

## **Neurofunctionalism Revisited: Learning is More Than You Think It Is**

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Studies of learning in simple systems (invertebrates and spinal cord) have revealed that organisms can encode stimulus-stimulus (Pavlovian) and response-outcome (instrumental) relations in multiple ways. It is suggested that nonassociative mechanisms contribute to learning and that there is value in adopting an approach that details the neural-functional mechanisms involved. Reactions to this approach are discussed. The link between the methods of Pavlov and associative ("true") learning is deeply ingrained and, some believe, should be maintained. We suggest that there is value in dissociating the concepts and seek to clarify the implications of a neurofunctionalist approach to learning. It is argued that a neural-functional approach provides a better framework for integrating behavioral and neurobiological observations.

For close to 15 years, we and our colleagues have examined some unusual forms of learning that do not fall within the traditional categories of learning theory. Building on a foundation laid by Thompson, Steinmetz, Patterson, and others (for reviews see Grau & Joynes, 2001; Patterson, 2001), we developed model paradigms that have now been shown to have considerable clinical significance (e.g., Grau et al., 2004). They have also proven amendable to uncovering the biological substrates of learning (e.g., Joynes, Janjua, & Grau, 2004; Liu, Crown, Miranda, & Grau, 2005). Indeed, with new discoveries within the field of spinal cord nociceptive plasticity, spinal systems now rival the hippocampus, amygdala, and cerebellum as one of the most well-characterized neurobiological systems (Ji, Kohno, Moore, & Woolf, 2003). But for 15 years, reviewers have questioned whether we were examining *true* learning, providing demerits for falling outside traditional categories. Oddly, no one questioned whether the examples of plasticity were clinically significant or had widespread implications, only whether the findings were relevant to learning. Frustrated by this continued assault, we have turned the question around and asked why learning should be so narrowly defined? Why should our examples of instrumental or Pavlovian conditioning be viewed in an inferior light just because we lack a Lockian association? Why is associative learning on the pedestal and blessed as the only true form of learning, and why have we inbred the concept with our methods of studying learning to such an ex-

We would like to thank the Editor for giving us the opportunity to question some basic tenets in the field of learning and for recognizing that such an enterprise requires commentary. We also want to thank those who provided the comments. We have known some of the commentators for many years. They agreed and disagreed with us in good spirit, and we hope that they accept our reply in a similar fashion. Finally, thanks are due to Kristy Acosta, Michelle Hook, Russell Huie, Marissa Maultsby, Christine Petrich, Denise Puga, and Stephanie Washburn for their helpful comments. Preparation of this paper was supported, in part, by Grants MH 60156 and NS 41548. Correspondence concerning this article should be addressed to James W. Grau, Department of Psychology, Texas A&M University, College Station, TX 77843-4235, U.S.A. (j-grau@tamu.edu).

tent that many find it hard to dissociate the concepts? In the process of considering these questions, we have knocked associative learning off its pedestal and questioned the way that we organize and present our data. Some applauded (Blaisdell, 2005; Sokoloff & Steinmetz, 2005; Staddon, 2005), acknowledging the timely nature of our critique. Others (Machado, 2005; Reilly & Schachtman, 2005) felt that the traditional story still reads well and sought to maintain the status quo.

Three of the five commentators agreed on two basic points: (1) the way we present the field to students and our colleagues is in need of revision; and (2) the field has not done a good job of integrating new discoveries on the neurobiology of learning. They generally shared our desire to expand the domain of learning, but struggled with how this should be accomplished. Their reactions ranged from an endorsement of our perspective to a more traditional view that maintains a special pedestal for true conditioning. The other two reviewers had no such struggle—to them, there is just one type of conditioning and it is associative in nature. Conditioning in the spinal cord and *Aplysia* fails on this criterion and is best swept out of the house of learning as a nonassociative artifact. We feel that this is the wrong tact. In the sections that follow, we attempt to clarify why we came to different conclusions. We begin by discussing a number of issues that appeared to stem from common concerns, seeking to clarify our position. We then deal with specific issues raised within each of the commentaries.

