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Alerts for Assessing "Biological Constraints" on Learning

J. Bruce Overmier[1] & Julia E. Meyers-Manor[2]

[1]University of Minnesota, USA
[2]Macalester College, USA

Many researchers have reported differential rates of learning and inferred selective associations between events reflecting adaptive specializations or biological constraints on learning that have evolved for each given species. Although we do not doubt that there are such biological constraints on learning, we suggest that some of the many claims may actually be spurious due to use of less than optimal research designs. We propose six methodological and inferential concerns that current researchers and reviewers of past research may find useful.

The psychology of learning has been dominated by the view that at least in mammals—and possibly all vertebrates—learning proceeds in much the same way and follows the same general principles and "laws" of learning. However, organisms seem to learn some relationships rather faster or to higher asymptotes than other relationships. Accounts of learning have sometimes reflected this by including in their derived equations different rate parameters or salience parameters for some events (stimuli or responses or outcomes) as opposed to others (e.g., Hull, 1950, Postulate XVIII; Wagner & Rescorla, 1972). More interestingly, however, is when the observed different rates of learning are due to particular combinations of events, typically across motivational systems, or when some responses seem not to be reinforced by known trans-situational reinforcers; these adaptive specializations (Rozin & Kalat, 1971) have commonly been referred to as *biological constraints* on learning and/or behavior. Shettleworth (1972) provided an informative analytic review in which she categorized these constraints into three classes: stimulus→reinforcer, stimulus-→response, and response→reinforcer. These classes, of course, may not all have the same underlying mechanisms. Biological constraints on behavior became a very popular and important topic in the study of learning by psychologists in the 1960s and 1970s because they were taken by some as challenges to the assumed generality of the laws of learning (e.g., Seligman, 1970).

One often cited classical experiment and important exemplar was carried out by Garcia and Koelling (1966). They reported that rats were given access to a saccharin

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solution from a drinking tube, and licks of the solution also caused visual and auditory cues to be presented (so-called tasty-bright-noisy water). When these exposures were followed in different groups by either electric foot-shocks or lithium induced illness, the rats came to avoid drinking the water. Tests of the separate cue-outcome combinations, however, showed that when the outcome was shocks, the avoidance of the water was based on the light/noise cues, but when the outcome was illness, the avoidance was based on the taste cue. This was taken as evidence for selective associations, and Garcia hypothesized an evolved biological relatedness of interoceptive tastes to illness and exteroceptive cues to pain as likely causal factors for these selective associations—although later work by Foree and LoLordo (1975) and Martin and Lett (1985) showed that in avians visual stimuli are better integrated with the appetitive behavior rather than taste. In any case, the idea of selective associations was not totally novel because Thorndike (1932) proposed a concept of *belongingness* as a determiner of whether an association could be formed between sets of events or whether some responses could be trained. Later, Kohler (1941) noted that, generally, "little attention [is given] to the *nature* of the things which become associated" (p. 153; bracket and italics added).

Importantly—but generally ignored by those who cite it—is the fact that the classic Garcia and Koelling (1966) experiment lacked a number of important controls that could have been the basis of the purported selective associations. We now know based on a number of later experiments by Garcia and associates and especially by Domjan and associates (See Domjan, 2015) that Garcia's inference of selective associations is, in fact, valid. Many other researchers have carried out quite different experiments that purport to show other selective associations that seem to challenge the generality of the laws of learning (e.g., Hinde & Stevenson-Hinde, 1973; Seligman & Hager, 1972).

Herein, we want to simply call to the readers' attention various methodological issues that plague some of the claimed demonstrations of selective associations. We will raise these methodological issues in a general form as guiding future researches and not as criticisms of specific claimed demonstrations. We leave it to the readers to consider the applicability to existing literature of the methodological issues we raise. We will call attention to the importance of six methodological and inferential concerns. These include (1) the problem of temporary or permanent shifts in an organism's state (motivations, attention, etc.), (2) the need for controls for *non-associative* learning, (3) the lack of attention to the particular behavior selected for observation, (4) the problems that arise from using behavioral baselines that are *unidirectional* – typically from zero, (5) the need to consider what law of learning is being *violated*, and (6) advantage of using the power of double dissociation experiments.

