# Memory Consolidation, Multiple Realizations, and Modest Reductions

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This article investigates several consequences of a recent trend in philosophy of mind to shift the relata of realization from mental state–physical state to function-mechanism. It is shown, by applying both frameworks to the neuroscientific case study of memory consolidation, that, although this shift can be used to avoid the immediate antireductionist consequences of the traditional argument from multiple realizability, what is gained is a far more modest form of reductionism than recent philosophical accounts have intimated and neuroscientists themselves have claimed.

**1. Introduction.** Biological organisms, from fruit flies to human beings, have the capacity to learn—to undergo "lasting alteration[s] in behaviour or in . . . behavioural potential, due to . . . behavioural experience" (Dudai 2002, 140). *Learning*, generally defined, is one psychological capacity that organisms with radically different constitutions share in common. In philosophy of mind, such observations have traditionally been taken to indicate the multiple realizability of the mental and its irreducibility to the physical (Putnam [1960] 1975, [1967] 2002). However, contemporary proponents of reductionism continue to argue for the reducibility of psychological capacities or functions (e.g., Polger 2003; Kim 2005), with learning serving as the paradigm case for the reductionist cause (Bickle 2003, 2006). Given the aforementioned facts about learning and the support that they lend to the multiple realizability thesis, how can such tenacity to reductionism be justified?

In this article, I investigate one justification that is part of a recent

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trend in philosophy of mind to shift the traditional relata of realizability from that of mental state–physical state to that of cognitive function–physical mechanism in order to avoid the traditional implications of the argument from multiple realizability.[1] Shapiro (2000, 2004) develops the conceptual foundations of the new relata framework. Polger has such a shift in the relata of realization in mind in making a recent "plea for mechanism" (2003, 209). However, this shift is not exclusive to the philosophy of mind, as it is implicit in neurobiological approaches to the study of learning and memory consolidation. And although Bickle (2003, 2006) never makes an explicit argument against Putnam on the basis of the legitimacy of shifting the relata of realization, the form of reductionism that he advocates can only be achieved if he makes this conceptual shift.

The aim of this article is to investigate the precise nature of the conceptual shift and the consequences of its adoption for reductionism. In Section 1, I contrast the old (Putnam [1960] 1975, [1967] 2002) and new (Shapiro 2000, 2004) relata of realization and the adjoining multiple realizability/multiple realization hypotheses. I suggest that, in order for the new framework to be readily applicable to capacities exhibited by biological systems and to serve to differentiate "significant cases of multiple realization from trivial ones" (Shapiro 2004, 68), it must be complemented by recent insights concerning functional analysis and mechanism discovery put forward in the philosophy of science (e.g., Bechtel and Richardson 1993; Machamer, Darden, and Craver 2000; Craver 2001; and Craver and Darden 2001).

In Section 2, I apply both frameworks to the case study of memory consolidation. My intent, in part, is to get clear on what I take to be two separate claims for reductionism made both by neuroscientists (e.g., Squire and Kandel 2000; Barco, Bailey, and Kandel 2006) and philosophers of neuroscience (e.g., Bickle 2003, 2006).[2] These claims correspond directly to the two frameworks. I argue, in agreement with Aizawa (2007), that, on Putnam's relata of realization, the argument from multiple realizability goes through and the adjoining reductionist claim is defeasible. I then demonstrate that a second reductionist claim that corresponds to Shapiro's framework may be established but that the result is far more modest than Bickle and others intend. I conclude by attempting to draw out of the case study some residual problematic issues that we may encounter

1. The idea for this shift is not new, having been proposed by Elliot Sober (1999). It is Sober who draws the distinction between 'synchronic' and 'diachronic' explanation.

2. The types of reductionist claims expressed by neurobiologists pertain to a set of 'unifying ideas' (Barco et al. 2006, 1529) that suggest that the same intracellular molecular cascades could be operative in the consolidation of different kinds of memories (e.g., implicit and explicit) across different memory systems and different species.

in adopting the new framework. I suggest that the consequences are more grave than simply involving an inability to reduce *qualia*, as both Shapiro (2004) and Kim (2005) have suggested.