### **The Semantic Connection: Established Associations Make it Difficult to Dissociate Association**

Our aim in the target article was to challenge some long-held views. But challenging a paradigm requires that we convince the reader to temporarily suspend a well-established way of viewing the world and consider an alternative perspective. With a framework as well entrenched as the doctrines of learning theory, this is not always easy to do. The problem is that some concepts have been so strongly associated that it is difficult for us to see that a single linguistic token refers to two distinct entities. We believe that this issue arose with regard to the distinction between Pavlovian conditioning and associative learning. As Staddon reminded us, Pavlov saw a conditioned response (CR) as a response that was “conditional upon” the “history of pairing between the CS and the US. It was simply a descriptive term, nothing more” (p. 38). We use the term Pavlovian conditioning in just this sense, to refer to a kind of learning that depends on the relationship between two stimulus events. For many, though, the term Pavlovian conditioning has additional meaning, for it also implies a form of associative learning. The semantic confusion is natural. Pavlovian conditioning involves (by definition) two stimulus events (S1 and S2) that have a physical relation—the distal/proximal cues are physically associated. Add to this the long history of associative learning within philosophy and psychology, and the elegance and power of the concept, and Pavlovian conditioning becomes inexorably linked to the mechanism of associative learning.

If the only way a S1-S2 pairing could have a lasting impact was through the development of a new association, then it would make sense for Pavlovian conditioning and associative learning to be forever married. Given this, the first question we had to address is whether there are other viable mechanisms. Two

were identified: protection from habituation and pairing-specific enhanced sensitization. We also outlined several operations that could be used to distinguish protection from habituation from associative learning and provided evidence that our example of spinal conditioning relied on the former mechanism. Here was a case where learning depended on pairing two stimulus events, and the learning exhibited a variety of Pavlovian phenomena (e.g., latent inhibition, extinction, and overshadowing). Our conclusion was that Pavlovian conditioning is not necessarily associative in nature.

We then recognized that unlinking the concepts of Pavlovian conditioning and associative learning had a variety of implications that went well beyond our own data. If the concepts are disconnected, the methods of Pavlov lose some of their import. They still have value, but attention is shifted to detailing the underlying mechanisms. Further, what holds for Pavlovian conditioning would seemingly apply to other standard methods. Indeed, one could argue that such a view was already accepted in some circles. For example, researchers studying habituation have long recognized that an array of mechanisms can bring about a decrement in response magnitude and they do not appear to hold any one (not even the associative account) in greater esteem. Moreover, as noted by Staddon (2005), detailing the operational principles that constitute a functional mechanism can provide a better framework for linking behavior to neurobiological systems. Together, these considerations led us to conclude that the future of learning lies with a decreased emphasis on the methods of Pavlov and Skinner and an increased emphasis on the underlying functional mechanisms and neurobiology.

### **Associative Learning and the Ghost of a Straw Man**

Having unpacked our rationale for a broader approach to learning, we face the issue that dominated much of the commentaries. The issue concerns the extent to which the soul of learning is tied to the concept of association. Some suggested that few still hold this view—that it is a straw man (Blaisdell, 2005). If so, we are lucky indeed because we need look no further than the commentaries for evidence that the ghost of this straw man still holds sway.

There are actually two versions of the argument that learning is necessarily associative. The most general holds that nonassociative effects do not count as learning. Staddon (2005) maintains this position when he questions whether learning is essential. Our point was that researchers who have chosen to study learning view the process as providing a key adaptive capacity. Against this, Staddon notes that some simple organisms (protists) negotiate their environment with nothing more than the capacity for habituation. The implication was that learning is unnecessary because nonassociative habituation does not count as learning. We come to a different conclusion because learning for us is not limited to associative processes—nonassociative habituation counts as learning. (For a discussion of the criteria for learning see Grau and Joynes, 2001.)

The other version of the argument is equivalent to the view we asked the reader to suspend—that Pavlovian (or instrumental) conditioning is necessarily associative in nature. We seek to counter this belief, but again, one could question whether we are battling a straw man. Blaisdell argues this (last paragraph), but a few sentences later he suggests that some invertebrate preparations are superior to

Kandel's because they exhibit "true Pavlovian conditioning" (p. 26; for a discussion of how the results in vertebrate and invertebrate models compare, see Pittenger & Kandel, 2003). It would seem that the ghost of the straw man has once again wheeled his influence. Reilly and Schachtman (2005) adopt the view in full when they ask whether we should broaden "the domain of Pavlovian learning, or alternatively, should we simply acknowledge that we do not currently have ample control conditions to rule out all possible nonassociative (i.e., artifactual) effects? Grau and Joynes argue for the former, we prefer the latter" (p. 35). It would seem that many still see Pavlovian conditioning as married to just one mechanism—associative learning.