A full treatment of each of these issues is beyond the scope of this article. Therefore, we hope the readers will take the expositions below as "alerts" for things to keep in mind when reading experiments making special claims or when designing experiments intended to support special claims. Unfortunately, just to make our tasks as researchers harder, the methodological issues we raise below are not necessarily independent of one another.

Methodological Alerts and Challenges

Shifts in an Organism's State

The presentation of biologically important stimuli (e.g., unconditioned stimuli/reinforcers) may well induce sensitization, a well-known phenomenon of change in response to a repeated stimulus. Such sensitization may include shifts in attention to stimuli other than the presented biologically important stimulus, and these shifts in attention may differ for different stimuli or may manifest themselves as differences in responsivity to the different stimuli. Rescorla and Holland (1976) introduced this possibility as an explanation for observed apparent biological constraints on learning. On this view, any differences in observed learning are attributable not to differences in learning, per se, but to differences in selective attention that in turn modulate attention or responsivity to the stimuli. Miller and Domjan (1981 a, b) have indeed shown just such selective sensitization can occur, albeit a relatively short term effect in their hands. This suggests that one common control used to study biological constraints on learning, that of having a control group that receives a neutral event as a substitute for one or both of the unconditioned stimuli/reinforcers, is an inadequate control.

One other approach to this problem of control for sensitization or of non-specific behavioral activation (or non-specific behavioral suppression) is the use of the yoked control design. In this design, the experimental group gets pairings (e.g., typically of response and reinforcer 1 or response and reinforcer 2) and the yoked control group can make responses yet gets only the same reinforcers generated by the experimental animal but without specific contiguity between the two kinds of events. Such a yoked design should in principle control for sensitization—if the individual animals are equally sensitive to or activated by the reinforcer/US. But no two animals can be perfectly matched on sensitivity. So inevitably, for example, some sensitive experimental subject will be yoked to some insensitive control subject (or *vice versa*). Church (1964) has illustrated that this inability to match animals results in systematic differences in sensitized behavior and activity levels that can lead to false inferences about the cause of the differences between master and yoked animals and even whether or not some behavior has been learned. Such an inferential error combined with the assessment of qualitatively different unconditioned stimuli/reinforcers could mislead to an inference of biological constraints on behavior where it is unwarranted.

Need for Controls for *Non-associative* Learning

Controls are needed in the assessments for learning. A pseudo-conditioning group is common, but not perfect because it is subject to some of the concerns of induced temporary shifts in sensitivity noted above. Nonetheless, such a control can be critical for assessing whether the behavioral changes seen across trials are due to learning (i.e., dependent upon the contiguity between the events) or attributable to non-associative learning like pseudo-conditioning, which is changes in target responses to the CS as a result of experiencing the US but absent any CS-US signaling relation between them. This is easily illustrated by an experiment by Hollis and Overmier (1982). The *aggressive fighting* display of the Siamese fighting fish (*Betta splendens*), which is shown in Figure 1, appears as a species characteristic integrated response of erecting the gill covers (opercula) making the head appear larger and spreading all the fins. It is beautiful. They were studying whether brain lesions altered the Pavlovian conditioning of this display to a CS signal for an impending visualization of male US. They recorded

the components of the display separately. Both of the major components of the display came to appear readily after the presentation of the CS in the Pavlovian conditioning treatment. As can be seen in the left panel, the erection of the gill covers was acquired only in the Pavlovian conditioning treatment and not in their pseudo-conditioning treatment (in which CS and US were presented but uncorrelated) suggesting associative learning. However, the fin spreading was different! Fin spreading was elicited by the CS in both the Pavlovian and pseudo-conditioning treatments, suggesting that it was not acquired as an associative response but acquired as a non-associative response attributable to pseudo-conditioning. This was a surprising result. Clearly, to determine whether or not a behavior change is due to a specific operation, one must have control groups that get all the same events and experiences as the experimental group except for the one thing that is being tested for—here and in most cases the contiguity of events. And, we note that the exemplar Garcia and Koelling experiment described above lacked such controls leaving them open to non-associative explanations.

