Associative change in Pavlovian conditioning: A re-appraisal

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Abstract

Robert A. Rescorla changed how Pavlovian conditioning was studied and interpreted. His empirical contributions were fundamental and theoretically driven. One involved testing a central tenet of the model that he developed with Allan R. Wagner. The Rescorla-Wagner learning rule uses a pooled error term to determine changes in a directional association between the representations of the conditioned stimulus (CS) and unconditioned stimulus (US). This learning rule predicts that two equally salient CSs (A and B) will undergo equivalent associative change when they are conditioned in compound (i.e., AB→US). Rescorla's results suggested that this was not the case (e.g., R.A. Rescorla, 2000, Associative changes in excitors and inhibitors differ when they are conditioned in compound. *Journal of Experimental Psychology*: *Animal Behavior Processes*, *26*, 428-438). Here, we show that these results can be reconciled with a model that uses a learning rule with a pooled error term once that rule is applied equivalently to all of the stimuli presented on a given trial, and the resulting reciprocal associations (directly and indirectly) contribute to performance. This model, called HeiDI, integrates several features of Rescorla's research and theorizing while addressing an issue that he recognized required further analysis: How learning is translated into performance.

Keywords: Pavlovian Conditioning, Associative structures, Performance.

Robert A. Rescorla's most cited work is a book chapter describing the model of Pavlovian conditioning that he developed with Allan R. Wagner (Rescorla & Wagner, 1972). However, the Rescorla-Wagner model is only one highlight in a career full of fundamental theoretical and empirical contributions. His principal interest and broad influence can be illustrated in two ways. The first is the title of one of his many funded projects: *Pavlovian conditioning*. That was the title, no need for embellishment. The second was one of his relatively rare review articles, entitled: *Pavlovian Conditioning: It's not what you think it is* (Rescorla, 1988). The paper deconstructed outdated (textbook) descriptions of Pavlovian conditioning in the context of research, including some of his own, that had helped to shape the field.

One component of this deconstruction was – Rescorla recognized – something of a double-edged sword: It involved the nature of the conditioned response (CR) generated by pairing a conditioned stimulus (CS) with an unconditioned stimulus (US). Rescorla noted that textbooks still adopted Pavlov's description of the CS becoming a substitute for the US, coming to elicit the same response and the US. However, this principle of *stimulus substitution* (Pavlov, 1927) failed to capture the fact that the form of the CR is influenced by the nature of the CS. The results reported by Timberlake and Grant (1977) provide a vivid example. One group of hungry rats received trials on which the presentation of a small wooden block was paired with food, and a second group received trials where the presentation of a conspecific was paired with food. The first group came to orient towards the wooden block, whereas the second group came to exhibit an increase in social behaviors directed toward their conspecific. These results are based on the use of an unorthodox procedure, but the conclusion they support is a general one: The form of the CR is affected by the nature of the CS and the US (for a review, see Holland, 1984).

The observation that a given conditioning procedure can result in the development of CRs that reflect the nature of the CS as well as the US (e.g., Derman, Schneider, Juarez, & Delamater, 2018; Patitucci et al., 2016) undermines textbook descriptions of Pavlovian conditioning and the principle of stimulus substitution to which they were linked; but contemporary theories of Pavlovian conditioning had surprisingly little to say about the very same observation. This led Rescorla (1988, p.158) to conclude that "*We are badly in need of an adequate theory of performance in Pavlovian conditioning*". However, in the ensuing years the frequency or vigor of a given CR continued to be used as an index of the strength of the presumed directional association from the CS to the US: An analysis of performance that provides no mechanism for the CR developed through conditioning to be influenced by the nature of the CS.

Associative structures and performance

$$\Delta V_{CS\text{-}US} = \alpha_{CS}\beta_{US}(\lambda - \Sigma V_{TOTAL\text{-}US}) \qquad \text{(Equation 0)}$$

The Rescorla-Wagner model assumed that pairing a CS with a surprising US resulted in the strengthening of an association from the CS to the US: With the CS→US association being updated on a given trial (i.e., $\Delta V_{CS\text{-}US}$) according to Equation 0. In this equation, λ is the maximum associative strength supportable by the US and $\sum V_{TOTAL\text{-}US}$ is the sum of the associative strengths of stimuli present on the trial; α and β are learning rate parameters related to the intensities of the CS and US, respectively. However, as a trial-based model, it would have been natural to allow an association to also form from the US to the CS. The fact that the model did not allow this has sometimes puzzled students of animal learning theory and made life more difficult for those involved in their education. Indeed, once such reciprocal associations are assumed there is a ready basis for performance to reflect the properties of the US

(based on the CS→US link) and the CS (based on the reciprocal US→CS link): The CS will associatively activate the US representation, which will then associatively activate the CS via the US→CS association. This suggestion was outlined by Asratian (1965; p. 179; see Figure 1), but to the best of our knowledge it was not implemented as a formal model.

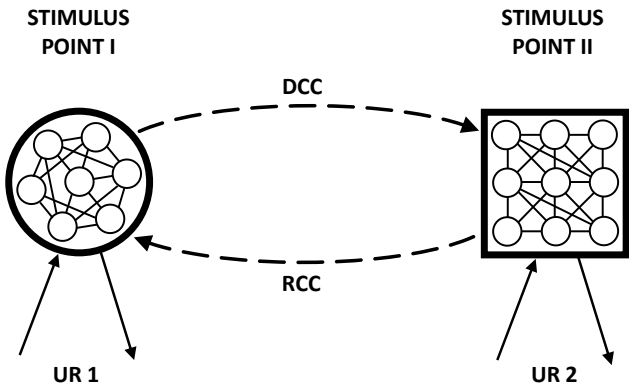

Figure 1. "FIG. 8. Scheme of the conditioned reflex with bidirectional connection. DCC, direct conditioned connection; RCC, reverse conditioned connection; UR 1, unconditioned reflex No. 1; UR 2, unconditioned reflex No. 2." [Adapted by Honey & Dwyer (2021a) from: E.A. Asratian. (1965). *Compensatory Adaptations, Reflex Activity & the Brain*. Oxford: Pergamon Press.]. Stimulus Points I and II are reciprocally connected (by DCC and RCC), but the elements shown within these points are not fully interconnected and their nature was not made explicit.

The Rescorla-Wagner model could have been extended to generate the associative structure depicted in Figure 1 by complementing the standard rule for the CS→US association (DCC; i.e., $\Delta V_{CS\text{-}US} = \alpha\beta(\lambda_{US}-\sum V_{TOTAL\text{-}US})$) with rule a for the US→CS association (RCC; $\Delta V_{US\text{-}CS} = \alpha\beta(\lambda_{CS}-\sum V_{TOTAL\text{-}CS})$). However, such a process of extension results in a proliferation of free parameters: A separate λ for the CS (λ_{CS}) to set the asymptote for the US→CS association; and a separate α value for trials on which the US is presented but the CS is not, to allow extinction of the US→CS association to occur. With the latter requirement matching the need for a separate β value to allow the CS→US association to extinguish on nonreinforced CS trials. These additional α and β parameters are required because otherwise the product of the

learning rate parameters ($\alpha\beta$) would be 0 on trials on which either the CS or US was absent, and no learning would occur.

Honey, Dwyer and Iliescu (2020a; see also, Honey, Dwyer & Iliescu, 2020bc) developed alternative learning rules, within a model called HeiDI, that retained the important contribution of the Rescorla-Wagner model (i.e., the pooled error term) while reducing the number of free parameters (see Equations 1 and 2; where c refers to a constant of 1 in units of V, which serves to balance the equations). According to these rules, the (perceived) intensities of the CS (related to α_{CS}) and US (related to β_{US}) serve as learning rate parameters for the CS→US (α_{CS} in Equation 1) and US→CS (β_{US} in Equation 2) associations; but they also determine the asymptotes for the CS→US (β_{US} in Equation 1) and US→CS associations (α_{CS} in Equation 2).

$$\Delta V_{CS\text{-}US} = \alpha_{CS}\left(c.\beta_{US} - \Sigma V_{TOTAL\text{-}US}\right) \qquad \text{(Equation 1)}$$

$$\Delta V_{US\text{-}CS} = \beta_{US}\left(c.\alpha_{CS} - \Sigma V_{TOTAL\text{-}CS}\right) \qquad \text{(Equation 2)}$$

$$V_{COMB} = V_{CS\text{-}US} + \left(\frac{1}{c}.V_{CS\text{-}US} \times V_{US\text{-}CS}\right) \qquad \text{(Equation 3)}$$

HeiDI assumes that the overall associative strength of the CS→US/US→CS assembly (V_{COMB}) – upon presentation of the CS – is given by Equation 3. V_{COMB} influences the overall levels of conditioned behavior (e.g., at asymptote), but not the form of that behavior. According to Equation 3, V_{COMB} is equal to the sum of the associative strengths of the CS→US ($V_{CS\text{-}US}$) and US→CS ($V_{US\text{-}CS}$) links, with the US→CS link being multiplied by the numerical value of the CS→US link (i.e., 1/c).[1] This

[1] An associative value can be converted into a dimensionless scalar (like α_{CS}) through multiplying it by the reciprocal of c (i.e., 1/c), where c = 1 in units of V. Multiplying the output value for the US→CS association by the converted CS→US dimensionless value means that the product is in units of V.

modulation of the contribution of the US→CS link means that, other things being equal, the link from the stimulus that is present (i.e., the CS) contributes more to V_{COMB} than the link from the stimulus that is not (i.e., the US). It also means that once conditioning has taken place, nonreinforced presentations of a CS will result in V_{COMB} approaching 0 in spite of the fact that the US→CS association is left completely intact by such an extinction treatment (cf. Iliescu, Hall, Wilkinson, Dwyer, & Honey, 2018).