**2. Relata of Realization: Old and New.** In proposing his version of *physicalism* in "Sensations and Brain Processes," Smart makes three claims about the relationship between the mental and the physical: (1) organisms are composed exclusively of physical particles, (2) mental processes/states are identical with brain processes/states, and (3) human consciousness, like human behavior, will eventually be explicable exclusively in terms of "physico-chemical mechanisms" (1959, 142).

In both "The Nature of Mental States" ([1967] 2002) and "Minds and Machines" ([1960] 1975), Putnam introduces *machine functionalism* as *the* rival hypothesis to Smart's physicalism. To contrast his model of the organism with that of Smart's, Putnam draws an analogy between organisms and Turing machine computing devices. He accepts that both types of systems have a physical organization; computing devices are made out of such things as silicon chips and wires; organisms contain cells and molecules. Then he suggests that just as the physical components of a computing device endow it with the capacity to run or instantiate (machine) programs, so, too, do the physical components of an organism endow it with the capacity to run mental functions or programs. For Putnam, when a machine or an organism runs a given program, it can be characterized as in a *functional state* relative to a set of inputs, its other functional states, and a subsequent set of outputs. Organisms can be viewed as having similarly abstract functional states understood by the causal relationships they bear to (1) sensory inputs, (2) other functional states, and (3) the motor outputs of the organism.

With this model of the organism, Putnam has the tools requisite to attack Smart's identity thesis. Although he accepts, for lack of a better set of descriptive terms, the idea that organisms can occupy different "physicochemical states," he construes the relationship between physicochemical and functional states as one of "realization" (Putnam [1967] 2002, 75). Putnam's notion of realization has been interpreted as a constitutive relationship between physical and mental states. For example, Wilson takes Putnam's view of realization to include two hypotheses: physicochemical realizers are (1) "metaphysically sufficient for the properties or states that they realize" (2001, 4) and (2) "exhaustively physically constituted by the intrinsic physical states of the individual whose states or properties they are" (2001, 5).

Once Putnam articulates the relationship that holds between physicochemical and functional states, he then raises the possibility that two creatures with different physical constitutions could both be in the same

functional state yet be in different physicochemical states. On his example, both a sea mollusk and a human being could be described as "being in pain." Yet, in the human case "C-fiber firing" would realize that state, whereas in the sea mollusk a correlate of C-fiber firing would realize it. Putnam takes the possibility that functional or mental states can be *multiply realized* at the physical level as ruling out Smart's identity hypothesis. As Putnam claims, the brain-state theorist is required to show that a functional state across its many instances is identical to the same physical state. As Fodor (1974) demonstrates, multiple realizability rules out the possibility that bridge laws can be established that express identities between mental state terms, and as a consequence also rules out the possibility of explaining the wide array of mental phenomena consistent across the animal kingdom in terms of a simple set of physical principles.

In both "Multiple Realizations" (2000) and *The Mind Incarnate* (2004), Shapiro suggests that the realization relationship between the mental and the physical should be construed as a causal relationship holding between mechanisms and functions rather than as an identity relationship holding between mental and physicochemical states. He uses an example of two corkscrews to illustrate this distinction. A corkscrew is a functional kind in so far as it has the function of removing corks. Corkscrews can be made out of different physical constituents, such as steel or aluminum. They also come in different colors and shapes. Shapiro points out that if we apply Putnam's view of the relationship between the mental and the physical to the case of corkscrews, even two waiters' corkscrews that differ only in terms of physical composition or structure will be taken as different realizations of corkscrew. However, he suggests that such differences are trivial precisely because they have no impact relative to the *realization of the function* of a corkscrew. On his view, the only differences that should matter to us when we engage in a comparative analysis of functional kinds like corkscrews are those constituents or properties that make a difference in how the function (e.g., cork removal) is carried out. The way that we can get clear on such differences, according to Shapiro, is to subject the objects of our inquiry about realization to a Cummins's (1975) style functional analysis.

Shapiro suggests that the world can be carved into things that are simply "defined by some capacity," namely, "functional kinds" (2004, 46). Nonbiological examples of functional kinds of the type he has in mind abound: corkscrews, calculators, and watches are common examples. On Shapiro's interpretation, to conduct a Cummins's style functional analysis of a functional kind is to decompose that kind into those physical constituents and their properties that occupy the causal roles relevant to bringing the function about. So, when we undertake a functional analysis of two waiters' corkscrews differing only in terms of composition, one made of aluminum,

the other made of steel, and we abstractly *reconstruct* and describe how cork removal is brought about using each one, Shapiro claims that we encounter no real difference between the two because they realize the function of cork removal in an identical way—by means of a single lever. However, if we engage in functional analyses of a winged corkscrew and a waiter's corkscrew, we discover that cork removal is achieved differently in the one compared to the other; for example, one brings the removal about via a single lever, the other by two levers working in combination. Shapiro takes these differences in how the function is achieved in the two cases to constitute different realizations of the function of cork removal and, consequently, different realizations of corkscrew.