Why do we seek to broaden the domain of Pavlovian learning? To address this question, let's consider where Reilly and Schachtman's position would lead us. Examples of learning that involve pairing-specific enhanced sensitization are relegated to artifactual status. Add to this the nagging problem that many CSs have a nasty habit of generating a CR-like response prior to conditioning, and you may find your favorite preparation placed along the nonassociative curbside. The cost of preserving the unity between Pavlovian conditioning and associative learning would be a very narrow field of study. Next, what are we to make of the equivalent use of the terms? If the words have the same meaning, why have two terms? To this, it might be argued that there are different kinds of associative learning. For example, in some cases, Pavlovian conditioning seems to reflect a S-S association while in others it appears S-R in nature (Rescorla, 1975). This move breaks the circularity and broadens the sphere of influence because now Pavlovian conditioning refers to two distinct kinds of learning, S-R versus S-S. But those instances of S-R conditioning, so cleverly used to break the circularity, have a formal similarity to pairing-specific enhanced sensitization. It would seem that the circularity was broken by recreating a hierarchical scheme similar to that illustrated in Figure 4 of the target article.

Citing Rescorla (1988a), Reilly and Schachtman suggest that Pavlovian conditioning is much more sophisticated than we suppose, involving the capacity for abstracting informational value and representing hierarchical relations. It is, of course, true that some examples of Pavlovian conditioning have an underappreciated level of complexity. Recognizing that researchers in other areas often viewed conditioning as a low-level mechanical process, Rescorla outlined a series of findings that suggested greater sophistication. However, we do not believe (as Reilly and Schachtman seem to suggest) that Rescorla intended to provide a singular view of what constitutes conditioning. If so, then any example of Pavlovian conditioning that was insensitive to complex conditional discriminations would be deemed inadequate. The field of study would be narrowed even further, perhaps to those forms of learning that are hippocampally dependent. We are certain Rescorla did not intend this. Indeed, Rescorla noted elsewhere how S-S relations can be encoded in multiple ways and cites protection from habituation as an example (Rescorla, 1988b). Recognizing the complexity of some instances of Pavlovian conditioning does not negate the importance of simple model systems that may lack the capacity to exhibit occasion setting or mediated acquisition/extinction effects (Holland, 1990).

### Levels of Analysis

A number of commentaries raised issues concerning the relationship between different levels of analysis. Our intent was to show that shifting attention to the functional mechanisms that underlie learning has value. But we apparently made this push with such force that some perceived a more insidious intent—to effectively assassinate the superordinate category and call an end to detailed behavioral analysis. This was not our intent. We see behavioral studies as essential to detailing the efficient causes; to derive an accurate (hopefully, mathematical) description of the circumstances under which a phenomenon occurs and its ecological significance. Further, the delineation of new behavioral categories and their underlying relations will, of course, depend on detailed behavioral analyses. Our push was designed to encourage a shift in focus, from the usual tripartite (single stimulus learning, Pavlovian conditioning, and instrumental learning) to the underlying mechanisms, a shift we (and Staddon) believe is essential to uncovering the underlying neurobiological mechanisms. But such a shift in focus does not, in any way, eliminate the reality of the (superordinate) behavioral categories. Recognizing this, we listed Pavlovian conditioning as the superordinate category within Figure 4. In this scheme, the three functional mechanisms were depicted as subcategories of Pavlovian conditioning. Given that these relations were explicated in Figure 4, we do not understand why Machado would claim that we “failed to notice that” (p. 30) the functional mechanisms were subcategories of Pavlovian conditioning. It seems odder still that he would argue against our position by suggesting that the functional mechanisms “identify in greater detail how the CS and US arrangements in a particular case affected behavior; they do not show the inconsistency” (pp. 30-31) Machado perceives a rebuttal in language that summarizes some key features of Figure 4.

