger is caused by ghrelin which is caused by empty stomachs. But the mental may be implicated in these processes in more subtle ways. Perhaps the causes of hunger pangs in part psychological. We are all familiar with the ways in which our desires to eat are affected by our psychological state. I for instance at this moment am struggling to handle my usual case of the writing-inducing munchies. But how do psychological events and states do this? A recent study entitled “Mind over milkshakes” (Crum, Corbin, Brownell, & Salovey, 2011) suggests one way. Crum and her colleagues studied ghrelin response to the consumption of milkshakes. The two groups of subjects consumed identical 380 calorie milkshakes with different labels – one was labeled a “sensible” milkshake with 180 calories, while the other was labeled an “indulgent” milkshake with 620 calories. The researchers reported that «[t]he mindset of indulgence produced a dramatically steeper decline in ghrelin after consuming the shake, whereas the mindset of sensibility produced a relatively flat ghrelin response. Participants’ satiety was consistent with what they believed they were consuming rather than the actual nutritional value of what they consumed» (ibid., p. 424).

While this is another case of the causes of phenomena turning out to be mental, it has somewhat different features than the ED example. In the first place, this result is not supposed to show something about any specific case of hunger, but to show something general about the sources of hunger pangs across individuals, and across time within individuals. Relatedly, it is not obviously about a problem. This research suggests a mechanism by which beliefs about food consumed, and not just the food itself, can affect feelings of satiety, but it does not suggest that this mechanism is abnormal or pathological. In

highlighting hunger as a mentally-caused, the researchers are not suggesting that beliefs are the primary cause of feelings of hunger or satiety, but simply highlighting it as a hitherto unidentified part of a multi-level mechanism.

4. Solutions

We have discussed two different circumstances under which a phenomenon may be said to be of some level. First, there is the level of the phenomenon itself, as when we call hallucinations mental. Second, there is the level of the causes that produce, underlie or sustain phenomena, as when we call LSD-induced hallucinations chemical. In the final part of this paper I want to consider a third kind of level – the level of interventions that could change a phenomena. Interventions meant to change or correct the behavior of multi-level systems can typically operate at many different heuristic levels, so the challenge is to figure out why some levels might be preferred.

Think for a moment about a program running on a computer. How may we alter its behavior? One way is to change the program, another is to change the hardware (say by adding memory), and a third way is to unplug it. The program's behavior is thus, in this sense, both a software phenomenon, a hardware phenomenon and an electrical phenomenon. We may say similar things about human behavioral phenomena. Say a man is suffering from acute anxiety. What level of intervention can change this? Perhaps he can be calmed by interpersonal interventions – talking to him, touching him, and so on. He could also perhaps self-soothe by various actions like exercising or engaging in a favorite activity. One could also administer a sedative or even – though let's hope not – knock him out with a blow to the head.

Here again, constraints can be found by considering phenomena that are problems, and figuring out which interventions could count as solutions. Knocking a person on the head may alleviate anxiety in the short term, but it is hardly a solution to an anxiety problem. If the aim of the intervention is to restore someone to a healthy or normal state, the object would be to find an intervention that would target the source of malfunction in the most specific way possible, with the fewest possible side-effects.

But this constraint, while significant, does not imply there is a unique level for an intervention that counts as a solution. For many psychiatric disorders both talk therapies and pharmacological interventions can improve functioning, helping restore a person's mood and behavior to something more like a normal

condition. This means that disorders like depression are in this third sense both psychological and chemical problems, because they admit both of psychological and chemical solutions. Contemporary treatment practices often involve a multi-pronged approach involving both talk-based and pharmacological interventions. To the extent that this multi-pronged approach is more effective than either talk-based or pharmacological interventions alone, it is even more appropriate to say that these disorders are both chemical and psychological.

The level of a solution will not always match the level of the causes that produce, underlie or sustain the problem. Take for example the case of various clinical symptoms caused by traumatic experiences. While it is possible to intervene at the same level of the traumatic experience – say by using desensitization therapy – it may be that the most effect approach involves pharmacological interventions.

Whether something is subject to an intervention at a particular level is dependent upon the epistemic situation, and particularly upon the availability of technologies for intervention. No doubt much of the reason that Montaigne interpreted impotence as a psychological problem was the fact that psychological interventions were the only interventions available to address the problem. Nowadays we have Viagra and Cialis. At the present time, pharmacological and other medical interventions are typically not powerful and specific enough to restore those with behavioral disorders to something like normal function, but it may be that this will change with time. Right now, we may be inclined to think of post-traumatic stress disorder (PTSD) primarily as a psychological problem – both in the sense that its causes are mediated through perceptual mechanisms (i.e., perception and recall of traumatic experiences) and that the best available therapies involve psychological interventions by therapists, groups etc. But perhaps in the future our capacity to surgically alter memories will be such that the offending experiences may be effectively excised. Were this to happen, PTSD would look more like a neurological problem.