While it is not difficult to intuitively grasp his notion of realization with respect to the corkscrew example, Shapiro never clarifies how detailed a functional analysis must be, how superficial it is permitted to be, and relatedly, how much detail is requisite to describe either a function or a realization for the purposes of contrasting them. Furthermore, although it is obvious that a realization of a function ends when the function has occurred, it is not clear at which point the realization starts and where the functional analysis is supposed to begin. Perhaps it could be made clear in the case of a corkscrew. For instance, we could begin our description at the point at which the corkscrew is placed on the lip of the bottle. Then, we could detail each of the mechanical steps involved in removing the cork from the bottle as if providing a set of instructions that could later be used to instruct or explain to others how to remove a cork from a bottle. We could compare such a set of instructions for one corkscrew to that of another and decide whether and at which points the overall mechanisms differ. Shapiro, however, is interested in psychological capacities or functions such as attention, cognition, and perception—capacities realized by nervous systems. The interesting question, then, is how similar comparative analyses of the realization of functions will work with respect to systems in which the functions and realizers of interest are inside a black box, as it were, whose parts are not as obvious as those of a corkscrew.

Shapiro needs to provide a field guide for identifying functions and mechanisms with respect to biological systems in a way that will enable a determination of when two mechanisms are identical or different realizations of the same function. Although he makes some suggestions in this general direction, the details are not robust enough for the kinds of functions he is ultimately interested in. This may turn out not to be a serious problem, however. There is a wealth of recent philosophical literature on scientific discovery that bears on the very issues of functional analysis and the identification of causal mechanisms in biological systems. Details from this literature may be readily borrowed to enhance Shapiro's

account and to provide the criteria requisite for comparative analyses of functions and mechanisms. In what follows, I aim to do just this. I want to note, however, that providing a complete and detailed synthesis of the available ideas in the literature with Shapiro's conceptual framework is beyond the scope of the current article. I intend only to lay the groundwork for such a project by gathering some additional conceptual tools that make Shapiro's account more readily applicable to a neurobiological case study.

Psychological functions have historically been identified by studying the observable behaviors of organisms in natural and unnatural environments (i.e., laboratories), presenting them with different types of stimuli, depriving them of certain kinds of stimuli, removing their components (e.g., lesioning studies, gene knockouts), or introducing stimuli directly into them (e.g., pharmacological agents). On a classic account of functional analysis relative to biological and psychological systems (Bechtel and Richardson 1993), the successful ascription of a function to a system requires not only *decomposing* the system into its components but also *localizing* the function relative to those components. Shapiro has both ideas in mind when he talks about functional analysis. As the corkscrew example indicates, determining sameness and difference of function of a given kind is tied directly to an investigation of what the components of the thing are and how they are put together. Such features provide some indication of how those components may realize the function. Still, we require a more robust notion of mechanism to get a grasp on how to describe how those components work in sequence to bring about the function.

One definition of *mechanism* recently introduced into the literature by Machamer, Darden, and Craver (2000) may be used to bracket functions relative to systems and to put constraints on the identification of mechanisms in a way that is beneficial to determining sameness and difference of mechanisms. On this definition, *mechanisms* are "entities and activities organized so that they are productive of regular changes from start or set-up to finish or termination conditions" (Machamer et al. 2000, 3). Functional analysis in turn, involves the identification of where a mechanism begins and those intervening entities and activities involved in the production of the function of interest in which the mechanism terminates.

When we engage in a comparative analysis of different mechanisms that have been identified to realize the same function, we are required to investigate those mechanisms across specific parameters (cf. Craver and Darden 2001 on constraints on mechanisms). For instance, it may be that the *timing* of a mechanism—the duration of the activities of one or more of the entities that are operative from set-up to termination conditions—differs between two systems that are taken to realize the same function.