What implications does our approach have for the way we analyze instances of learning? To answer this question, it is helpful to think of the problem in terms of a two-stage process. The first issue concerns the superordinate category. Does the instance of learning represent an example of single-stimulus, Pavlovian, or instrumental learning? (It is assumed here that we have established that the behavioral effect qualifies as an instance of learning; see Grau & Joynes, 2001.) For Pavlovian conditioning, researchers have established a set of operations that can be used to demonstrate that the S-S relation matters. We will designate this set of operations as set “X” and assume that these conditions have been met. The next question concerns the nature of the underlying mechanism. Let us suppose that the example in question involves a case of protection from habituation. In Joynes and Grau (1996), a set of operations was derived (set “Y”) and used to show that this mechanism seems to underlie our example of spinal conditioning. Notice that the operations needed to classify the behavioral effect as an instance of Pavlovian conditioning (X) are not equivalent to those needed to classify the effect as a case of protection from habituation (X+Y). Given this, we are confused as to why Machado would question whether there is a “difference in the *logical* status of the two definition” (p. 31). The two sets of operations are not logically equivalent.

Does classifying a behavioral effect as a case of Pavlovian conditioning have mechanistic implications? Yes, of course it does. Within our framework it would suggest that one of three mechanisms is at work. Does Pavlovian conditioning have a biological reality? Again, of course it does. Neither here, nor in the target article, are we concerned with environmental relations in the absence of a processing organism. Nor are we concerned with stimulus events that the organism cannot sense. Pavlovian conditioning is of interest because it provides a sensible way of demonstrating that the *organism* is sensitive to a S1-S2 relation, independently of whether that sensitivity is due to associative or nonassociative mechanisms.

### **Views We Did Not Intend to Endorse**

In a number of instances, those commenting on our target article read into it meaning that we did not intend. For example, as discussed above, we did not mean to disparage the importance of detailed behavioral analysis. Another example arose in Reilly and Schachtman's commentary when they suggested that *mechanism* for us meant *biological mechanism*. As illustrated in Figure 4 of the target article, we did not intend this narrow meaning. As indicated, mechanism was used in reference to both biological and functional systems. We believe that our use of *functional* mechanism is similar to what Reilly and Schachtman have in mind when they refer to *psychological* mechanism. We prefer functional because psychological has connotations that we would like to avoid and because the term function fixes our attention on the most pressing issue (for learning theorists)—what the mechanism is designed to do. As Staddon clarifies, mechanism here concerns a theory of operation, and as he suggests, this is often the most useful meaning of the term. Further, this use of the term can be applied at multiple levels of analysis and we agree that specifying how the components at each level operate is key to deriving their relation.

Schematically representing different levels of analysis can pose a challenge. In both Figures 1 and 4, we highlighted the difference between functional and biological descriptions by presenting each at a different level with the constructs connected by arrows. As Staddon reminds us, both levels of analysis refer to the same anatomical substrate.

Our presentation sidestepped some complexities that require clarification. In discussing behavior, function, or neurobiological systems, we can frame questions at either a local or global level. As an example of a local effect, consider a functional/biological mechanism designed to prime behavioral responding when a stimulus is reencountered (a form of sensitization). This priming effect might be linked to the secretion of a particular neural transmitter. At the level of the behavior system, the release of this transmitter could enhance food directed behavior. The global function might be described in terms of arousal and appetitive drive, and the biological system would involve a widely distributed neural circuit and a host of brain regions. We mention these possibilities because many of our examples concerned local mechanisms and simple behaviors, a focus that fits well with the basic categories of learning. It should be recognized, however, that we do not see our approach as limited to such local issues—learning can involve a local

modification within a particular component (module) of a system or a restructuring of the network that defines the system.

We also need to clarify some issues concerning the type of modularity assumed. Following Timberlake and Gallistel (Gallistel, 1980; Timberlake & Lucas, 1989), we envision learning as occurring in a type of lattice hierarchy. Suppose that we have a system that is sensitive to S-S relations and has embedded within it the capacity for protection from habituation, pairing specific enhanced sensitization, or associative learning, with the relative contribution of each varying as a function of training and other variables. How do we envision these mechanisms being distributed within the nervous system? One possibility is that each type of learning is mediated by a distinct neural component and that each mechanism handles its respective function across a range of learning phenomena. For example, the associative system might handle the linking of representations for both appetitive and aversive USs. Similarly, another neural mechanism might provide the capacity for protection from habituation and so forth. In some cases, nature may provide such simplicity, but this is likely the exception rather than the rule—that in many instances, the capacity for a given type of learning (e.g., protection from habituation) is multiply represented across the nervous system.

