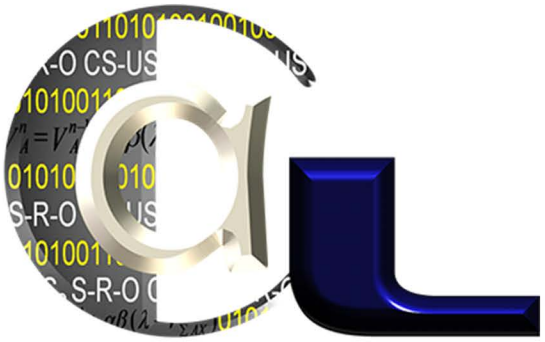

Technical Notes

Hall and Rodríguez model as a particular case of the Pearce and Hall model: A formal analysis

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Hall and Rodríguez model as a particular case of the Pearce and Hall model: A formal analysis

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This report introduces a formal comparison between Hall and Rodríguez's elaboration of the Pearce and Hall model and the original. It shows mathematically and with a set of simulations that with a provision to set an initial excitatory value in Pearce and Hall's algorithm, both models are equivalent in structure and that they result in identical predictions for non-reinforced exposure conditions. Unlike Pearce and Hall's model however, Hall and Rodríguez's lacks an inhibitory mechanism to counteract excitation confining its scope to non-reinforced trials. Pearce and Hall's model with an initial excitatory value thus subsumes Hall and Rodríguez's.

Learning in Pavlovian conditioning is conceptualized as the formation of an association between the representations of two stimuli: a signal (conditioned stimulus, CS) and an outcome (unconditioned stimulus, US). Most current conditioning theories assume that the growth in the associative strength (V) of the link formed between the stimuli is proportional to the degree of error in predicting the outcome: the more accurately the CS predicts the US, that is, the smaller the error, the greater the increase in associative strength between the two stimuli (see e.g., Pearce & Bouton, 2001).

1. Pearce and Hall model

Pearce and Hall (1980) proposed an error correction model driven by the changes in the CS processing as a consequence of learning. In their model, the associability (α) of a CS (i), a factor

signifying its individual rate of conditioning to a US, varies according to the error: as the error decreases, so does the stimulus's associability.

Similar to the Rescorla-Wagner model (Rescorla & Wagner, 1972), the prediction error in Pearce and Hall's model denotes the discrepancy between the total net most recent outcome prediction, $\sum_{i=A}^{Z} V_{\text{NET}_i}^{n}$ (the US expectation at the end of trial *n*) and the current outcome value, λ^{n+1}. With each CS-US pairing the error is reduced, and the strength of their associative connection increased until learning is complete when the CS fully predicts the outcome, at which point, and unlike the Rescorla-Wagner model, the CS associability approaches zero. Hence, according to Pearce and Hall's model – later revised by Pearce, Kaye, and Hall (1982), whose version is used henceforth – a CS loses associability as learning progresses and the error is reduced.

Formally, the associability of a stimulus *i* is estimated as follows:

$$\alpha_i^{n+1} = \gamma \left| \lambda^{n+1} - \sum_{i=A}^{Z} V_{\text{NET}_i}^{n} \right| + (1-\gamma)\alpha_i^{n} \qquad (1)$$

$$\textit{where} \quad 0 \le \gamma \le 1$$

The γ parameter controls the relative influence of the preceding trials. Thus, for $\gamma \approx 1$, α is determined mainly by the events of the immediately preceding trial *n*; if $\gamma \approx 0$, α is determined almost exclusively by earlier trials.[1]

The total net prediction is defined in the model as the difference between the overall excitatory, $\sum_{i=A}^{Z} V_i^n$, and inhibitory, $\sum_{i=A}^{Z} \overline{V_i^n}$, predictions, following Konorski's (Konorski, 1967) analysis in which inhibition entails a link functionally opposed to the excitatory association.