While the value of V_{COMB} affects the overall levels of performance, the influence that the properties of the CS and the US exert on the nature of the CRs is determined by Equations 4 and 5. In Equations 4 and 5, the (perceived) intensity of the CS relative to that of the (retrieved) US determines the distribution of V_{COMB} into two components: R_{CS} (Equation 4) and R_{US} (Equation 5). These components affect CS-oriented responses (R_{US}) and US-oriented responses (R_{US}) through their multiplicative influences on the (unconditioned) links from the CS and US to different response-generating units (see Honey et al., 2020a). It is assumed that R_{CS} predominantly affects CS-oriented behavior, via (unconditioned) links from the CS to response-generating units, and R_{US} mainly affects US-oriented behavior, through (unconditioned) links from the US to different response-generating units (see Honey et al., 2020a).

$$R_{CS} = \frac{\alpha_{CS}}{\alpha_{CS} + \frac{1}{c}.|V_{CS\text{-}US}|} V_{COMB} \qquad \text{(Equation 4)}$$

$$R_{US} = \frac{\frac{1}{c}.|V_{CS\text{-}US}|}{\alpha_{CS} + \frac{1}{c}.|V_{CS\text{-}US}|} V_{COMB} \qquad \text{(Equation 5)}$$

In Equations 4 and 5, the perceived intensity of the CS is aligned to α_{CS} whereas the perceived intensity of the US is given by the (absolute) strength with which the US is being retrieved (i.e., $1/c|V_{CS\text{-}US}|$); because the β value of the US is not available when the CS is presented and conditioned responding is ordinarily measured (for further discussion, see Honey et al., 2020a). The general idea that the (perceived) intensity of stimuli affects performance was foreshadowed by Hull (1949). The parenthetical term (perceived) allows the values of R_{CS} and R_{US} to vary between individual animals and to provide a basis for individual differences in the form and vigor of conditioned responding (e.g., Iliescu et al., 2018; Patitucci, Nelson, Dwyer & Honey, 2016).

The analysis outlined above can be extended to include all of the stimuli that are present on a given trial. For example, when a stimulus compound (AB) is followed by a US then reciprocal associations form between all three components of the trial: A→US, US→A, B→US, US→B, A→B, and B→A. In fact, associations between A and B, which Rescorla and colleagues dubbed *within-compound associations*, extend considerably the explanatory powers of associative models (e.g., Durlach & Rescorla, 1980; see also, Honey et al., 2020ab; McLaren, Kaye & Mackintosh, 1989; McLaren & Mackintosh, 2000). It is therefore surprising, from a variety of perspectives, that reciprocal (e.g., within-compound) associations have not been integrated into a formal model of Pavlovian conditioning. Instead, they have often been used in an *ad hoc* and informal way to explain inconvenient observations or have themselves been treated as inconveniences that need to be controlled for. Rescorla (2002) even mused that he wished that he had not invented them, when he was Guest Lecturer at the Associative Learning Symposium, Gregynog Hall.

The idea that A competes with B for association with the US underpins the capacity of the Rescorla-Wagner model to explain overshadowing (Mackintosh, 1978;

Pavlov, 1927) and blocking (Kamin, 1969). The inclusion of reciprocal connections from the US to A and B provides one basis for the interpretation of a broader range of phenomena including what is arguably Rescorla's most significant contribution in the 21st century: *Associative changes in excitors and inhibitors differ when they are conditioned in compound* (Rescorla, 2000; see also, Rescorla, 2001). This fact is not predicted by the Rescorla-Wagner model. However, we will show how it is predicted by HeiDI using a series of detailed simulations; remembering that HeiDI was originally developed to provide an associative framework to account for the important influences of the CS and the US in determining the form of the CR, and the marked individual differences in the form of the CR (e.g., Patitucci et al., 2016).

Associative change of stimuli conditioned in compound

The use of a pooled error term means that any change in associative strength of one stimulus (A) on a trial with respect to a given US is affected by the presence of other stimuli (e.g., B) that have (nonzero) associative strength for the same US. The use of this error term allowed the Rescorla-Wagner model to provide a ready explanation for a range of cue interaction effects, including the fact that prior conditioning trials with one stimulus (B→US) blocks the development of conditioned responding to another stimulus (A) with which it is conditioned in compound (i.e., AB→US; Kamin, 1969). However, provided it is the case that two stimuli have the same intensity, then the Rescorla-Wagner model predicts that the amount of associative change in one component of a compound should match that of the other; for example, in a blocking procedure, both the blocking agent (B) and the target (A) are predicted to undergo equivalently little associative change as a result of trials on which they are conditioned in compound. It was this simple prediction – of equivalent associative change to cues conditioned in compound – that Rescorla (2000) sought to investigate.

He developed two ingenious experimental designs that enabled a comparison of the associative change in CSs that elicited very different levels of conditioned behavior (i.e., a conditioned excitor and conditioned inhibitor). These designs are summarized in Table 1.

Table 1: Experimental Designs 1 and 2 from Rescorla (2000)

Stage 1	Stage 2	Test
Design 1: AB reinforced in Stage 2		
A→food, C→food, X→food BX→no food, DX→no food	AB→food	AD < BC
Design 1: AB nonreinforced in Stage 2		
A→food, C→food, X→food BX→no food, DX→no food	AB→no food	AD < BC
Design 2: AB reinforced in Stage 2		
A→food, C→food AB→no food, CD→no food	AB→food	AD < BC
Design 2: AB nonreinforced in Stage 2		
A→food, C→food AB→no food, CD→no food	AB→no food	AD < BC

Note: A, B, C, D and X were different conditioned stimuli. When the designs were conducted with pigeons, A-X were different visual stimuli and food was a small amount of grain; and when conducted with rats, A-X were different auditory and visual stimuli, and food was in the form of a small pellet. The stimuli serving as A, B, C, D and X were counterbalanced, and the experimental events were delivered in standard conditioning chambers. In the final test, conditioned responding to AD was lower than to BC.

Consider first the training given to animals in stage 1 of Design 1 (Experiments 1-4, Rescorla, 2000): A, C and X were independently paired with food, while the BX and DX compounds were separately presented and followed by no food. According to the Rescorla-Wagner model this training should result in the formation of independent (directional) excitatory associations: A→food, C→food and X→food; and inhibitory

associations between B and food, and between D and food. During the second stage, the simultaneous compound of A and B was either paired with food (in one pair of experiments) or no food (in a second pair of experiments). This training should result in equivalent increases (when reinforced) and decreases (when nonreinforced) in the associative strength of both A and B (cf. Rescorla & Wagner, 1972). However, when A was tested with inhibitor D (AD) and B with the excitor C (BC), BC elicited more responding than AD. In the case where AB had been reinforced, Rescorla (2000) reasoned that the associative strength of B must have increased more than A, and when AB had been nonreinforced the associative strength of A must have decreased more than B: In both cases, this would mean that BC would elicit more conditioned responding than AD. The general conclusion that this reasoning supports is that the change in associative strength of the components of a compound is more marked for the component whose associative strength deviates most from the outcome of a trial. This conclusion represents a significant challenge to the Rescorla-Wagner model, but also for other formal models of Pavlovian conditioning (e.g., Pearce, 1994; Pearce & Hall, 1980; but see Holmes, Chan & Westbrook, 2019).

Instead of abandoning such models, Rescorla (2000) considered an alternative (informal) account that was based on the suggestion that within-compound associations (e.g., A→B) develop during the critical AB trials. These associations had already been implicated in phenomena that appeared to be beyond the scope of the Rescorla-Wagner model (e.g., *potentiation*: Durlach & Rescorla, 1980; *unblocking*: Rescorla & Colwill, 1983). Moreover, there is a significant body of independent evidence demonstrating their ubiquity, from studies of *sensory preconditioning* (e.g., Brogden, 1939; Rescorla, 1980; Rescorla & Cunningham, 1978) and *second-order conditioning* (e.g., Pavlov, 1927; Rescorla, 1982; Rizley & Rescorla, 1977; for a recent review, see

Honey & Dwyer, 2021ab). Indeed, recent evidence has revealed that such within-compound (or sensory) associations exhibit fundamental similarities to Pavlovian conditioning (e.g., Maes, Sharpe et al., 2020). The application of an analysis in terms of within-compound associations to the results from Design 1 is relatively straightforward.

The development of reciprocal A→B and B→A associations during stage 2 (in Design 1) would allow the AD compound to retrieve B and the BC compound to retrieve A. Given the fact that A was paired with the US during stage 1 and B was not, this would result in BC generating a more marked CR than AD. To address this alternative interpretation, Rescorla (2000, Experiments 5 and 6) used Design 2 (see Table 1). During the pretraining session, stage 1 in Table 1, when A and C were presented alone they were paired with food, whereas when they were present as the parts of a compound (i.e., AB and CD) they were not paired with food. Rescorla (2000) argued that when AB was then paired with food, during stage 2, it was less likely that changes in the A→B association would contribute to differential responding to AD and BC during the test. For example, in the general discussion of a companion paper in which within-compound associations were also a potential explanation for the results, Rescorla (2001; pp. 65-66) wrote: "*The pretraining* [in Design 2 of Rescorla, 2000] *guaranteed that A and B had been repeatedly presented jointly, presumably resulting in near asymptotic levels of a within-compound A-B association*." According to this analysis, when AD was tested it might retrieve B and C, while BC might retrieve A and D; but the net effects of the retrieved stimuli on performance to the two compounds should be similar. However, Rescorla (2000) reported that BC elicited more responding than AD, whether the AB compound had been reinforced or nonreinforced. Again, B appeared to have increased in associative strength more than A when both were reinforced in

compound, and A appeared to have decreased in associative strength more than B when both had been nonreinforced in compound.