What is the basis for the strong pseudo-conditioning of fin spreading in the Hollis and Overmier experiment? We cannot tell. But pseudo-conditioning can, it seems, be based on a variety of factors including things as surprising yet simple as the similarity of the onset conditions of the CS and the US. Wickens and Wickens showed this in a not-often-noticed paper back in 1942. They asked about the amount of pseudo-conditioning occurring in four groups of rats to a visual stimulus when using an escapable footshock US. The groups being compared differed only in the onset characteristics of the light and shock, the onsets of each were either gradual or abrupt, giving us a 2 x 2 factorial design as to onset combinations. Pseudo-conditioning was only observed when the onsets of both were gradual or the onsets of both were abrupt; if the onsets differed, there was no pseudo-conditioning; thus onset characteristics in common were sufficient to support pseudo-conditioning. Is it possible that the abrupt onset of the light cue and the arranged abrupt appearance of the conspecific image provide the basis for the pseudo-conditioning of the fin spreading? Beyond this possibility, Rescorla (1985) also discussed his demonstrations of the role of other similarities between events in Pavlovian conditioning as factors that might contribute to appearance of CS evoked responses.

Figure 1. Photo of a Siamese fighting fish in its full *fighting* display which appears to be an integrated response including spreading of fins, erection of the gill covers (opercula), and beating of the spread tail back and forth.

The Behavior Selected for Observation

One other point to be made from the Hollis and Overmier (1982) experiment is that that they reached different inferences about occurrence of associative Pavlovian learning or non-associative pseudo-conditioned learning depending on the response they measured (Figure 2). The methodological guidance we should take from this is that it is wise to take multiple behavioral measures even when there is no *a priori* reason to suspect a need for multiple measures. The Siamese aggressive display was always assumed to be a single integrated modal action pattern, but it turns out that is not so. Hollis and her associate were not the only people to have found merit in using concurrent multiple behavioral indices. Matzel, Schachtman, and Miller (1985) showed that taking multiple measures in their experiments that revealed that overshadowing is not a failure of

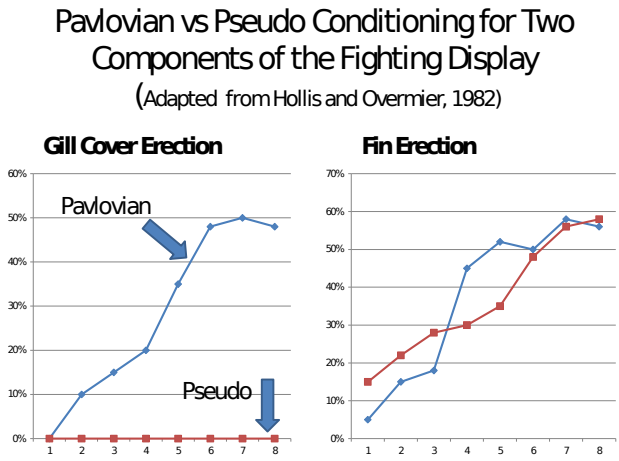

Figure 2. Sample data showing (a) that some apparently classically conditioned responses may well be pseudo-conditioned, and (b) that the components of apparently integrated action patterns may be dissociated and be differentially subject to associative and non-associative processes. Data excerpted from Hollis and Overmier (1982) and redrawn.

association but of behavioral manifestation. Dunham and Grantmyre (1982) gave us new insights into the effects of punishment on an act and related behaviors, and on how post-punishment behaviors get redistributed because they concurrently measured several behaviors during the experimental treatment. And, in the appetitive domain, Timberlake (2001) has shown us how measuring multiple responses can reveal different motivation-specific search behaviors that are dependent upon the qualities and timing of signals, reinforcers, and the structure of the environment. These revealed that multiple response behavior systems constitute rich information about biological constraints on behavior.

But, all too often we—we are guilty, too—still design our experiments with single behavior dependent variables.