Thus, the associability of a stimulus could also be expressed as:

$$\alpha_i^{n+1} = \gamma \left| \lambda^{n+1} - \left(\sum_{i=A}^{Z} V_i^n - \sum_{i=A}^{Z} \overline{V_i^n} \right) \right| + (1-\gamma)\alpha_i^{n} \qquad (2)$$

A learning episode strengthens the excitatory link if the outcome expectation is smaller than the outcome value, λ^{n+1}. Contrarily, it adds to the inhibitory association if the US prediction is larger than the actual outcome. Hence, the effective inhibitory value of the outcome in inhibitory associations, $\left|\overline{\lambda^{n+1}}\right|$, is determined by the degree the expected outcome is disproved, and can be computed by subtracting the prediction from the US value, as follows:

[1] The original associability equation in Pearce and Hall (1980) can be considered as a particular case of the revised formula for $\gamma = 1$, which solution results in $\alpha_i^{n+1} = \left| \lambda^{n+1} - \sum_{i=A}^{Z} V_{\text{NET}_i}^{n} \right|$.

$$\overline{\lambda^{n+1}} = \lambda^{n+1} - \left(\sum_{i=A}^{Z} V_i^n - \sum_{i=A}^{Z} \overline{V_i^n} \right) \tag{3}$$

The amount of change in associative strength at each learning trial is governed by the following equations:

For excitatory associations $$\Delta V_i^{n+1} = \beta_E \cdot \alpha_i^n \cdot \lambda^{n+1} \tag{4}$$

For inhibitory associations $$\Delta \overline{V_i^{n+1}} = \beta_i \cdot \alpha_i^n \cdot \left| \overline{\lambda^{n+1}} \right| \tag{5}$$

Where β_E and β_I are parameters related to the US that can adopt values from 0 to 1.

Then, the total amount of excitatory and inhibitory strength at a given trial is given by the sum of the excitatory and inhibitory increments and the corresponding previous associative value.

For excitatory associations $$V_i^{n+1} = V_i^n + \Delta V_i^{n+1} \tag{6}$$

For inhibitory associations $$\overline{V_i^{n+1}} = \overline{V_i^n} + \Delta \overline{V_i^{n+1}} \tag{7}$$

Finally, the net associative strength of a stimulus *i* is computed subtracting the excitatory and inhibitory links' values.

$$V_{\text{NET}_i}^{n+1} = V_i^{n+1} - \overline{V_i^{n+1}} \tag{8}$$

Perhaps the most significant contribution of the Pearce and Hall model is its ability to account for slow acquisition observed in latent inhibition related procedures: conditioning to a stimulus previously exposed in isolation – or paired with a weaker US – is often delayed in comparison to conditioning to a novel, similar stimulus. According to the model's Equation (1) training on a series of consistent trials, *i.e.*, with a constant λ^{n+1} value, reduces the associability of the stimulus, therefore retarding subsequent learning. However well suited it might be to handle latent inhibition, Pearce and Hall's model is not the only model able to do so (e.g., Kokkola, Mondragón, & Alonso, 2014; McLaren & Mackintosh, 2000; Wagner, 1981), nor is it exempt of difficulties. In an attempt to cope with some of the drawbacks of the original model, Hall and Rodríguez (2010) presented an extension of the Pearce and Hall model focused exclusively on non-reinforced stimulus exposure.

2. Hall and Rodríguez elaboration

The core idea of Hall and Rodríguez's elaboration rests on the assumption that non reinforced stimulus exposure results in a distinctive learning process in which an animal learns about the absence of consequences of the stimulus, postulating the formation of an association between the CS representation and a generic *no-event* representation (**ne**). Activation of this no-event node

during preexposure requires a further critical assumption: the notion of a *preexisting* associate event (**e**).

Hence, presentations of any putative neutral stimulus (e.g., $i = A$, the CS) is likely to evoke the prospect of an associate event (an outcome) with a given strength $V_{e_i}^{n}$. During preexposure, the nonappearance of the expected consequence will lead to the formation of a new association, represented as $V_{ne_i}^{n}$, able to counteract the initial excitatory link, a process that parallels that suggested by Pearce & Hall (1980) to account for inhibitory learning. Thus, the growth of the no event association is defined as:

$$\Delta V_{ne_i}^{n+1} = s_i \cdot \alpha_i^{n} \cdot \lambda_{ne}^{n+1} \tag{9}$$

No rules are defined in this elaboration for changes in the strength of the preexisting associative link $V_{e_i}^{n}$, which, given the absence of an explicit outcome, must therefore remain unchanged. Consequently, the net associative strength will decline as $V_{ne_i}^{n}$ increases,

$$V_{\text{NET}_i}^{n} = V_{e_i}^{n} - V_{ne_i}^{n} \tag{10}$$

Similar to Pearce and Hall's original formulation, the effective value of the inhibitory reinforcer is defined as a function of the total net event expectancy[2],

$$\lambda_{ne}^{n+1} = \sum_{i=A}^{Z} V_{e_i}^{n} - \sum_{i=A}^{Z} V_{ne_i}^{n} \tag{11}$$