The foregoing analysis of the role of within-compound associations in Design 2 did not (explicitly) consider the fact that during stage 1, the A→US (and C→US) trials will result in extinction of the A→B association (and C→D association); and the AB trials of stage 2 will allow the A→B association (but not the C→D) association to increase. As we shall see, this observation provides one basis for an alternative analysis of the results from Design 2: An increase in the ability of A (and AD) to borrow the associative properties of B. However, the complexity of the experimental designs requires the provision of a formal analysis not least because intuitive analyses can be inaccurate. This analysis is presented below.

HeiDI simulations

The simulations that we present here are based on a simplified version of the treatment of the effects of reinforcement (US presence or stimulus presence) and nonreinforcement (US absence or stimulus absence) employed by Rescorla and Wagner (1972; see Equations 1 and 2; cf. Konorski, 1967; Zimmer-Hart & Rescorla, 1974): When the target of an association is present its perceived intensity determines the value of its corresponding parameter (e.g., β_{US}), and if it is absent this parameter is set to 0. While the Rescorla-Wagner model has the obvious advantage of providing an elegant analysis of the formation of excitatory and inhibitory associations resulting from reinforcement and nonreinforcement, its application to directional associations between selective components of a given trial (from CSs to USs) provides no account for a broad range of other phenomena (most obviously higher-order conditioning). HeiDI uses a simplified form of the Rescorla-Wagner learning rule for changing the reciprocal links between any two stimuli. Equations 1 and 2 illustrate this rule in the context of

Pavlovian conditioning trials, with the general form of the rule for any two stimuli (1 and 2) being: $\Delta V_{1\text{-}2} = \alpha 1(_{c.}\alpha 2 - \sum V_{TOTAL\text{-}2})$. The consistent application of the rules generates associative chains (e.g., CS1→CS2→US), which can also contribute to performance (see Honey & Dwyer, 2021ab).

The resulting simulations are necessarily more complex than those derived from the Rescorla-Wagner model. However, in addition to providing an analysis of the results that seemed to undermine the sufficiency of a central tenet of the Rescorla-Wagner model (the pooled error term) they generate several intriguing predictions. They also confirm that intuitive analyses of the role of within-compound associations can be inaccurate. To foreshadow the results of the simulations, according to HeiDI the effects reported by Rescorla (2000; see also, Rescorla, 2001) involving reinforcement of a compound (consisting of an excitor and inhibitor) have a different origin to those involving nonreinforcement of the same compound: They rely differentially on reciprocal (US→CS) associations and within-compound (CS→CS) associations. This analysis, therefore, generates predictions about the (differential) impact of manipulations that target these types of association. The simulations also yield predictions about how the effects will be translated into different forms of conditioned responding, CS-oriented and US-oriented, which is quite beyond extant theories.

Honey et al. (2020a) presented a proof-of-principle simulation illustrating the idea that allowing US→A and US→B associations to change (as well as A→US and B→US associations) on AB→US trials resulted in a greater change in the US→B association than the US→A association. Consequently, the combined strength of the reciprocal associations (V_{COMB}) was greater for BC than AD, and this was reflected in both R_{CS} and R_{US}. However, accepting Rescorla's (2000, 2001) arguments – about the limited scope for within-compound associations to contribute to the results that he reported –

these original proof-of-principle simulations were reported without presentation of the concurrent influence of within-compound associations. This approach necessarily obscured any role for these associations in generating (or indeed preventing) changes of importance.

Here, we provide a more comprehensive analysis of the experimental designs used by Rescorla (2000; see also, Rescorla, 2001) in which all of the potential reciprocal associations involving the CSs and US change in the way specified in Equations 1 and 2, and generic versions of these rules for any pair of stimuli (e.g., within-compound associations between two CSs). As already noted, these within-compound associations form part of associative chains that also contribute to performance. Thus, according to Equation 6, the links from CS1 (or a compound of CSs) to CS2 would allow that CS1 (or the compound) to borrow the combined value of the reciprocal CS2→US, US→CS2 associations (i.e., $V_{COMB\ CS\text{-}US}$). According to this equation, the V output for the associative chain CS1→CS2→US ($V_{CHAIN\ CS1\text{-}CS2\text{-}US}$) is calculated by multiplying the (numerical) value of the CS1→CS2 link by $V_{COMB\ CS2\text{-}US}$.[2]

$$V_{CHAIN\ CS1\text{-}CS2\text{-}US} = \frac{1}{c}.\Sigma V_{CS1\text{-}CS2} \times V_{COMB\ CS2\text{-}US} \quad \text{(Equation 6)}$$

In Design 1 there are 30 potential binary associations involving the 5 CSs and US (only 5 being links from the US to the CSs); while in Design 2 there are 20 potential binary associations involving the 4 CSs and US (only 4 being links from the US to the CSs). The simulations are correspondingly complex, but the inclusion of all of the

[2]Honey and Dwyer (2021ab) describe different ways in which the contribution of associative chains could be implemented, including modulating their efficacy by the similarity of the perceived intensity of retrieved component (e.g., CS2 via $V_{CS1\text{-}CS2}$) to its corresponding conditioned intensity (α_{CS2}). The inclusion of this additional complexity does not materially affect the pattern of simulated outcomes presented here, and we therefore report only the simulations involving the chains described in HeiDI (Honey et al., 2020a).

binary associations is theoretically principled: The learning rules are being consistently applied. All simulations used the same set of parameters with the α_{CS} values for the CSs set to .40, and β_{US} set to .80 on reinforced trials. As already noted, on trials when a stimulus is absent its α value was set to 0 and on nonreinforced trials β_{US} was set to 0. However, the pattern of results from the simulations described below is not restricted to this specific choice, but rather extends to a broad range of parameters.

Simulations of stage 1 for Design 1. The results from the simulations of the first stage of training for Design 1 are shown in Figure 2. The important features of each panel will be described, alongside their relationship to changes in other panels where those changes are important. Panel 1 depicts the output values for the 5 CS→US associations and panel 2 depicts the output values for the 5 US→CS associations. Inspection of panel 1 confirms that A and C develop strong associations with the US, X develops a somewhat weaker association with the US, while stimulus B and stimulus D gain negative associative strength. Panel 2 shows that the US develops excitatory associations with A, C and X. The US also develops inhibitory associations with B and D: On X→US trials the error terms for the US→B and US→D associations are negative, because of the contribution of the excitatory X→B and X→D associations to the error terms. The X→B and X→D associations form on nonreinforced BX and DX trials (see panel 6).

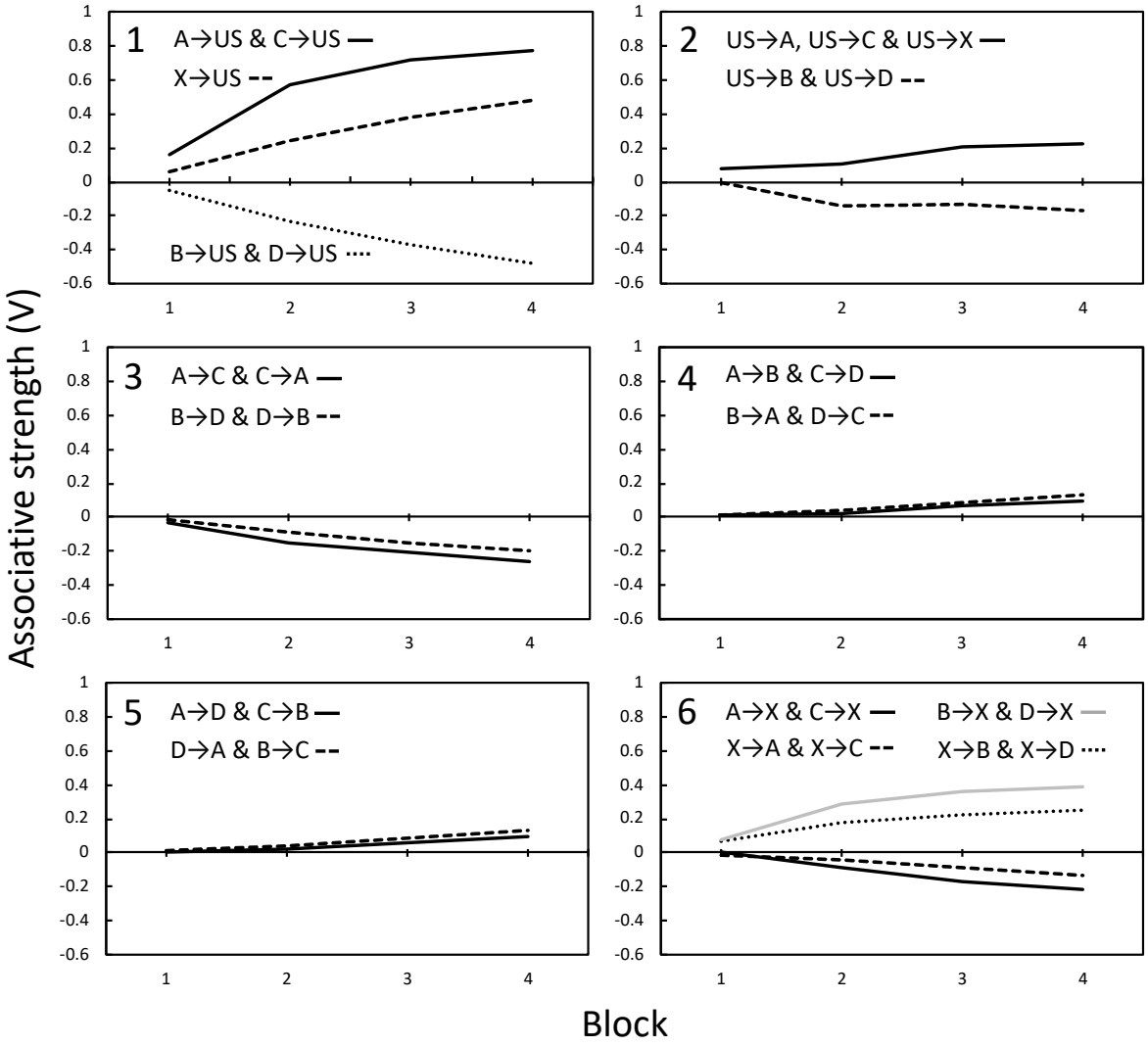

Figure 2. Simulations of stage 1 training for Design 1 that involved 5 trial types: A→US, C→US, X→US, BX→no US, and DX→US. The panels show the output values for the associative strengths (V) of the 30 potential binary associations involving the CSs (A, B, C, D, and X) and the US. Associations that are formally equivalent, within the experimental design, are grouped to simplify presentation.