To take an example, it may be that the activation of a specific protein kinase involved in a metabolic process in one system has a different duration in another despite sameness of function. It may be that certain components—either the entities or the activities in which they engage—may be slightly different between two systems that realize the same function despite other marked similarities between them. For example, a molecule activated during classical conditioning in a sea mollusk may activate two downstream intracellular targets, whereas during classical conditioning in a fruit fly, it activates only one. If the realized function is the same form of learning in both cases, are the mechanisms similar enough to be considered identical realizations or not? Similarly, the components and activities of the mechanisms of two systems may be identical, but where they occur spatially in the system may differ. A set of biochemical experiments may reveal that the same protein phosphorylates the same downstream target during the induction of long-term potentiation in both the rat's hippocampus and amygdala. However, in one case it may turn out that the molecule achieves this phosphorylation by relocating into the cell nucleus, whereas in the other the phosphorylation is achieved in the cytoplasm. Would this constitute a difference in mechanism? Clearly, investigating the various parameters that constrain how the components in a given mechanism operate may be relevant to determining whether two mechanisms are identical or different. With the aforementioned distinctions in mind, I turn now to an analysis of the case study.

**3. Memory Consolidation and the Relata of Realization.** Even on a very narrow definition of *learning* as "the acquisition of an altered behavioral response, due to an environmental stimulus" (Sweatt 2003, 3), it is a capacity that is observed to be present across diverse biological taxa. A dominant assumption in contemporary neuroscience is that, when an organism learns, a memory for the event is stored in its nervous system as a long-lasting change in communication between pre- and postsynaptic neurons that communicate across a synapse (Hebb [1949] 2002). The process during which such changes in communication become stable is referred to as "memory consolidation," and it has been shown to require the activation of intracellular molecular signaling cascades in those pre- and postsynaptic neurons that are activated during learning events in a variety of model systems.

Bickle (2003, 2006) has described in painstaking detail neurobiological experiments on learning and memory undertaken in model systems including fruit flies, sea mollusks, rats, and mice. His aim has been to show that memory consolidation is a paradigmatic example of the reduction of the psychological to the physical. For my purposes, I will only consider two types of experiments here on two forms of learning: (1) *sensitization*

of the defensive gill-siphon withdrawal reflex in the sea mollusk *Aplysia Californicum* (*Aplysia*) and (2) *contextual fear-conditioning* in the rat. I describe the empirical findings from such experiments in only enough detail to achieve an adequate comparison of the implications of Putnam's and Shapiro's views of the relata of realization. In the process, I will consider how evidence from such experiments bears on two types of reductionist claims that have been put forward by Bickle (2003, 2006) and neuroscientists (e.g., Squire and Kandel [2000]; Barco et al. [2006]) that directly correspond to Putnam's and Shapiro's relata of realization, respectively. The first claim concerns the idea that the consolidation of memories in the case of both simple and complex forms of learning in simple and complex systems involves the same molecular *states* or *elements*. The second claim pertains to the idea that memory consolidation involves the same molecular *mechanisms* across species.

In sensitization experiments on *Aplysia*, a tactile stimulus is applied to the siphon, a fleshy area on the underside of the organism, which results in a subsequent moderate (baseline) elicitation of a defensive reflex, the gill-siphon withdrawal reflex. A noxious stimulus—a shock—is then applied to the organism's tail four or five times. For up to 2 days afterward, application of the tactile stimulus to the siphon alone is followed by a more pronounced and enduring reflex compared to baseline. Kandel and colleagues (e.g., Pinsker et al. 1973; see Kandel 2001 for a comprehensive bibliography) discovered that during sensitization the connection between a presynaptic sensory neuron originating in the skin of the siphon and a postsynaptic motor neuron that innervates the gill (its external respiratory organ) is strengthened.

Kandel and colleagues have made several neurophysiological and biochemical findings about sensitization in *Aplysia* (see Squire and Kandel 2000; and Kandel 2001). First, strengthening between sensory and motor neurons during sensitization is mediated by modulatory interneurons that use the neurotransmitter serotonin (5-HT). These interneurons synapse onto a sensory neuron. During learning, an increase in 5-HT is released into the synaptic cleft and binds to metabotropic G-protein coupled receptors on the sensory neuron. Activation of these receptors is followed by the phosphorylation of three molecules in the sensory neuron that comprise a molecular signaling cascade: cyclic adenosine monophosphate (cAMP), protein kinase A (PKA), and cyclic adenosine monophosphate response element binding protein (CREB). If the phosphorylation of any element in this cascade is blocked, sensitization of the gill-siphon withdrawal reflex does not occur. In other words, *Aplysia* does not learn, and the memory is not consolidated.

