CHAPTER TWO
REDUCTION-IN-PRACTICE IN CURRENT MAINSTREAM NEUROSCIENCE

Most philosophers of mind and many cognitive psychologists still doubt that "genuinely cognitive" psychological theories will reduce to neurobiological counterparts. As I emphasized in Chapter One, this attitude contrasts starkly with the reductive aspirations of "mainstream" cellular and molecular neuroscientists. My first substantive task in this book is to characterize "reduction-in-practice," as it is generating experiments, results, and explanations in current mainstream neuroscience. For reasons sketched in the previous chapter, I'll ignore at first the concerns typically assumed by philosophers to occupy center stage in discussions of psychoneural reduction, namely philosophical anti-reductionist arguments and the problems of characterizing a concept of intertheoretic reduction for science generally. Instead, I'll begin by presenting a detailed example, recent discoveries about the molecular mechanisms of long-term potentiation (LTP), an important type of experience-driven synaptic plasticity, and the behavioral data these mechanisms explain. This story is an accomplished neurobiological reduction of psychology's "memory consolidation switch" that mediates the conversion of short-term to long-term memories. These recent scientific details show the nature of reduction at work and succeeding in current cellular and molecular neuroscience. Later in this chapter, as a piece of new wave metascience, I'll generalize from this example, hopefully to provide a template for additional psychoneural reductions.

There is a second reason why the details of this example are important for philosophers and cognitive scientists. There is a widespread misconception about the content of "neuroscientific explanations" of behavior. Many still think of these explanations on the model of what neuroscience provided three decades ago, namely, circuit diagrams of anatomical connections between neural regions known to subserve specific functions, with some molecules (e.g., neurotransmitters) indicated in a few spots. Indeed, there is a widespread misconception that "we don't know much about how the brain works." This misconception ignores completely the "molecular-biological wave" that began sweeping through neuroscience more than two decades ago. I gave sociological data in support of this trend in Chapter One and there suggested that more than sociological justifications for it could be offered. Now I want to make good on the latter assertion by presenting an example of a detailed neuroscientific investigation, showing how this approach has begun generating novel *behavioral* experiments, results, and explanations. This example is intended to demonstrate clearly the character of mainstream neuroscientific investigations in the present and the foreseeable future. Finally, it will show that we do know a lot about "how the brain works," at least its fundamental components, and how we can manipulate these components to produce specific, measurable behavioral effects. Even if philosophers, cognitive scientists, and cognitive neuroscientists find reasons to reject the "ruthless reductionism" I urge from this current example, they will at least see some details about actual practices in mainstream neuroscience circa the beginning of the 21st century.

1 A PROPOSED "PSYCHONEURAL LINK"

Much has been written over the past twenty years about a "learning and memory—long term potentiation (LTP) link" And much of this scientific and theoretical literature has been misunderstood. Not even its most strident proponents think that LTP is *the* cellular/molecular mechanism for all "forms" or "types" of memory (although their writings sometimes give this mistaken impression.) About the "simple proposition" that "LTP is a substrate of memory," neurobiologist Gary Lynch has written recently:

> Substituting the definite article in this proposition converts it to a form that is manifestly *not* true (it is easily shown that forms of memory exist that do not involve LTP), and yet it is widely employed, perhaps unwittingly, in LTP/behavior arguments. These difficulties provide ample reason to be pessimistic about the still-popular search for an *experimentum cruces* that "proves" or "disproves" the role of LTP in memory. (2000, 139).

Lynch has been among the most forceful and influential proponents of a "learning and memory-LTP link" for more than two decades.

Memory-like features of LTP have been explored vigorously since its discovery in the early 1970s. These include its

- *selectivity*: It only affects transmission efficacy at specific synapses on a given neuron.
- *cooperativity*: Larger effects and more stable induction occur when more afferent fibers (input lines to the neuron) are stimulated.