In addition to the 10 potential links involving the US there is the potential for the 20 links involving pairs of the 5 CSs (A, B, C, D and X) to change over the course of training. Panel 3 shows the output values for the reciprocal links between A and C, and between B and D. Inspection of the panel shows that A and C develop reciprocal inhibitory links with one another. This reflects the fact that when, for example, A is present (i.e., on A→US trials) the error term for the A→C association will be negative,

because the US has excitatory association with C (see panel 2). Similarly, reciprocal inhibitory associations develop between B and D. This reflects the fact that when, for instance, B is presented on BX→no US trials, the error term for the B→D association will be negative, because X has excitatory association with D, formed on the DX→no US trials (see panel 6).

Panel 4 presents the output values for the reciprocal links between A and B and between the equivalently treated C and D. The links from A to B and from C to D become excitatory. This prediction is a counterintuitive one: After all, neither A and B nor C and D have been paired. In fact, the prediction is equivalent to one made by the Rescorla-Wagner model in the context of a second-order conditioned inhibition procedure. If CS1 is paired with a conditioned inhibitor (CS2; i.e., a stimulus with negative associative strength) then CS1 should develop an excitatory association with the US. This is the case because $\sum V_{TOTAL\text{-}US}$ will take a negative value (reflecting $V_{CS2\text{–}US}$), and the error term will be positive in: $\Delta V_{CS1\text{-}US} = \alpha\beta(0\text{–}\sum V_{TOTAL\text{-}US})$. Of course, in the case of second-order conditioned inhibition (and excitation), the Rescorla-Wagner model needs to rely on the (informal) idea that associative chains (or indeed mediated learning) can outweigh the predicted effects on direct conditioning (for a formal application of HeiDI to higher-order conditioning; see Honey & Dwyer, 2021ab). In the case under consideration here, the predicted excitatory association between A and B, for example, reflects the fact that on a reinforced A→US trial the US will inhibit B (see panel 2) and the error term for the change for the A→B association will be positive (i.e., $0\text{–}V_{US\text{-}B}$, with $V_{US\text{-}B}$ itself being negative). The reciprocal links from B to A and from D to C also become excitatory; because on an BX→no US trial, for example, X will inhibit A (see panel 6) and the error term for the change in the B→A association will be positive (i.e., $0\text{–}V_{X\text{-}A}$, with $V_{X\text{-}A}$ being negative). X will inhibit A, for example, because the error

term for the X→A association on X→US trials will be negative, as a result of the excitatory US→A (formed on A→US trials; see panel 2).

Panel 5 shows that the output values for the reciprocal associations between A and D, and between C and B, become positive over the course of training. The basis for these changes is relatively simple. For example, the error term for the A→D association, on an A→US trial, will be positive because the US will inhibit D (see panel 2; i.e., $0–V_{US\text{-}D}$, with $V_{US\text{-}D}$ itself being negative). Similarly, the error term for the D→A association will be positive on DX→no US trials, because X will inhibit A (see panel 6; i.e., $0–V_{X\text{-}A}$, with $V_{X\text{-}A}$ being negative). Finally, panel 6 depicts the reciprocal links between X to the 4 remaining CSs (A/C→X, X→A/C, B/D→X and X→B/D). Of these links, those involving A/C and X become inhibitory. They do so because, for example, the error term for the A→X association on A→US trials will be negative, given the fact that the US→X association is positive (see panel 2). Links involving B/D and X become excitatory because they are paired on the nonreinforced compound trials.

Simulations of how reinforced AB trials impact AD and BC in Design 1. Figure 3 provides a summary of the impact of each AB→US trial on the test compounds (AD and BC), and the key changes in the individual (reciprocal) associations that contribute to the differences between AD and BC. Panel 1 shows the combined strength of the reciprocal associations for the two compounds, calculated using Equations 7 and 8.

$$V_{COMB\ AD} = \Sigma V_{AD\text{-}US} + \left(\tfrac{1}{c}.\Sigma V_{AD\text{-}US} \times \left(V_{US\text{-}A} + V_{US\text{-}D}\right)\right) \quad \text{(Equation 7)}$$

$$V_{COMB\ BC} = \Sigma V_{BC\text{-}US} + \left(\tfrac{1}{c}.\Sigma V_{BC\text{-}US} \times \left(V_{US\text{-}B} + V_{US\text{-}C}\right)\right) \quad \text{(Equation 8)}$$

It is evident that after the first trials, on which the AD and BC values are necessarily equivalent given the counterbalanced nature of the simulations, BC has

higher output values than AD. Panel 3 confirms that this effect is a consequence of a more marked increase in the US→B than the US→A associations, because while V_{US-B} is 0 at the outset of compound conditioning, V_{US-A} is positive. Inspection of panel 3 also confirms that the increases in the A→US and B→US associations are equal, for the same reasons as the Rescorla-Wagner model: Without the additional features of HeiDI (e.g., the reciprocal US→CS associations together with within-compound associations), the effects reported by Rescorla (2000) would not be evident.[3] Panel 2 shows the effect of adding the influence of the chains activated by AD and BC to the values in panel 1: AD→C→US, AD→B→US, AD→X→US, BC→A→US, BC→D→US, and BC→X→US. The value of these chains (in units of V) is calculated in the manner shown in Equation 6, but using the combined strengths with which the compounds (e.g., AD) activate their (potential) associates (e.g., C, B, and X). While the AB→US trials have relatively little impact on the efficacy of these chains (see description of panel 4 below), their overall effect is to increase the absolute difference between the AD and BC compounds (relative to panel 1).[4]

[3]The potential reduction in the US→C association, which could have resulted from the presentation of the US on the AB→US trials, is minimized because A comes to inhibit C during stage 1 (cf. panel 3 of Figure 2; Honey et al., 2020a, p. 842). Otherwise, this reduction would counteract the impact of the increase in the US→B association on the BC test compound.

[4]The differential impacts of the reciprocal associations and associative chains (based on within-compound associations) on the output values for AD and BC, also applies when B is novel at the outset of reinforced AB trials (Experiments 1 and 3, Rescorla, 2001).