What about within a particular neural/behavior system? Should we expect to find each type of learning capacity residing within a distinct component of the structure? Again, nature may occasionally simplify our analysis in this fashion, but we also anticipate more complex scenarios. For example, a single anatomical structure may be capable of all three forms of learning—what may vary is the neurochemical system engaged. In this case, dissecting their contributions will require a different methodology. Rather than the traditional neural lesion approach (used in cases where distinct modules are thought to reside in distinct anatomical loci), local application of various agents may be needed to biochemically manipulate the learning processes. In terms of both operation and neurochemistry, each form of learning may exhibit a form of independence, but reside within the same set of neurons. A further complexity arises from an inherent quality of a lattice hierarchy, for the same component may subserve many masters. Such complexity undermines the plausibility of simpler views of modularity that aspire to link discrete functions to particular anatomical substrates. Rather, the function of a unit will likely depend upon the system to which it contributes. We mention these alternatives because Staddon (2005) appeared to believe that we endorsed a simpler view.

We have pushed for a focus on mechanism over methodology. Does this necessarily imply that everyone needs to change the way they attack a problem within the laboratory? No, not at all. First, as mentioned above, detailed analysis of behavior is still needed, in part because some basic questions within the field of learning have yet to be addressed, and in part because we have only just begun to flesh out the details of how the component modules are brought together to form an integrated behavior system. Do we see any problems with researchers pursuing particular model paradigms? Again, not at all. Current paradigms are built upon a rich behavioral history, and there is much to be gained from this history and the capacity to compare results across laboratories. Indeed, much of what we currently know about the principles and neurobiology of learning has been derived using well-established model systems. We only become concerned when those who have adopted a particular paradigm become dogmatic regarding its benefits and

prejudicial in their evaluation of new paradigms. But as Sokoloff & Steinmetz remind us, learning theorists are not the only ones capable of becoming dogmatic. Neuroscientists too can become single minded. For example, some might worry that the focus on NMDA-mediated plasticity has led researchers to ignore other potential mechanisms.

### **Specific Rebuttals**

Beyond the general issues discussed above, each of the commentators raised a number of specific issues that require further attention. In the sections that follow, we respond to a selection of these issues. Due to space limits, we cannot address every comment, but instead, focus on those issues that are most central to our thesis.

#### ***Blaisdell: No Madness in Mechanism***

Blaisdell (2005) agreed with many facets of our target article, and that the “disconnection between facts and framework probably contributes significantly to the tendency for students to perceive courses on learning as difficult or uninteresting” (p. 23) However, he expressed concerns regarding the perceived disparaging of behavioral approaches. We agree that behavioral analysis is required to identify and describe the operational principles that guide learning. We believe that Blaisdell would agree that the most informative behavioral data are those that yield new insights into how the system operates, and if so, the implicit focus remains on the functional mechanism.

#### ***Sokoloff and Steinmetz: An Inclusive View of Memory Research***

Again, we found little to disagree with and feel that their comments helped to clarify a number of important issues. They also provided an interesting example of how multiple mechanisms can contribute to the encoding of a CS-US relation within an eyeblink paradigm and how learning within a more sophisticated system can sometimes usurp control over the process. Our only worry stems from the way such issues have been handled within the literature, where it sometimes appears that researchers hope to argue a mechanism out of existence. We expect that Sokoloff and Steinmetz (2005) would agree that a full description of the functional/neurobiological system must include all components.

#### ***Staddon: On Respondent and Operant Behavior***

Staddon (2005) too helped to clarify a number of issues discussed above. He also described an interesting property of habituation (rate dependency) which, as he notes, helps to make our point. He did, though, differ on a few issues.