The Problems of Zero-based Unidirectional Baselines

To say that one condition produces more, better, or faster learning than another group, we must compare the behavior from the two conditions. Typically we look at the performance from the beginning of the conditioning—rate of change and/or asymptote

achieved. If one is higher than the other as in Figure 3 Panel A, we infer a difference in rate of learning or in the amount learned. But such a plot and inferences implicitly assumes that both CS stimuli are equivalent at the outset of learning. Frankly, we know that this is not in fact usual. So, we try to use some difference score for each CS stimulus that takes into account the differences in the unconditioned state. A couple of different approaches have been used. One is to use the first trial performance as an indicator of the baseline performance for a given CS; another is to use some control treatment that exposes the control subject to the biologically important stimulus but that is not supposed to lead to learning of associations, for example the so-called Truly Random Control (TRC). Thus, now we can compute difference scores for both CSs, as illustrated in Figure 3 Panel B. But, both of these approaches suffer from the same problem: they assume that the baseline performance is a zero value or some positive value. Yet we know that sometimes stimuli have—or acquire—inhibitory properties with respect to behaviors. And that cannot easily be detected when using a zero level baseline as is the case in most learning experiments (e.g., you cannot drink less than 0 drops of a fluid). A third approach is to design the experiment so that inhibitory effects as well as excitatory effects of stimuli can be detected. That typically requires at the outset some ongoing level of behavior that we hope to use as our dependent variable. A classic way to do this is to train a baseline of behavior and then superimpose the CS on that bidirectionally sensitive baseline. The CSs may be untrained or subject to some treatment thought not to support learning (e.g., TRC). The classic transfer of control design is useful here. In it, the dependent variable baseline is trained, the CSs are given associative or non-associative treatments, and then the CSs are presented while the animal is performing the baseline behavior. Changes from the baseline detect the stimulus specific effects for both the associative and the non-associative conditions. The differences between these for the two CSs then can be used to assess the rate of learning as separable from any differences in the asymptotic levels achieved in the associative condition. This is illustrated in Figure 3 Panel C.

In fact, Linwick, Paterson, and Overmier (1981) provided just such a demonstration as shown in Figure 3 Panel C (redrawn from their data). Had they used a design with no baseline (Panel A) or one with a unidirectionally sensitive baseline (Panel B), they might have inferred that their two stimuli (a tone CS and a light CS, each paired with footshock) were different in amount of fear learning that took place. But when they used the bidiectionally sensitive baseline to assess the TRC control treatment, we see that the non-associative effect of one of the stimuli is neutral while the non-associative effect of the other is inhibitory for that target behavior. When the *difference scores* between the non-associative reference condition and the conditioned fear detected (functionally the slope, rate, or amount of conditioned associative learning) are computed, they saw that the associative learning to the two different CSs appear equal! Although one can argue about the right index for learning, it seems that one cannot easily argue against using bidirectionally sensitive baselines (trained or natural pre-existing ones). Is it possible that the reason Cook and Mineka (1989,) in their now classic experiments on observational learning of fear, failed to find emergence of fear to flowers (flowers being so-called “fear-irrelevant”) was because flowers are inherently initially inhibitory of fear responses? Their unidirectional baseline would not allow us to see that. We recognize that there are other approaches for detecting inhibitory influences like resistance to conditioning, but we believe that the bidirectionally sensitive baseline is the most straight forward approach.