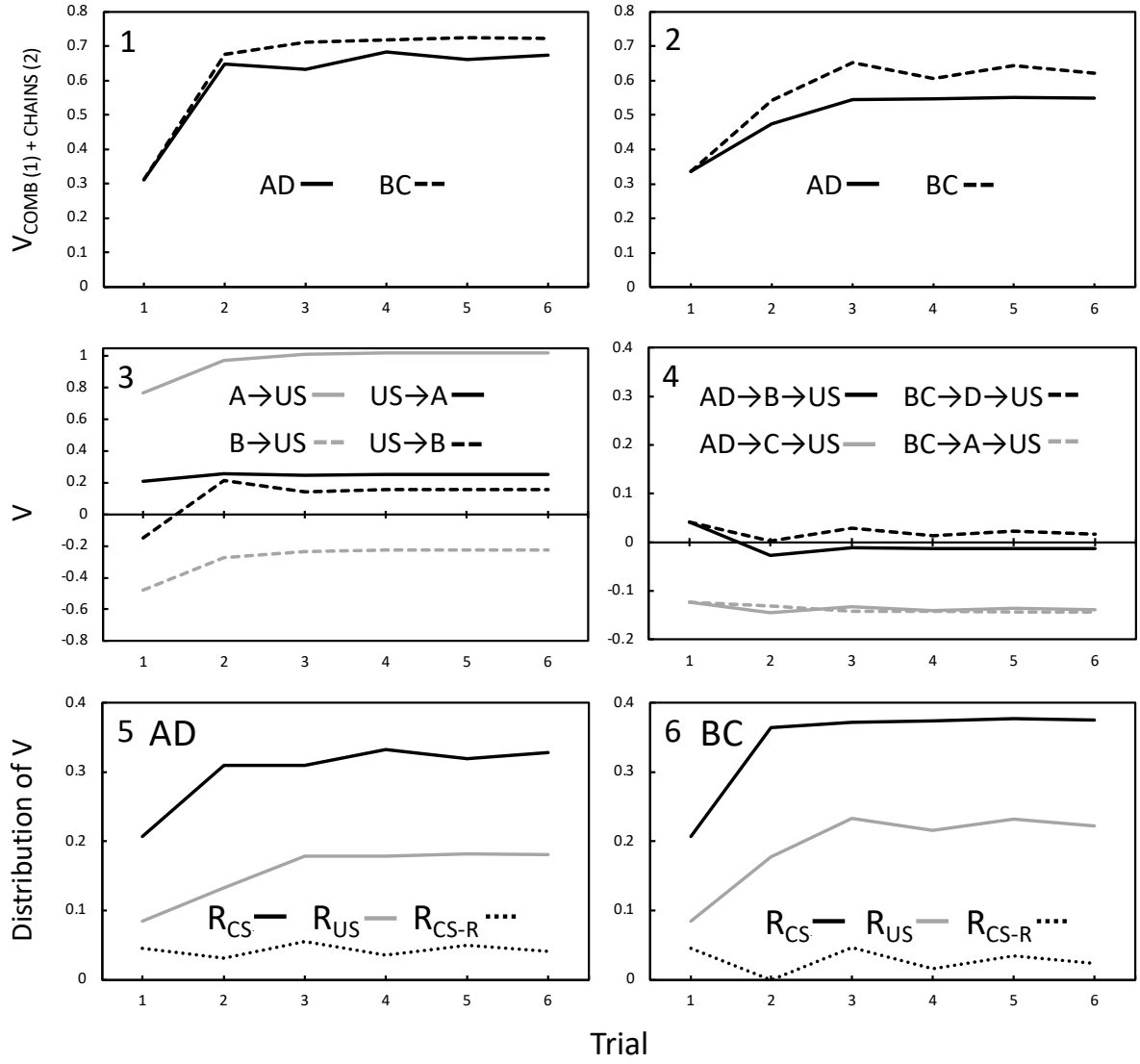

Figure 3. Simulations of how reinforced AB trials during stage 2 of Design 1 impact the associative strengths (V) of AD and BC. (The stage 1 trial types were: A→US, C→US, X→US, BX→no US, and DX→US.) Panel 1 shows the V_{COMB} output values for AD and BC and panel 2 combines the V_{COMB} values with the V_{CHAINS} output values for AD and BC. Panel 3 presents the output values for the associative strengths (V) of reciprocal associations between A/B and the US, and panel 4 depicts the chains from AD to B and C and from BC to D and A. Panel 5 shows the distribution of the $V_{COMB+CHAINS}$ values (from panel 2) into R_{CS}, R_{CS-R} and R_{US} for AD and panel 6 shows the equivalent values for BC.

Panel 4 shows how the individual chains change over the course of AB→US trials; the chains involving X are not shown because they are necessarily equivalent for the two compounds, AD and BC. The principal difference is that the BC→D→US chain is consistently excitatory whereas the AD→B→US chain begins as excitatory but

becomes inhibitory. The basis for these changes is complex: The BC→D association has an inhibitory value, while the AD→B association starts with an inhibitory value but becomes excitatory. This reflects the fact that when the inhibitory value of the BC→D association is multiplied by the inhibitory $V_{COMB\ D\text{-}US}$ value it results in a positive output value, while when the excitatory AD→B value multiplied by the inhibitory $V_{COMB\ B\text{-}US}$ value results in a negative output value. The AD→B association takes a positive value because the A→B association has been strengthened across AB→US trials (outweighing the consistently inhibitory D→B link); while the equivalent increase in the B→A association is blocked by the US→A association.

The output values in panel 2 confirm that the combined values (V) of the direct associations (V_{COMB}) and associative chains (V_{CHAINS}) are lower for AD than BC. Panels 5 and 6 depict how V for AD and BC is distributed into three components: R_{CS}, $R_{CS\text{-}r}$, and R_{US}. These components are held to affect the extent to which responding is influenced by the CSs that are present (R_{CS}), the associatively retrieved CSs ($R_{CS\text{-}R}$), and the associatively retrieved US (R_{US}). They represent predictions concerning how the differences in V are evident in behavior. The proportion terms, according to which V is distributed, are calculated in an analogous way to Equations 4 and 5. In this case, the proportions are derived from the combined α values of the CSs that are present (for R_{CS}), the combined strengths with which absent CSs are being associatively retrieved (for $R_{CS\text{-}R}$), and the strength with which the US is being retrieved (for R_{US}). $R_{CS\text{-}R}$ is derived in an analogous way to R_{US}, but using the combined strengths with which the CSs that are present are activating those that are not: $R_{CS\text{-}R} = \alpha_{CS\text{-}R}/(\alpha_{CS}+\alpha_{CS\text{-}R}+\beta) \times V_{COMB+CHAINS}$; where $\alpha_{CS\text{-}R} = \sum V_{CS\ PRESENT\text{-}CS\ ABSENT}$ (see Honey & Dwyer, 2021ab). Comparison of these proportions, across panels 5 and 6, shows that with the current set of parameters they are generally lower for AD (panel 5) than BC (panel 6), for R_{CS} and

R_{US}, but not for $R_{CS\text{-}R}$. The prediction is that the effect will be evident in both CS-oriented and US-oriented responding.

Simulations of how nonreinforced AB trials impact AD and BC in Design 1. Figure 4 shows the equivalent set of simulations to those presented in Figure 3, but for the case where AB was nonreinforced in stage 2. Inspection of panel 1 confirms that the V_{COMB} scores for AD and BC decline as the result of nonreinforced AB trials, but that they no longer differ (the lines overlie one another). Of course, this is no surprise: The adoption of a simplified treatment of nonreinforcement used by Rescorla and Wagner (1972; i.e., setting β to 0), means that there is no equivalent to the US on nonreinforced AB trials to undergo differential change with the components of AB (i.e., there is no "No US", Konorski, 1967; see Honey et al., 2020a). Panel 3 confirms that the output values for the associations between A and the US, and B and the US, decrease across training trials and do so equivalently, while the reciprocal US$\rightarrow$A and US$\rightarrow$B associations are left unchanged by the nonreinforced AB trials (because $\beta_{US} = 0$). Panel 2 shows that when the V_{COMB} output values are combined with those from the associative chains, the resulting overall output scores for BC are higher than for AD.[5] According the HeiDI model, associative chains alone form the basis of the difference between AD and BC when the AB compound is nonreinforced; with the obvious prediction that the difference between AD and BC should be reduced by manipulations that weaken these associative chains.

Panel 4 presents the detailed basis for the origin of this differential effect of the chains on AD and BC over the course of AB$\rightarrow$US trials; the chains involving X are again not shown because they are necessarily equivalent for AD and BC. The principal

[5] The differential impact of associative chains (based on within-compound associations) on test output values for AD and BC, also applies when B is novel at the outset of nonreinforced AB trials (Experiments 2 and 4, Rescorla, 2001).

difference is that across AB training trials, both AD chains converge on inhibitory values whereas those involving BC converge on excitatory values. Most notably, the net effect of the associations from B and C is to excite the excitatory $V_{COMB\ A\text{-}US}$. This is because the strengthening of the excitatory B→A association across stage 2 outweighs the consistently inhibitory C→A link. In contrast, the net effect of the associations from A and D is to excite the inhibitory $V_{COMB\ B\text{-}US}$; this is because the strengthening of the excitatory A→B association across stage 2 outweighs the consistently inhibitory D→B link. There is relatively little change in the AD→C→US chain (or the BC→D→US chain) across AB- trials in stage 2, because by the end of stage 1 A→C is inhibitory and B→C is excitatory, with the result that there is little error in the prediction of C on nonreinforced AB trials. Similarly, there is little error in the prediction of D on AB trials, because A→D is excitatory and B→D is inhibitory. Finally, comparison of panel 5 with panel 6 in Figure 4 shows that in contrast to when AB had been reinforced (see panels 5 and 6 in Figure 3), when AB has been nonreinforced the HeiDI model predicts that the difference between AD and BC should be most evident in CS-oriented responding (which depends on R_{CS}) rather than US-oriented responding (which depends on R_{US}). This simply reflects the fact that R_{US} is dependent on the retrieved value of the US, which is reduced by extinction, unlike the perceived intensity of the CS (see Iliescu et al., 2018).