One difference concerns the distinction drawn between instrumental and operant conditioning. The components of our argument were first outlined in Grau, Barstow and Joynes (1998) where we recognized a potential to overstate our claims. In that paper, we examined whether spinal cord systems are sensitive to a response-outcome (R-O) relation, the distinguishing feature of instrumental learn-

ing. Building on earlier reports (Buerger & Chopin, 1976), we provided evidence that spinal cord neurons are sensitive to R-O relations and discounted a non-instrumental reactive model (a mechanical system that does not encode the R-O relation). The overall pattern suggested that neurons within the spinal cord could exhibit a form of instrumental learning. Given that many treat the terms operant and instrumental as synonyms, it was tempting to conclude (as others have done) that we also demonstrated a form of operant learning. Yet, we were nagged by a problem with this reasoning. Skinner saw behavior as falling into two categories, respondent or operant. He viewed respondent behavior in reflexive terms, as a type of elicited response. This raised a dilemma for us because we suspect that Skinner would argue that our example of spinally-mediated instrumental learning represents a case of respondent behavior (because the effective reinforcer—shock onset [Grau et al., 1998]—elicits our target response, leg flexion). One implication of this (to us) was that instrumental and operant learning do not refer to identical constructs. Another is that Skinner seemingly had additional criteria in mind when he drew the distinction between respondent and operant behavior—the key difference does not appear to depend on the R-O relation alone. Indeed, it is not clear that Skinner would necessarily deny that a R-O relation can influence a respondent. More formally, sensitivity to the R-O relation may be a necessary, but not a sufficient, condition for classifying a behavior as a Skinnerian operant. If so, the distinction between operant and respondent must depend on additional criteria. Of course, there is the well-known quality of “emitted,” but we were reluctant to build a definition on the inability to identify an effective cue. It seemed to us more profitable to ask how our examples of spinal learning differed from instances of behavior that Skinner would have likely seen as good examples of operant behavior. Two factors were identified, both of which concerned the degree to which the behavioral effect was biologically constrained. In an ideal operant situation, we could train a variety of behaviors (e.g., an increase or decrease in the response) using a variety of reinforcers (e.g., appetitive or aversive). Of course, we recognized that no learning situation is ever completely free of biological constraints. Nonetheless, it is not too difficult to find cases of human and animal behavior that seem far less constrained than spinal learning. By this analysis, demonstrating instrumental learning requires a set of operations that show that the system is sensitive to the R-O relation (set “A”). Operant learning requires evidence that the R-O relation matters plus additional criteria (e.g., that neither the behavioral change nor the reinforcer are constrained; Grau et al., 1998). If the additional criteria are defined by set “B”, a demonstration of operant behavior requires A+B.

Thus, we agree with Staddon that behavior in traditional operant paradigms is often biologically constrained to some extent. Yet, we expect that Staddon would agree that instrumental learning within the spinal cord is less flexible and depends on an elicited response. If so, it would seem our example of learning has a respondent quality. More generally, one could argue that respondent behavior is inherently more biologically constrained than operant learning. Indeed, it is tempting to posit that this continuum contributes more to the distinction between respondent and operant behavior than sensitivity to the R-O relation.

We believe that our analysis remains historically true and side steps a host of problems. If we had referred to our instrumental learning as an example of operant behavior, someone would have quickly pointed out its respondent character.

This would have then been followed by a list of criteria that seemingly distinguish traditional operants from our learning phenomena, with the conclusion being that we had failed to demonstrate operant learning. In the end, we agree that the learning differs and rather than wait for the critics charge, we chose to admit the differences. Staddon may reasonably wonder whether our attempt to ascribe additional meaning to the terms instrumental and operant has lasting merit, but we expect that he would agree that struggling with these issues is preferable to the difficulties that usually follow the casual application of behavioral terms.

Staddon concludes by questioning whether it is reasonable to suppose that we can foretell what a complete theory of learning must address. Of course we cannot, and we did not mean to be so presumptuous. Keeping with his analogy to the development of physical laws, we face a situation where many have focused on the movement of the sun and the way it is pulled across the sky. They see questions regarding the movement of other bodies as less important. We seek to broaden the class of phenomena deemed relevant. Does this mean that we may occasionally attend to an irrelevant property? Probably so, but we believe that the potential benefit of a more integrated theory is worth that risk.