Akin to its predecessor, the associability of a stimulus declines when it consistently predicts its consequences,

$$\alpha_i^{n+1} = \left| \lambda_{e}^{n+1} - \left(\sum_{i=A}^{Z} V_{e_i}^{n} - \sum_{i=A}^{Z} V_{ne_i}^{n} \right) \right| \tag{12}$$

Provided that the reinforcing conditions remain unchanged, learning during exposure will cease as the no event strength $V_{ne_i}^{n}$ approaches the pre-existing event value $V_{e_i}^{n}$, at which point both λ_{ne}^{n+1} and α_i^{n+1} will fall to zero.

Hall and Rodríguez's extension spurred research in the latent inhibition paradigm, predicting new cue competition related effects, whose analysis lays outside the scope of Pearce and Hall's original proposal. Nonetheless, Hall and Rodríguez's elaboration does not propose an algorithm to compute how learning would actually develop during conditioning, and thus their predictions are in fact confined to non-reinforced trials. This entails a mismatch between simulated data and empirical results which require conditioning to follow non-reinforced stimulus exposure.

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[2] Hall and Rodríguez's model applies exclusively to non-reinforced trials in which λ^{n+1} is by definition zero, thus the formula in the original report omits the λ^{n+1} term included in Pearce and Hall's formulation.

Perhaps this disadvantage could be overcome by considering the formal relationship between the two models. This analysis may allow us to ascertain to what extent Hall and Rodríguez's is a novel departure from Pearce and Hall's with its own theoretical corpus or rather an ingenious mechanism that can and indeed requires to be accommodated within the original model – that is, whether the former can be subsumed in the latter. In what follows, we show that indeed Hall and Rodríguez's model can be reformulated exactly as Pearce and Hall's with the provision that an initial excitatory value is set to any arbitrary value (other than 0), that is, that, under such an assumption, Pearce and Hall's model subsumes and therefore extends Hall and Rodríguez's.

3. Formal comparison

Beyond the psychological conceptualisation framework that characterises each approach, both models rely on the simultaneity of two opposing connections whose respective strengths subtract to yield an effective, net, associative value. In Pearce and Hall's model a CS forms two associations, an excitatory association to a particular outcome which increases its value V_i^{n+1} when the outcome is presented with the CS, and an inhibitory association, whose value $\overline{V_i^{n+1}}$ grows when the expected outcome is omitted. In Hall and Rodríguez's two connections are also considered, a pre-existing association between the CS and an unspecific outcome event with a value of $V_{\boldsymbol{e}\,i}^{\,n}$ that remains constant throughout exposure and a no-event association $V_{\boldsymbol{ne}i}^{\,n}$ that grows as the CS is experienced in the absence of a generic outcome event. Table 1 shows the algorithms side by side for comparison purposes.

In both models, the changes in the associability of a stimulus is a function of the discrepancy between the total outcome expectation and the current outcome effective value. For $\gamma = 1$, Pearce and Hall's associability algorithm structure is identical to that of Hall and Rodríguez's – as the discrepancy between the current outcome value and the total net outcome (or event) expectation. Table 1 displays the corresponding equations.

The learning algorithms in these models are also equivalent in form, being proportional to the associability and the current effective outcome. Whereas in the Pearce and Hall model the effective excitatory outcome is λ^{n+1} and the effective inhibitory reinforcer is $\left|\overline{\lambda^{n+1}}\right|$, in Hall and Rodríguez's only the no-event effective reinforcer value $\lambda_{\boldsymbol{ne}}^{\,n+1}$ is defined. The models however, conceptualise different proportional constants: outcome specific in the former, which also differentiate between excitatory and inhibitory learning (β_E or β_I, respectively), and CS specific (s_i) in the latter, otherwise irrelevant given the absence of outcomes in Hall and Rodríguez's model. Regardless of the conceptualisation adopted, the fact remains that in terms of the computational output this difference is indistinguishable, and, thus, equivalent to an undefined k constant. Hence, we can rewrite the algorithms as in Table 1.