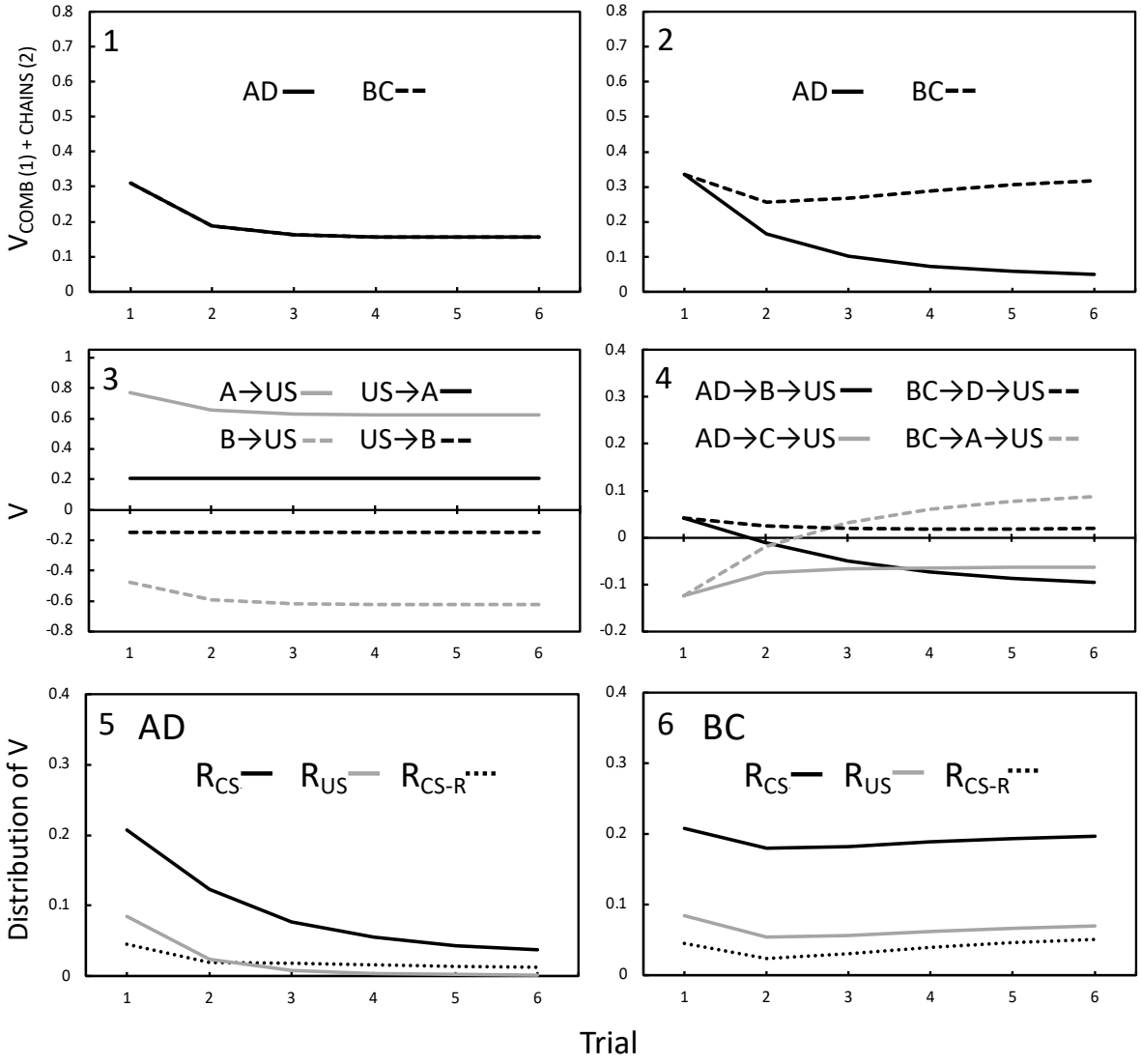

Figure 4. Simulations of how nonreinforced AB trials during stage 2 of Design 1 impact the associative strengths (V) of AD and BC. (The stage 1 trial types were: A→US, C→US, X→US, BX→no US, and DX→US.) Panel 1 shows the V_{COMB} output values for AD and BC and panel 2 combines the V_{COMB} values with the V_{CHAINS} output values for AD and BC. Panel 3 presents the output values for the associative strengths (V) of reciprocal associations between A/B and the US, and panel 4 depicts the chains from AD to B and C and from BC to D and A. Panel 5 shows the distribution of the $V_{COMB+CHAINS}$ values (from panel 2) into R_{CS}, R_{CS-R} and R_{US} for AD and panel 6 shows the equivalent values for BC.

Simulations of stage 1 for Design 2. The results from the simulations of the first stage of training for Design 2 are shown in Figure 5. Panel 1 shows the output values for the 4 CS→US associations, and panel 2 depicts the corresponding output values for the 4 US→CS associations. Inspection of panel 1 confirms that A and C develop strong

associations with the US, while stimulus B and stimulus D come to have negative associative values. Panel 2 shows that while the US develops excitatory associations with A and C, it develops inhibitory associations with B and D. This is because on A→US trials, the error term for the US→B association will be negative given the fact that A has an excitatory association with B (formed on AB→no US trials). Also, on C→US trials the error term for the US→D association will be negative, since C has an excitatory association with D (formed on CD→no US trials).

In addition to the 8 potential associations involving the US, there are 12 potential associations involving pairs of the 4 CSs (A, B, C, and D), which could change over the course of training. Panel 3 depicts the reciprocal links between A and C, and between B and D. For the same reasons as in Design 1, A and C develop reciprocal inhibitory links with one another. Inhibitory associations between B and D develop because, for example, the error term for the B→D association is negative on AB→no US trials, as the A→D association has excitatory strength (see panel 5 and the description in the next paragraph). Panel 4 shows the reciprocal links between the A and B and between the equivalently treated C and D. The links from A to B, and from C to D, become excitatory; because they are presented on the same trial. The fact that the A→B and C→D associations are weaker than the B→A and D→C associations reflects the fact that the former but not the latter are subject to extinction on A→US and C→US trials (cf. Rescorla, 2000, 2001).

Panel 5 shows that the reciprocal associations between A and D, and between C and B, become positive over the course of training. This is because, for example, the error term for the A→D association is positive on A→US trials, given the fact that the US→D association is inhibitory (i.e., $0–V_{US\text{-}D}$, with $V_{US\text{-}D}$ being negative). The error term for the D→A association, for example, will be positive since on CD→no US trials the

C→A association has a negative value (see panel 3; i.e., 0–$V_{C\text{-}A}$, with $V_{C\text{-}A}$ being negative).

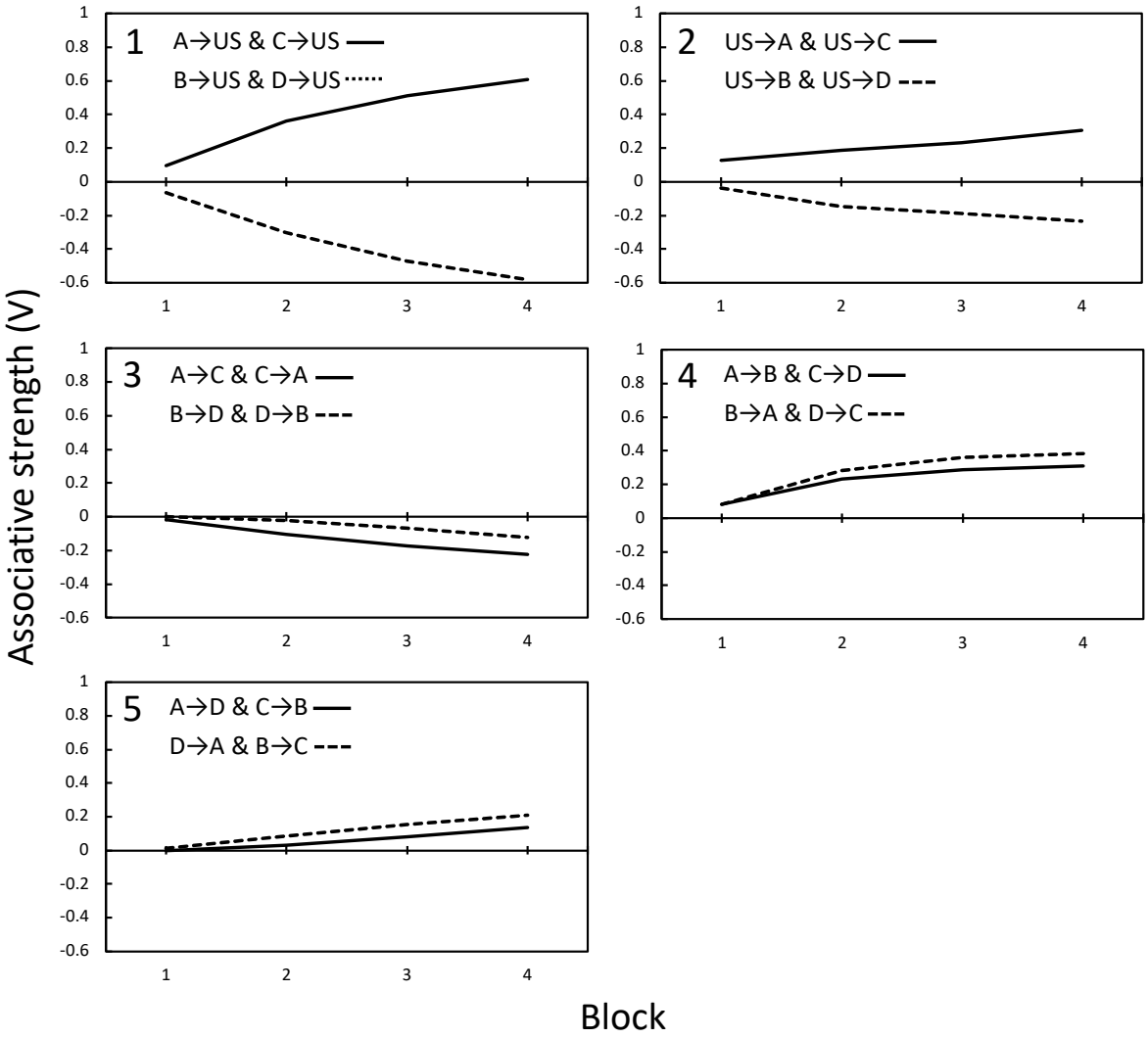

Figure 5. Simulations of stage 1 training for Design 2 that involved 4 trial types: A→US, C→US, AB→no US, and CD→no US. The panels show the output values for the associative strengths (V) of the 20 potential binary associations involving the CSs (A, B, C, and D) and the US. Associations that are formally equivalent, within the experimental design, are grouped to simplify presentation.

Simulations of how reinforced AB trials impact AD and BC in Design 2. Figure 6 shows the impact of each AB→US trial on the two test compounds: AD and BC. Panel 1 shows the combined strength of the reciprocal associations for the two compounds. After the first trials, when AD and BC values are equivalent, BC has higher output values than AD. This is a consequence of both an increase in the US→B association,

but also a decrease in the US→A association, which reflects the fact that the sum of the US→A and B→A associations exceeds the asymptote (i.e., c.α_A = .40). As with Design 1, reductions in the US→C association are constrained by the fact that A comes to inhibit C during stage 1. Panel 2 shows the effect of adding the influence of the chains activated by AD and BC: AD→C→US, AD→B→US, BC→A→US, and BC→D→US. The addition of the chains reduces the difference between AD and BC.[6] This primarily reflects the emergence of more marked negative values for BC→D→US than AD→B→US chains. This difference, in turn, reflects the fact that $V_{COMB\ D\text{-}US}$ has a more negative value than $V_{COMB\ B\text{-}US}$ resulting from the increase in the strength of the B→US association. Comparison of panels 5 and 6 shows how the combined influence of the direct associations and associative chains are distributed into three components: R_{CS}, $R_{CS\text{-}R}$ and R_{US}. The principal difference is the lower values for R_{CS} for AD than BC, with a smaller effect on R_{US}, and the opposite effect on $R_{CS\text{-}R}$. That is, the effect is predicted to be most marked for CS-oriented responding (which reflects the value of R_{CS}). This prediction could be readily assessed using procedures where CS-oriented and US-oriented responding can be readily distinguished (cf. Iliescu et al., 2018, Iliescu, Dwyer & Honey, 2020; Patitucci et al., 2016).