### ***Reilly and Schachtman: Learning to Include Nonassociative Factors***

Reilly and Schachtman (2005) are happy with the status quo and see little reason to change the way in which they characterize and study the phenomena of learning. They chide our grandiosity, suggesting that our colleagues in human learning/cognition would scoff and accuse us of lingering in days gone by when the field of learning was the centerpiece of psychology. Yes, we have ambitious aims. The integrative field of study we envision encompasses much more than “animal learning or, if you like, the field that explores a subset of learning processes” (p. 37). By focusing on the functional mechanisms that underlie learning and memory, neurofunctionalism could provide a bridge between behavioral application (human and infrahuman) and neurobiological observations. The delineation of operational principles and linking hypotheses is not, in our mind, a subspecialty, but rather a central theme that provides an essential bridge. Of course, cognitive psychologists will recognize that we are adopting key features of the information-processing paradigm. The differences are that: (1) our models will be informed by neurobiological observations (as has become the case within cognitive neuroscience) and (2) we do not limit our attention to higher brain processes (as in traditional cognitive psychology).

Reilly and Schachtman discredit our analysis by suggesting that we mix different levels of discourse. We believe that the mixing may lie elsewhere. They suggest that Pavlovian conditioning should be at the same level as protection from habituation and pairing-specific enhanced sensitization. They recognize that all three effects are produced by a similar environmental conditioning (“pairing of a CS and a US”), and thus, should be united under an unnamed superordinate category. Presumably, this step is taken to distinguish these effects from other types of learning (e.g., instrumental conditioning). Below Pavlovian conditioning, they envision the mechanism of associative learning. Their analysis implies: (1) that protection from habituation and Pavlovian conditioning are concepts of the same type; and (2) associative learning is a mechanism, while protection from habitua-

tion is not. Yet, it would seem that a US-induced disruption in a particular process (habituation) would have mechanistic implications on par with a US-induced capacity to strengthen a connection (contrary to 2). Further, if associative learning is the subordinate mechanism to Pavlovian conditioning, what is the comparable subordinate mechanism to protection from habituation—protection from habituation? Similarly, is the mechanism underlying pairing specific enhanced sensitization, pairing specific enhanced sensitization? For both protection from habituation and pairing specific enhanced sensitization, the labels imply the functional mechanism. For Pavlovian conditioning to be a concept of the same type (for 1 to be true), it too would have to have equivalent mechanistic implications, presumably achieved through reference to the concept of associative learning. But what then, in their scheme, does the label “Pavlovian conditioning” add? It would seem that associative learning, protection from habituation, and pairing-specific enhanced sensitization are concepts of the same type. Reilly and Schachtman agree that all three are produced by similar environmental conditions. The only item missing is a name for the superordinate category, a name that refers to cases where the response observed is conditional upon “a history of pairing between the CS and US” (Staddon, 2005, p. 42). The traditional terms of Pavlovian or classical conditioning would seem appropriate, but Reilly and Schachtman cannot take this course because it requires broadening the definition of conditioning to include nonassociative mechanisms.

Contrary to our target article, Reilly and Schachtman suggest that other areas of psychology organize their text around procedures and behaviors rather than mechanisms. It is unclear to us what areas and texts they refer to because the perceptual and cognitive texts that we have rely on mechanism rather than methodology to organize the material. Our texts on perception include chapters on color vision, perceptual organization, movement, space perception, audition, and the other senses (e.g., Goldstein, 1999; Matlin & Foley, 1997; Schiffman, 2000). Popular cognitive texts include chapters on pattern recognition, attention, models of memory, imagery, expertise, reasoning, and language (e.g., Anderson, 2005; Solso, MacLin, & MacLin, 2005). In both instances, the material is being grouped according to the nature of the underlying process (mechanism).

Reilly and Schachtman attempt to discredit our focus on nonassociative mechanisms by suggesting that the “mechanisms of protection from habituation and pairing-specific enhanced sensitization remain to be determined” (p. 36). Is the counter to this that the mechanisms of associative learning have been determined? Does Kandel’s work not count as a sophisticated explanation of a mechanism that can generate pairing-specific enhanced sensitization? Do Reilly and Schachtman believe we actually know more about the functional and biological mechanisms that underlie associative learning? That, to us, would seem to be a difficult position to defend.