Beyond the obvious epistemic and pragmatic factors like cost and availability of therapeutic techniques, what can be said generally about the criteria by which one decides which level intervention is “better” and hence, at what level the problem may be said to be? Two plausible candidates are that interventions should specific and stable (Waters, 2007; Woodward, 2010). The stability of a cause refers generally to the degree to which it has the same effect across a variety of background conditions. Stable causal relationships represent practically better opportunities for intervention because they are more reliable. Sup-

pose for instance that there is a drug that under some background conditions reduces anxiety while under other conditions greatly increases it. For instance, its effect might be highly sensitive to the level of some hormones with the brain. To the extent that these levels varied either within or across individuals, such a drug would not have a stable effect, and would not be an effective intervention. Relatedly, and more realistically, it appears to be a common feature of many pharmacological treatments of psychological disorders that their effectiveness decreases over time. An anti-depressant that had a positive effect on mood for some months might cease to be effective, at least at its current dose. To this extent, the pharmacological intervention is non-stable.

Specificity has, as Woodward has suggested, two related but distinct meanings. On the one hand, causes are specific to the extent that fine-grained changes in a cause will lead to correspondingly fine grained changes to an effect. Woodward uses the example of an amplifier's controls to illustrate. The power is a non-specific cause of the sound coming from the amplifier, while the volume dial is a specific one. The dial allows one to finely tune the volume, and is thus specific, in a way in which the on-off switch is not. Both the position of the power switch and the position of the dial are causally relevant to the volume, but only the dial controls it. Turning off the power with the power switch off to control the volume is analogous to knocking a person out to control their anxiety. We don't count this non-specific intervention of knocking someone out as a solution to their anxiety problem, because it did too much. Solutions require fine grained interventions that solve the problem and not more.

The second sense of specificity is connected to the idea of one-cause one-effect. For instance, one speaks of an antibiotic as specific to the extent that it targets only a limited number of bacterial strains. No cause, and no intervention ever has just one effect, but given some chosen set of variables, it is possible for interventions to affect more or less of them, and in the limit only one. One benefit of talk-based therapies is they can be more specific, in the sense that they can address cognitive and affective patterns surrounding very specific events, memories or behaviors. A therapist working with a patient can seek to manipulate a patient's response to specific memories or environmental cues in a way that currently drugs simply cannot. To the extent that the problem, whatever it is, responds to these specific treatments, that makes the problem more of a psychological than a chemical problem.

While considerations about specificity and stability go some way towards explaining choices for clinical interventions, it would be naïve to think that

these considerations fully determine those choices, and with it our understanding of the kind or level of the problems they address. Richard Lewontin points out that the preference for identifying individual-level micro-causes in biomedicine may say more about dominant political ideologies than about the causal structure of the world. As an example, he cites the question of the cause of tuberculosis:

It is certainly true that one cannot get tuberculosis without a tubercle bacillus... but that is not the same as saying that *the* cause of tuberculosis is *the* tubercle bacillus.... [T]uberculosis was a disease extremely common in the sweatshops and miserable factories of the nineteenth century, whereas tuberculosis rates were much lower among country people and in the upper classes. ...[W]e might be justified in claiming that the cause of tuberculosis is unregulated industrial capitalism, and if we did away with that system of social organization, then we would not need to worry about the tubercle bacillus (Lewontin, 1993, p. 42).

Lewontin's suggestion then is that this is not a microbial problem, but a social one.

A similar story can be told about the search for the causes of and solutions for the global obesity epidemic. While I have no reason to doubt the validity of the Crum et al's findings, the research does seem consistent with a pattern in biomedicine of looking for causes within us rather than in our environment. The research is interesting in part because it suggests possible partial solutions to obesity problems by manipulating labelling to affect individual's sense of satiety. But while this sort of intervention might well have an impact, this kind of research should not cause us to lose sight of the fact that the principle causes of the global obesity epidemic are likely changes to the food supply. Changes of diet that lead to the increased consumption of meat, fat, and processed sugars are doubtless major explanatory factors. Obesity is in this sense a social problem, and it is quite likely that the most effective solutions would be social ones that changed the character of the food supply. But to do this would require challenging the political and economic power of the food industry. How much easier to just call it a psychological problem!

The world is replete with phenomena that are the result of the operation of multi-level mechanisms. My aim here has been both to offer an account of how to understand inter-level causal claims in these mechanisms, and also to suggest some of the epistemic and pragmatic features that lead us to pick out certain

of these levels as particularly explanatorily salient or as appropriate areas for causal intervention. We've found that there are criteria grounded in the causal structure of multi-level mechanisms that can provide rationales for ascribing levels to problems, their causes and their solutions. But these constraints are not sufficient to explain actual choices or to work as norms. For that we need the assistance of those who study science and medicine as social phenomena.

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