[6]It is worth noting that the reduction in the difference between AD and BC, observed when the influence of the chains is included (panel 2), is not observed when the efficacy of the chains is modulated by the similarity of the perceived intensity of retrieved component (e.g., B via $V_{A\text{-}B}$) to its corresponding conditioned intensity ($\alpha_{B;}$ Honey & Dwyer, 2021ab). In the other simulations presented in this paper, however, such modulation reduces the size of the effect, with the result that the differences between AD and BC become more comparable when modulation by similarity is included throughout.

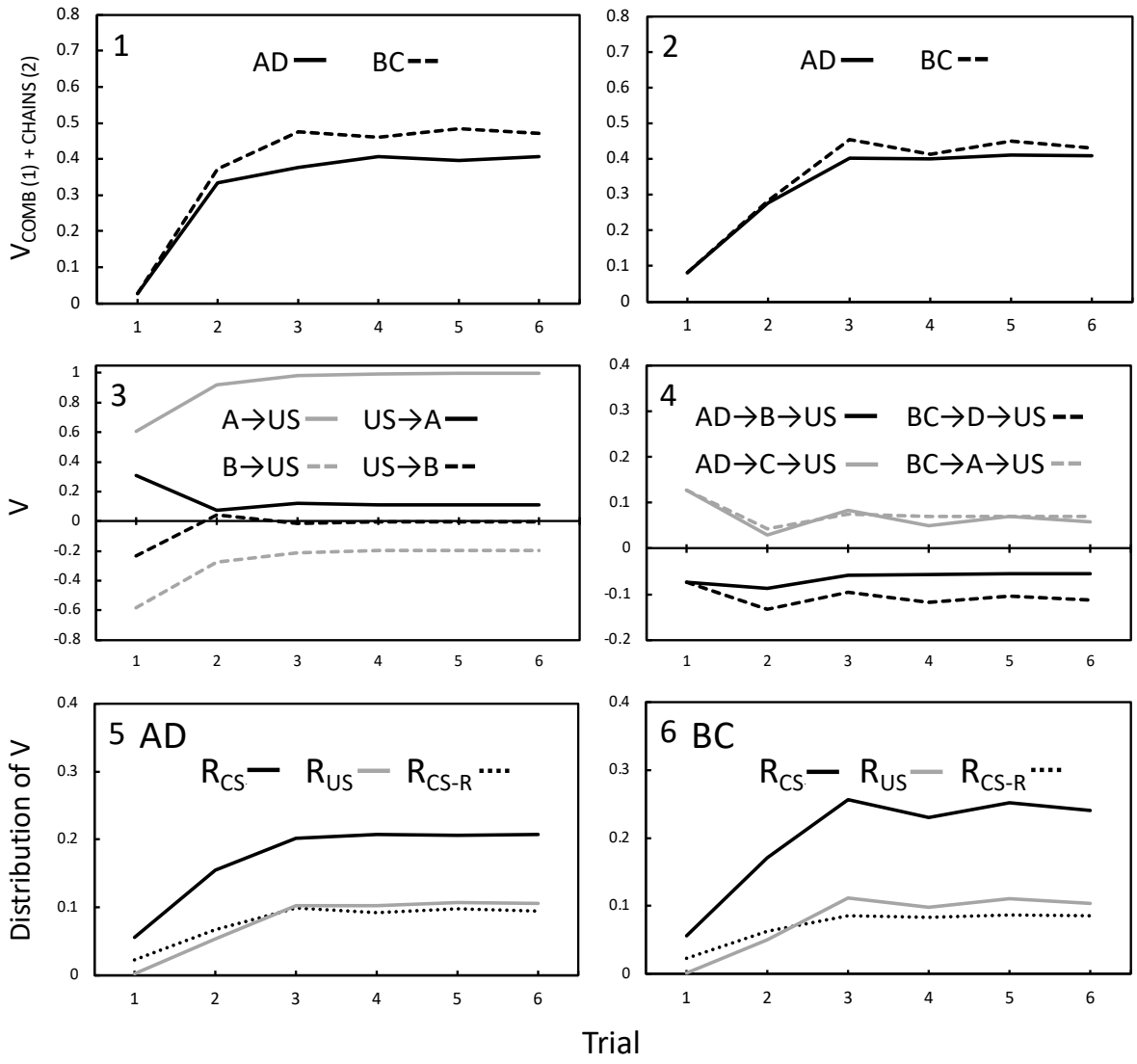

Figure 6. Simulations of how reinforced AB trials during stage 2 of Design 2 impact the associative strengths (V) of AD and BC. (The stage 1 trial types were: A→US, C→US, AB→no US, and CD→no US.) Panel 1 shows the V_{COMB} output values for AD and BC and panel 2 combines the V_{COMB} values with the V_{CHAINS} output values for AD and BC. Panel 3 presents the output values for the associative strengths (V) of reciprocal associations between A/B and the US, and panel 4 depicts the chains from AD to B and C and from BC to D and A. Panel 5 shows the distribution of the $V_{COMB+CHAINS}$ values (from panel 2) into R_{CS}, R_{CS-R} and R_{US} for AD and panel 6 shows the equivalent values for BC.

Simulations of how nonreinforced AB trials impact AD and BC in Design 2. Figure 7 shows the equivalent set of simulations to those presented in Figure 6, but for the case in which AB was not followed by a US in stage 2; noting the accompanying truncating of the scale. The nonreinforced AB trials result in small reductions in the

output values for the A→US and B→US associations (because the error term is negative, but close to 0), but no changes in the output values for the reciprocal US→A and US→B (because the US is absent and $\beta_{US} = 0$). Inspection of panel 1 of Figure 7 confirms that the V_{COMB} scores for AD and BC decline as the result of nonreinforced AB trials, but do not differ (their output functions overlie one another); with panel 3 showing the individual reciprocal associations between A and B and the US. In contrast, panel 2 shows that when the V_{COMB} output values for AD and BC are combined with those from the associative chains, the resulting overall scores for AD are lower than those for BC. This difference reflects the fact that during stage 1 the A→B association is weakened by the A+ trials, but the B→A association is not, and both associations are strengthened during the nonreinforced AB trials. This allows the AD compound to borrow more inhibition from B (see panel 4). Comparison of panels 5 and 6 shows that R_{CS}, $R_{CS\text{-}R}$, and R_{US} output values are higher for BC than AD, with this difference being most evident for R_{CS}. Thus, in contrast to when Design 1 was simulated, the simulations of Design 2 show that the difference between AD and BC was consistently more marked for CS-oriented responding (which depends on R_{CS}) than US-oriented responding (which depends on R_{US}). In principle, it would be relatively simple to assess the accuracy of this prediction within some conditioning paradigms (e.g., Patitucci et al., 2016). For example, in rat autoshaping procedures where approaching and interacting with the lever CS (i.e., sign-tracking) has been aligned to R_{CS} while approaching the food well (i.e., goal-tracking) has been aligned with R_{US} (see Iliescu et al., 2020).