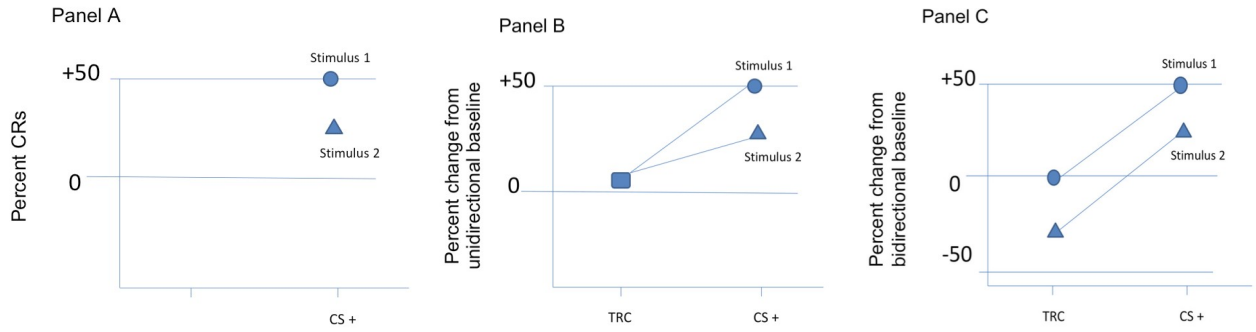

Figure 3. Different ways of evaluating obtained differences in asymptotic levels of conditioning. Panel A directly compares the asymptotic levels attained with the two CSs. Panel B compares the two CSs on the basis of the change or slope from baseline to asymptote, when the baseline is only unidirectionally modifiable. Panel C compares the conditioning to the two CSs on the basis of difference from a control treatment of the CS where all values are changes from baseline behavior where the baseline is bidirectionally sensitive to the properties of the stimuli (redrawn).

What Law of Learning is being *Violated*?

In addition to a basic demonstration of selective associations, Garcia and his associates (Garcia, Ervin, & Koelling, 1966) also demonstrated that the association of taste with illness seemed to occur over long delays—delays longer than had previously been used in associative learning tasks. This *long-delay* learning was pounced upon by theoreticians as a violation of some basic law of learning and thus evidence that there were no *general laws of learning* (e.g., Seligman, 1970). But what was this basic general law that was being violated? We presume that the referenced law is *contiguity*, that taste and illness could be associated without close temporal contiguity. The intervals in Garcia, Ervin, and Koelling over which associations were formed were in fact an hour or more. This was a surprise to many who had grown up with the paradigmatic example of classical conditioning being the conditioning of the eyeblink reflex and the usual CS onset to US onset interval was about 0.5 sec and cited in every introductory textbook. Indeed, some theorists actually argued that this 0.5 sec interval was determined by the neural system underlying learning and were likely general for all learning. Those swayed by this line of reasoning must have forgotten Pavlov's lectures (1927) wherein Pavlov routinely describes conditioning experiments on salivary conditioning with CS-US intervals of 30 sec or more!

We now know that the ratio of the CS-US interval to the ITI (or closely related values depending on the particular theory) is an important determiner of whether an association develops or not (e.g., Gibbon & Balsam, 1981). In the eyeblink experiments, the inter-trial interval (ITI) was seconds; in the salivary experiments, the ITI was several minutes; in the taste aversion learning, the ITI was day(s). It turns out that the general principle is that there exists — for each particular CS and particular US conditioning combination of a given response with a particular ITI — an optimal CS-US interval and CS-US interval values longer or shorter than that optimal value produce less effective conditioning. This is seen in a range of species conditioning different responses (e.g., McAllister, 1953 [human eyelid optimum 0.5 sec]; Noble, Gruender, & Meyer, 1959 [fish

motor movement optimum 2 sec]; Noble & Adams, 1963 [pig motor movements optimum 8 sec]; Longo, Klempay, & Bitterman, 1964 [pigeon motor movements optimum 10 sec]; Paré 1967 [rats CER optimum 2 min]). The general law seems to be that amplitude of conditioning is an inverted U-shaped function of CS-US interval; the central locus of that function is characteristic for each particular CS, US, response, and ITI combination—a summary that might fit well into Shettleworth's review. For example with regard to taste aversion learning, a much overlooked experiment by Krane and Wagner (1975) is consistent with this summary. They showed that *both* light-tone compounds and tastes can become CSs for illness and that *both* light-tone compounds and tastes can become CSs for shock, but they do so at very different CS-US intervals, thus challenging any *belongingness* hypothesis. (See also Westbrook, Clarke, and Provost, 1980, and Martin & Lett, 1985, for their data, not their conclusions.)