In a typical fear-conditioning experiment, a rat is placed into a chamber and allowed to explore and habituate to the context. A tone is presented,

and the rat's response is recorded and taken as the baseline response to the tone. At a later point, the tone is presented and immediately followed by a foot shock delivered through an electric grid in the chamber floor. Sometime later, the tone is presented again, this time alone, and the rat's response is recorded. A freezing response (i.e., the rat displays fear-related behaviors such as immobility) to the tone, compared to the baseline response, is taken as indicative of a learned association between the tone and the shock.

LeDoux and colleagues (Farb and LeDoux 1997; Schafe and LeDoux 2000; see also Rodrigues, Schafe, and LeDoux 2004) discovered that the neural circuit involved in this form of fear conditioning involves a presynaptic thalamic auditory neuron that synapses onto a postsynaptic neuron in the lateral area of the amygdala, a brain structure taken to be involved in learned fear. Thalamic auditory neurons use the neurotransmitter glutamate.

LeDoux and colleagues (Schafe and LeDoux 2000) have made several biochemical observations with respect to this synapse in fear conditioning. First, an increase in levels of phosphorylated PKA and CREB occurs. However, in contrast to sensitization in *Aplysia*, this increase takes place in postsynaptic neurons in the lateral amygdala. Second, when phosphorylation of PKA is blocked pharmacologically, the rats do not elicit a freezing response subsequent to the tone-shock pairing; that is, they do not learn the association between the tone and shock, and the memory is not consolidated.

Bickle has used evidence from the aforementioned types of experiments to argue that "there is a "physical-chemical state," the cAMP-PKA-CREB molecular biological pathway, which uniquely realizes memory consolidation across biological classes, from insects to gastropods to mammals" (2003, 148). Yet, when we apply Putnam's framework to the case of memory consolidation, we arrive at a different conclusion.

In order to apply Putnam's framework to memory consolidation, we have to admit to the possibility that this function can be parsed into separate functional states. Bickle claims that we are entitled to parse it as it has been historically parsed in psychology—as involving an initiation stage that precedes a consolidation phase ("the memory consolidation switch" (2003, 43). It is during this initiation stage across all forms of learning in all biological organisms, Bickle claims, that cAMP, PKA, and CREB are activated—so the initiation state of memory consolidation is identical with the activation of these three molecules. Yet, even if such identity is admitted, it amounts to a more modest claim than the one that Bickle intends. Whereas he claims that "memory consolidation" has been reduced to the molecular level, he is only entitled to claim that "memory consolidation initiation" has been reduced.

However, Aizawa (2007) has brought attention to biochemical findings that indicate that the amino acid sequences that underlie PKA and CREB differ across biological taxa. On Putnam's relata framework, it is enough to show that such physicochemical differences are present in order to refute the identity theorist, and Aizawa uses this as a basis to claim that memory consolidation is, contrary to Bickle's claim, multiply realized. Although I agree with Aizawa that evidence of structural differences at the level of amino acid sequences is sufficient to trump Bickle's claim of reduction, Bickle's argument does not have to end here. He can appeal to Shapiro's relata to make his case. He is entitled to do this by virtue of an additional claim that he makes about psychoneural reduction of the kind exhibited in the memory consolidation cases. Specifically, he characterizes "psycho-neural reduction in contemporary neuroscience" as "illustrated by the "structuring of psychology's purely functional posits into specific sequences and combinations of cellular and molecular entities, processes, and *causal interactions*" (Bickle 2003, 102). So, Bickle's alternative claim about memory consolidation may thus be formulated: activation of the cAMP-PKA-CREB signaling pathway *brings about* memory consolidation identically across diverse biological taxa and different forms of learning. Given this alternative formulation of the nature of the relationship between memory consolidation as a function and those mechanisms that realize it, he is at liberty to defer to Shapiro's relata framework. I think that there is good reason to believe that he does defer to it implicitly, given his persistent belief that memory consolidation is the paradigmatic case of psychoneural reduction.