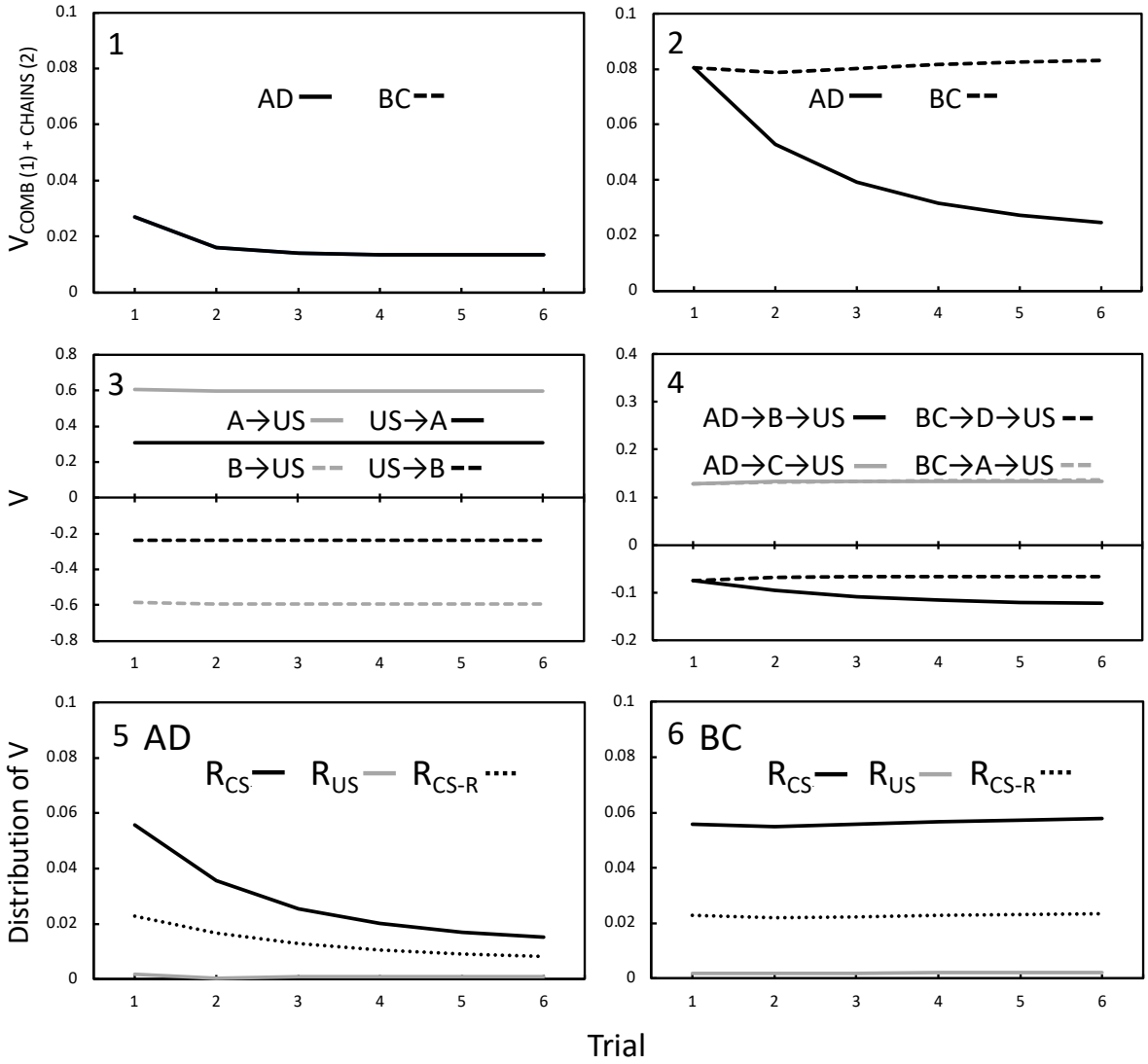

Figure 7. Simulations of how nonreinforced AB trials during stage 2 of Design 2 impact the associative strengths (V) of AD and BC. (The stage 1 trial types were: A→US, C→US, AB→no US, and CD→no US.) Panel 1 shows the V_{COMB} output values for AD and BC and panel 2 combines the V_{COMB} values with the V_{CHAINS} output values for AD and BC. Panel 3 presents the output values for the associative strengths (V) of reciprocal associations between A/B and the US, and panel 4 depicts the chains from AD to B and C and from BC to D and A. Panel 5 shows the distribution of the $V_{COMB+CHAINS}$ values (from panel 2) into R_{CS}, R_{CS-R} and R_{US} for AD and panel 6 shows the equivalent values for BC.

Summary of simulations. The model simulated here, HeiDI, is founded on allowing reciprocal associations to form between all of the components of a trial (Honey et al., 2020a), and for these associations to influence behavior directly (through binary associations) and indirectly (through associative chains; see also, Honey & Dwyer,

2021ab). The requisite associative structures are generated by learning rules that include only 2 free parameters (α and β). The way in which these structures generate different conditioned behaviors reflects the relative intensities of the CSs and the other stimuli that they retrieve (other CSs or the US). The model was developed to address an issue that had been relatively neglected: the translation of learning into performance (see Rescorla, 1988). However, HeiDI has general applicability. Here, we have focused on the analysis that it provides for the results presented by Rescorla (2000; see also, Rescorla, 2001). It accounts for these results not through the changes in the way in which the pooled error term is implemented within the learning rules, but instead through the consistent application of a given rule to all components of the trial (e.g., A, B, and the US). For example, when AB is paired with a US the US$\rightarrow$B association undergoes a greater increase in associative strength than the US$\rightarrow$A association, which provides a direct basis for BC to elicit greater conditioned responding than AD. However, the AB trials also have an indirect influence on responding to AD and BC through changes in the reciprocal (within-compound) associations between A and B, which allow AD and BC to recruit associative strength from stimuli that are absent. In fact, when AB is nonreinforced, the simulations reveal that the difference between AD and BC is entirely driven by the impact of such indirect influences, mediated by within-compound associations.

General Discussion

The principal contributions of the Rescorla-Wagner model derived from its use of a learning rule with a pooled error term. This rule has gained significant traction beyond its original application to Pavlovian conditioning, including in human learning (e.g., Rumelhart, Hinton, & Williams, 1986; Shanks, 1985) and neuroscience (e.g., Schultz,

Dayan, & Montague, 1997; see also, Maes, Sharpe et al., 2020). The possibility that any one of its central tenets does not hold is therefore of general importance. Rescorla (2000; see also Rescorla, 2001) investigated one such tenet: When equally salient stimuli are conditioned as components of the same compound they should undergo equivalent changes in associative strength. The experiments involved ingenious designs in which changes in associative strength could be assessed in CSs that elicited quite different levels of conditioned responding. The results were clear, reliable and replicable: They suggested that the tenet does not hold under a variety of conditions (see also, Allman & Honey, 2004; Allman, Ward-Robinson & Honey, 2005; Fam, Westbrook & Holmes, 2017). Instead, it appeared that the stimulus with associative strength that differed most from the asymptote determined by the outcome of the trial was subject to the greatest associative change. In effect, the stimulus that contributed most to the prediction error appeared to be preferentially changed in order to reduce it. Rescorla (2000) concluded that "*The implication is that a wide variety of models will require some modification in their learning rule*." He proceeded to consider a number of possible alternative modifications to extant models, none of which were without limitations.

Here, we present a detailed application of a different approach derived from a recent model of Pavlovian learning and performance: HeiDI (Honey et al., 2020a). The success of this approach is not based on modifying the essence of the Rescorla-Wagner learning rule (Equation 0), but rather from a different analysis of the associative structures acquired during (compound) conditioning; with an allied analysis of how those structures affect performance. The general idea that (reciprocal) links are formed between the components of a compound (e.g., A and B) is relatively uncontentious, and the role of such within-compound associations has been investigated in a variety of

contexts by Rescorla and his colleagues (e.g., Durlach & Rescorla, 1980; Rescorla & Colwill, 1983). However, the inclusion of links from the US to the components of the compound is more contentious (but see, Asratian, 1965). One theoretical constraint on their inclusion is the proliferation of parameters that it could entail, which is avoided by the learning rules in HeiDI (Equations 1 and 2). Moreover, the appeal to reciprocal associations goes some way towards providing an analysis for why Pavlovian conditioning results in CS-oriented and US-oriented behaviors. In fact, our simulations reveal that HeiDI predicts that Rescorla's (2000) results would have been differently evident in these two types of conditioned behavior, had they been concurrently measured within different versions of the experimental designs. It must be acknowledged, however, that this qualitative analysis of the translation of learning into different forms of behavior awaits a more quantitative approach, which would require the inclusion of a specification of the nature of the interactions between processes that more directly generate behavior (i.e., the response-generating units in the model; see Honey et al., 2020a).

The simulations presented here show how the adoption of a fully connected associative structure provides the basis for an analysis of the results reported in Rescorla (2000; Rescorla, 2001). We are aware that the resulting complexity this structure brings might be considered a sufficient reason to look elsewhere for simpler alternatives; for example, some combination of a separate error term with a pooled error term (Rescorla, 2000). However, the essence of the analysis we have developed is a simplified or rationalized trial-level learning rule (with a pooled error term) that is applied in a consistent way involving the stimuli that are present on a trial. The model is, in these respects, less complex than the Rescorla-Wagner model or indeed the use of a combined separate and pooled error term. HeiDI includes no (hidden or arbitrary)

theoretical assumptions about the order in which the stimuli are presented being important; and the resulting associative structures provide a natural way to accommodate the fact that conditioned behavior can be much more complex than conventional theories allow (e.g., Rescorla & Wagner, 1972; see also, Mackintosh, 1975; Pearce, 1994; Pearce & Hall, 1980; Stout & Miller, 2007; Wagner, 1981; Wagner & Brandon, 1989). The model also allows those processes involving the formation of associations, more generally, to be readily integrated with those underlying Pavlovian conditioning (see also, McLaren et al., 1989; McLaren & Mackintosh, 2000). In fact, in applying HeiDI more broadly (e.g., to higher-order conditioning), it has become clear that some phenomena (e.g., second-order conditioning) are more parameter dependent than other related phenomena (e.g., sensory preconditioning); and that the choice of parameters influences whether observations that appear inconsistent with one another (second-order conditioning and conditioned inhibition; Holland & Rescorla, 1975; Rescorla, 1976) can be generated simultaneously (see Honey & Dwyer, 2021ab). These predictions are important targets for future research.

Summary and conclusions. The Rescorla-Wagner model changed how Pavlovian conditioning is understood. With only simplifying assumptions about what was associated and how learning was translated into performance, it provided an elegant account of an impressive range of phenomena. It could be argued that the simplicity of these assumptions has served associative theorists well for 50 years. However, by elaborating a model that more fully reflects what is now known about the nature of the associative structures formed during Pavlovian conditioning and their expression in behavior, not only can a much broader range of findings be accommodated, but new directions for future research become apparent (see Honey et al., 2020abc; Honey & Dwyer, 2021ab). These elaborations were inspired by the

research conducted by Rescorla and the prescient questions he posed: The fact that HeiDI provides an explanation for results that appeared to undermine a central tenet of the Rescorla-Wagner model would likely have drawn a wry smile (😏).

Author note

All authors contributed to the ideas presented in this article and to its preparation for publication. The development of the ideas, together with our aligned research, was supported by the BBSRC (UK; BB/T004339/1; PI: RCH). HeiDI is available as an open source app (https://ynnna.shinyapps.io/HeiDI_model/); and the Excel sheets used to generate the simulations presented in this article are available on request from R.C. Honey. Correspondence about this article should be addressed to R.C. Honey: honey@cardiff.ac.uk.

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