Double Dissociation Paradigm

The Garcia and Koelling (1966) bright-noisy-tasty water aversion learning demonstration was readily accepted despite its lack of controls. This is perhaps because it used a powerful methodological design that allowed showing of a *double dissociation* in which there was a full reversal of which CS became associated with illnesses and which became associated with shocks. Double dissociation demonstrations in behavioral neuroscience are generally quite compelling and often required to show system disjunctions (Weiskrantz, 1968). And because of their compelling power, others studying adaptive specializations and biological constraints on learning have also adopted this design (Domjan & Wilson, 1972; Foree & LoLordo, 1973; Miller & Domjan, 1981). But the design in its basic form does have weaknesses, Weiskrantz (1968); describes conditions under which a double dissociation demonstration is not sufficient for validating qualitative differences in treatment effects (e.g., see pp. 418-419).

One case is illustrated in Figure 4. An apparent double dissociation can arise when performance is an inverted-U shape. One example would be in the Yerkes-Dodson Law where performance is an inverted-U shape function of level of motivation (or some other some resource function). Then, even when two different CSs stand in the same *associability* relation to each other, if the two reinforcers used place the CS contrasts at opposite sides of the inverted U-function between performance and motivation: CS2 will be greater than CS1 at low motivation and CS2 will be less than CS1 at high motivation. [Think of moving a two-tine fork along the x-axis of an inverted U-shaped function.] Thus, one could obtain the data for a double dissociation in the absence of any associative mechanism difference. Double dissociations are not typically all-or-none, and such task sensitivity then plays a role.

Additionally, double dissociation designs have typically not controlled well for differential sensitization or differential pseudo-conditioning across USs. Conditioning designs with controls that include presentation of *both* CSs and presentation of *both* USs are needed for this, and they have been developed where all subjects get all stimuli (e.g., Rescorla & Holland, 1976).

Conclusions

We accept that differential rates of learning to different stimulus-reinforcer-response event combinations occur and that these may reflect selective associations between events thus reflecting adaptive specializations and biological constraints on learning. But we think that many simple demonstrations of such differences in learning found when using different CSs or different USs are sometimes all too readily initially characterized as reflecting biological constraints on learning. That such constraints do exist does not imply that every apparent difference in learning *is* an instance of biological constraints on learning. Indeed, we think that some such inferences may be spurious. We have highlighted a few methodological and inferential concerns and challenges that current researchers going forward—and reviewers of past research—may find useful as they think about research on biological constraints on learning. In a sense, these are old methodological issues and unfortunately sometimes forgotten ones, but they are of current relevance. Our list is not exhaustive; for example, we have not discussed *between vs within subject* designs (Charnes, Gneezy, & Kuhn, 2012; Grice,

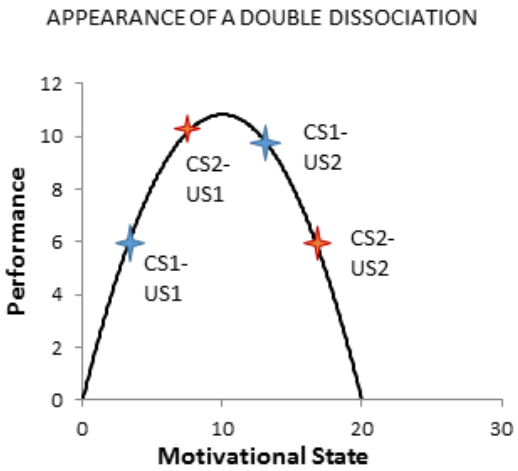

Figure 4. Shows how an experiment that pairs two different CSs with two different USs of strikingly different motivational power might lead to an appearance of a double dissociation in the absence of different associability (here CS2 > CS1) if the performance function is non-monotonic with respect to the motivational function (or other task sensitivities).

1966), but rather our list is illustrative that careful analysis and rich designs are needed as hallmarks for such demonstrations to support strong inferences and claims. We do not want to claim that heeding these various alerts will lead to successful identification of a biological constraint, but rather to do so may help us avoid erroneous inference.

We would like our note here to be in the spirit of—or better an extension of—Wesikrantz's (1968) provocative chapter "Some traps and pontifications" and Domjan and Galef's (1983) "Retrospect and prospect". If you have not read these, you should be sure to do so because (a) they, too, are relevant to what inferences we can draw from what kinds of data sets and how we sometimes get led astray, and (b) they offer guides as to ways forward in this challenging area of research.

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