- *multiple forms*: Different molecular mechanisms underlie its variable stability over time, reminiscent of the short-term/long-term memory distinction.
- *cumulative nature*: Successive episodes of high-frequency stimulation in the same afferent fibers produce increasing amounts of synaptic potentiation.
- *regional distribution*: LTP has been documented in synapses in mammalian cortex, hippocampus, other "limbic" structures, and spinal cord (there called "wind up"). All of these areas have been associated behaviorally with important types of long-term memory.[1]

Attempts to correlate the temporal dimensions of these synaptic features with those from behavioral studies of memory have been prominent since LTP's discovery. The advent and dominance of molecular techniques and investigations in mainstream neuroscience over the past two decades have influenced LTP research strongly. Work on the molecular mechanisms of LTP and attempts to relate these to measurable memory-guided behavior are at present among the hottest and best-funded research areas in all of science. Some decry this fact, but no one denies it. Since I want a detailed example that reflects accurately the aims, goals, and aspirations of current cellular and molecular neuroscience, this research program is a clear choice.

I should confess at the outset to finding this program and its mechanisms to be among the most beautiful research in contemporary science. I am inspired by it and what I see as its explanatory veracity and potential. Accuracy and aesthetics aside, however, my purpose in describing it is to present a paradigmatic example of an accomplished psychoneural reduction, and hence a template for additional reductions. Like it or not, this example illustrates the force of reductionism—including its ruthlessness—at work in current cellular/molecular neuroscience.

However, I am discussing a limited proposal: that memory *consolidation* has been reduced ("linked") to the molecular mechanisms of LTP. This is the extent to which a reduction is on offer, at least in the serious scientific literature, and we'll even see in this chapter and the next a number of distinctions that have been drawn between types of memory systems. Scientists virtually never assert a global "memory is LTP" hypothesis in primary research publications. Incidentally, the limited scope of this proposal reveals errors in one of Schouten and de Jong's (1999) criticisms of my previous appeal to this example as an accomplished new wave psychoneural reduction. They write: "The functional characterization of associative, Hebbian learning in terms of weight changes of synaptic connections has driven LTP research by staging certain features of learning and memory. The molecular account would have to provide the necessary and sufficient conditions for these features if a reductive explanation was to be achieved" (1999, 249). They next point out that LTP is found in a variety of brain systems, including some that apparently have nothing to do with learning and memory. They cite approvingly the list given in McEachern and Shaw's review (1996, section 9.1) and the lesson urged there: "LTP ... serves functions other than, or in addition to, memory" (1996, p. 80, quoted in Schouten and de Jong 1999, p. 250). So the "sufficiency" condition fails. They also cite Saucier and Cain's (1995) study, where a potent and specific antagonist (blocker) of NMDA (N-methyl-D-aspartate) receptors, NPC17742 (2R,4R,5S-2-amino-4,5-(1,2-cyclo hexyl)-7-phosphonoheptano acid) completely blocked LTP induction in hippocampal dentate gyrus neurons. Nevertheless, rats treated with this receptor antagonist that had been made familiar with water maze task demands by non-spatial pre-training displayed normal spatial learning in the water maze as compared to pharmacologically untreated controls (Saucier and Cain 1995, Figures 2 and 3).[2] So LTP isn't necessary even for spatial learning and memory. Schouten and de Jong conclude, contra to my answer to the "put up or shut up challenge," that a memory-to-LTP reduction isn't yet accomplished, as these concepts now stand in psychology and neuroscience.

Two mistakes infect their criticism. First, the serious scientific hypothesis links only one process of memory—consolidation—to LTP. Second, no "necessity and sufficiency" requirements attach to reduction projects in cellular and molecular neuroscience. No serious LTP researcher is unaware of the results Schouten and de Jong cite. In fact, we'll see in the next chapter that the molecular mechanisms of LTP are not even unique to neurons, much less to neurons exclusively involved in memory consolidation! And yet the "memory consolidation-molecular mechanisms of LTP link" continues to thrive. Clearly, some other sense of reduction is at work than the one Schouten and de Jong (1999) find lurking in some neuroscientists' writings. It is this other sense that I want to bring to the surface by looking in some detail at the actual neuroscientific accomplishments and assertions, as they have been presented in the primary experimental literature (as opposed to review papers, critical surveys, and the like).[3]

2 TWO PSYCHOLOGICAL FEATURES OF MEMORY CONSOLIDATION

Consider first some feature of memory consolidation revealed by experimental psychology. These features are the explanatory targets of the reductionistic search for molecular mechanisms. Since the seminal research of Ebbinghaus, Müller, and Pilzecker more than a century ago, and then

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