Regarding the nature of spinal learning, it was suggested that the weaker response observed to a CS- in a Pavlovian paradigm could reflect the development of conditioned inhibition. This is logically possible, but there is no evidence to support the proposal. Consequently, parsimony would favor maintaining an account based on simpler processes. As to their claim that we assume that the “associative basis of long-term habituation applies to spinal cases” (p. 36), we do not (Joynt & Grau, 1996), and this claim was based on prior research (Groves, Lee, &

Thompson, 1969). (The claim here is only that spinal habituation seems nonassociative in nature. As acknowledged earlier, associative learning has been shown to contribute to other examples of behavioral habituation.)

Reilly and Schachtman are correct in noting that tradition does not necessarily force us to ignore neuroscience. However, in practice, this has often been the outcome. Are we just pushing for neuroscience? No, we are not. We see a detailed description of the functional (psychological) mechanisms as central. Further, it is worth remembering that the domain of neuroscience is much broader and that much of that field works happily at the biochemical/biophysical level with little (or no) reference to the functional systems in which the entity under study might be embedded. Work that ignores function may be excellent science, and of profound long-term significance, but if it is not coupled to its function (specifying its contribution to behavior within a living organism), it is not neurofunctionalism.

### ***Machado: Conceptual Confusion***

Machado (2005) attempts to discount our approach by suggesting that we make some conceptual errors. For example, he claims that we failed to notice that Pavlovian conditioning within our framework functions as a superordinate category. But our summary figure (Figure 4) explicitly depicts this relation.

Machado then goes on to show that, if we semantically switch the meaning of our terms, confusion arises. This is hardly surprising. His example involves a CS that generates a CR-like response. Machado makes the reasonable assumption that the repeated presentation of the CS would lead to habituation and that the presentation of an extraneous stimulus counters this effect. Normally, in the absence of any evidence that the S-S relation matters, we would say that the extraneous stimulus produced a form of dishabituation. Machado suggests, instead, that we call this phenomenon protection from habituation. We would not endorse this step because, following Humphrey (1933), we require evidence that the CS-US relation matters. In the way we have used associative learning, pairing-specific enhanced sensitization, and protection from habituation, dependence upon the S-S relation is integral to their definition. Further confusion arises when Machado implies that evidence that a CS is sensitive to dishabituation, and that the S-S relation matters, is sufficient to conclude that the learning depends on protection from habituation. It is not. His example does not, in any way, discount an explanation of the S-S learning in terms of associative learning or pairing-specific enhanced sensitization. He is right to suggest that his example should arouse suspicion, but this is because it involves a semantic shell game and conclusions that do not follow.

He then suggests that our true opponents are the neuroscientists, not learning theorists. We disagree because we believe that the blame lies at our feet—with those who have spent their lives studying and characterizing learning. The neurobiologists have looked to us for guidance. If the leaders in our field argue that there is just one form of true learning, neurobiologists will focus on those examples that meet the learning theorists dictate. If our textbooks outline a perspective on learning that rang true 30 years ago, who can blame the neuroscientists for following the old course. Neuroscientists recognize that something is not working here—that the traditional description of learning does not provide a useful mapping to biological systems and fails to incorporate new findings.

Machado views our push for increased attention to the neurobiology of learning as a peculiar bias. Our claim is that a typical course on learning does not incorporate much of what has been discovered in the last 20 years and that many of these discoveries occurred within the area of neuroscience. Visits to conferences, attention to funding and hiring trends, and comparisons of relative output (in terms of number and impact) would seem to support our peculiar position.

Machado doubts that broadening the scope of a learning course to include greater emphasis on mechanisms and neuroscience will increase student enthusiasm for learning. Perhaps he is right—maybe students prefer the old. Stories of studies from the laboratories of Tolman and Sheffield routinely amuse the class. But amusement alone cannot be our criterion for inclusion. Adding structural biochemistry to a biochemistry curriculum probably does not make the course more entertaining, but it does make it more informative and up-to-date. Machado concludes that we must be exhausted with the teaching of learning and that we have mistakenly concluded the field itself is exhausted. He is right that we are disgruntled with the traditional way in which material is presented, but those who are tired do not push the field to change. Being dissatisfied is not the equivalent of tired. We have entered a new era of learning and we believe that it is time to retool (see <http://graulab.tamu.edu/j-grau/psyc606.html> for an example of how our recommendations have impacted our approach to teaching).

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Received October 28, 2004.

Revision received January 7, 2005.

Accepted January 7, 2005.