On Shapiro's framework, *memory consolidation* can be understood as a function or capacity of nervous systems. The cAMP-PKA-CREB pathway may be regarded as a component of the mechanism that brings about memory consolidation across species. As I mentioned, when it comes to biological systems, we first need to be able to identify where a function begins and where it ends. As Machamer et al. (2000) claim, mechanisms have starting conditions and termination conditions, and we need to isolate both in order to bracket off a function like memory consolidation. It is difficult to say precisely where memory consolidation begins, but deferring to the neurobiologists, we could say that it begins when stimulus patterns are presented to an organism and ends when learning has occurred. Yet, if this is true, when we analyze memory consolidation functionally, even on Shapiro's account we will be interested in events both upstream and downstream of activation of the cAMP-PKA-CREB pathway. For example, we may be concerned with the types of neurotransmitters and receptors involved upstream of activation of the pathway and the consequences that its activation has for downstream changes at the synapse that must occur in order for the function of memory consolidation

to be realized at the level of an organism's behavior. Memory consolidation requires the activation of different neurotransmitters in the two systems: in *Aplysia*, serotonin (5-hydroxy-tryptamine, or 5-HT) is the transmitter mediating memory consolidation; in the rat, it is glutamate. In *Aplysia*, the relevant activation of the cAMP-PKA-CREB pathway occurs in the presynaptic neuron, whereas in the rat it occurs in the postsynaptic neuron. If we consider some simple differences between memory consolidation in Aplysia and the rat, we are forced to conclude, even on Shapiro's framework, that the mechanisms that realize memory consolidation are not identical.

That said, the reductionist is entitled to one more move. Bickle could posit the more modest claim that the function of "memory consolidation initiation" is brought about by activation of the cAMP-PKA-CREB pathway across different forms of learning and different species. He could offer a *narrow* identification of the starting conditions of the function, bracketing it at the point in time when PKA becomes activated in intracellular compartments on one end with the realization of CREB activation serving as the termination point of the function on the other. I want to say for the sake of argument that as long as such activation is occurring intracellularly, it does not matter in which neuron, pre- or postsynaptic, it is occurring. In addition, I want to accept the claim made by neurobiologists that differences in the amino acid sequences that underlie PKA and CREB are irrelevant to the realization of the function. Then, on Shapiro's framework, memory consolidation is identically realized in the two cases. In fact, Shapiro's framework nicely captures what neurobiologists have in mind when they argue for the reduction of memory to molecules—for all intents and purposes, relative to their explanatory interests, when it comes to memory consolidation, the cAMP-PKA-CREB signaling cascade is identical across all species and forms of learning.

However, even if we limit our functional analysis of the cAMP-PKA-CREB signaling pathway to intracellular compartments in the two forms of memory consolidation under consideration, there is no guarantee that we will arrive at the conclusion that the mechanisms by which memory consolidation initiation is achieved are identical. Although parametric studies of the kind I am interested in relative to the mechanisms of memory consolidation have yet to be undertaken extensively in contemporary neurobiology, it is possible that investigating even the temporal and spatial features of the activation of cAMP and PKA during memory consolidation initiation will yield differences across species and forms of learning in terms of the duration of the activation of these molecules and where they are operative spatially within cells. Such considerations bring us to the issue of how extensive a functional analysis must be and, consequently, how complete a description of a mechanism must be in order for us to

be able to say, on Shapiro's framework, when two mechanisms are identical or different.

**4. Conclusion.** I take the conclusions reached in this article to converge on a common theme, namely, that the recently suggested paradigm shift in the relata of realization in the philosophy of mind falls short of its intended goals. Only if cognitive functions and the mechanisms that produce them are defined very narrowly will they be reducible to the mechanisms that produce them.[3] Yet, if this is true, it is a form of reductionism that is far more modest than what proponents of the shift have intended.

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3. This corresponds to a point made by Bechtel and Mundale (1999), who claim that the success of Putnam's argument from multiple realizibility can be attributed to the fact that he defines mental states broadly but the physical states that realize them very narrowly. As I am pointing out here, a similar argument may be made against reductionists like Bickle who have to define functions and mechanisms very narrowly in order to get reduction to go through. Yet, even on very narrow descriptions, reduction may not always go through.

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