Table 1

Comparison between Pearce and Hall and Hall and Rodríguez algorithms

	Pearce and Hall		*Hall and Rodríguez*	
	Excitatory	Inhibitory	Event	No-Event
Associative Strength	V_i^{n+1}	$\overline{V_i^{n+1}}$	$V_{e\,i}^{n}$	$V_{ne_i}^{n}$
Associability	$\alpha_i^{n+1} = \left\lvert \lambda^{n+1} - \left(\sum_{i=A}^{Z} V_i^n - \sum_{i=A}^{Z} \overline{V_i^n} \right) \right\rvert$		$\alpha_i^{n+1} = \left\lvert \lambda_{e}^{n+1} - \left(\sum_{i=A}^{Z} V_{e\,i}^{n} - \sum_{i=A}^{Z} V_{ne_i}^{n} \right) \right\rvert$	
Learning	$\Delta V_i^{n+1} = k \cdot \alpha_i^n \cdot \lambda^{n+1}$	$\Delta \overline{V_i^{n+1}} = k \cdot \alpha_i^n \cdot \lvert \overline{\lambda^{n+1}} \rvert$	---	$\Delta V_{ne_i}^{n+1} = k \cdot \alpha_i^n \cdot \lambda_{ne}^{n+1}$

Despite the apparent formal equivalence of the models, the role played by the inhibitory connection controlling excitation in the Pearce and Hall model cannot operate in Hall and Rodríguez's conceptualisation. In Pearce and Hall, the growth of each type of association depends on the gross outcome expectation $\left(\sum_{i=A}^{Z} V_i^n - \sum_{i=A}^{Z} \overline{V_i^n} \right)$ estimated by the difference between the cumulative excitatory values for all present stimuli and the sum of the inhibitory associations retrieved by all present CSs. If the outcome expectation exceeds the actual US value λ^{n+1} then the inhibitory association is strengthened regardless of the presence of the US, providing the model with an effective way to counteract excitation and thus stopping learning. In Hall and Rodríguez's model the growth of the no-event link $V_{ne_i}^{n}$, which is the formal equivalent of Pearce and Hall's inhibitory value $\overline{V_i^{n+1}}$, is driven by a similar process in which the effective no-event reinforcer is given by the total event expectation, i.e., $\lambda_{ne}^{n+1} = \sum_{i=A}^{Z} V_{e\,i}^{n} - \sum_{i=A}^{Z} V_{ne_i}^{n}$. However, if an outcome were to be introduced, the conceptualisation of this mechanism, which implies the absence of any given outcome, would render it insufficient to oppose excitation, and excitatory learning would increase indefinitely. That is, lacking of a proper inhibitory mechanism to counteract excitation, learning would not cease at any given asymptote, potentially increasing *ad infinitum.*

Setting Pearce and Hall's model to have an initial excitatory value larger than $V_i^{n=0} > 0$ would allow it to replicate not only Hall and Rodríguez's predictions but also would extend them to conditioning phenomena. The rationale for an initial value, analogous to Hall and Rodríguez's premise, assumes that $V_i^0 > 0$ reflects a trace of the history of learning and generalisation experienced by an animal, that is, it would represent the fact that any stimulus experienced by an animal is "likely to evoke the expectation of some consequence" (Hall & Rodríguez, 2010, pp. 121).

Although this primary expectation might be theoretically unspecific, that is, non-directed to a given outcome but rather to any outcome, for an animal highly motivated to obtain a specific variety of reinforcer – as it is the case in most learning procedures – it is conceivable that it adopts a more oriented expectation. Insomuch as the animal is eager for cues that anticipate the yearned outcome, once the rewarding outcome is obtained, the primary expectation would simply be added into an initially null, and more specific, outcome prediction. In fact, Hall and Rodríguez, (2010, pp. 129) acknowledged the need for such an assumption to account for latent inhibition.

3.1. Simulation comparison

The following section contains a series of simulations run with the Pearce & Hall Simulator (Grikietis, Mondragón, & Alonso, 2016), comparing Hall & Rodríguez's elaboration which is included as one of the available 'Model Elaborations' in the software package, with a further modification – also in the 'Model Elaborations' menu – which allows setting an initial excitatory value other than zero into the standard Pearce and Hall model.

The first simulation is shown in Figure 1. The left displays Hall & Rodríguez's predictions for associability (top panel) and associative strength (bottom panel) during 6 non-reinforced trials.

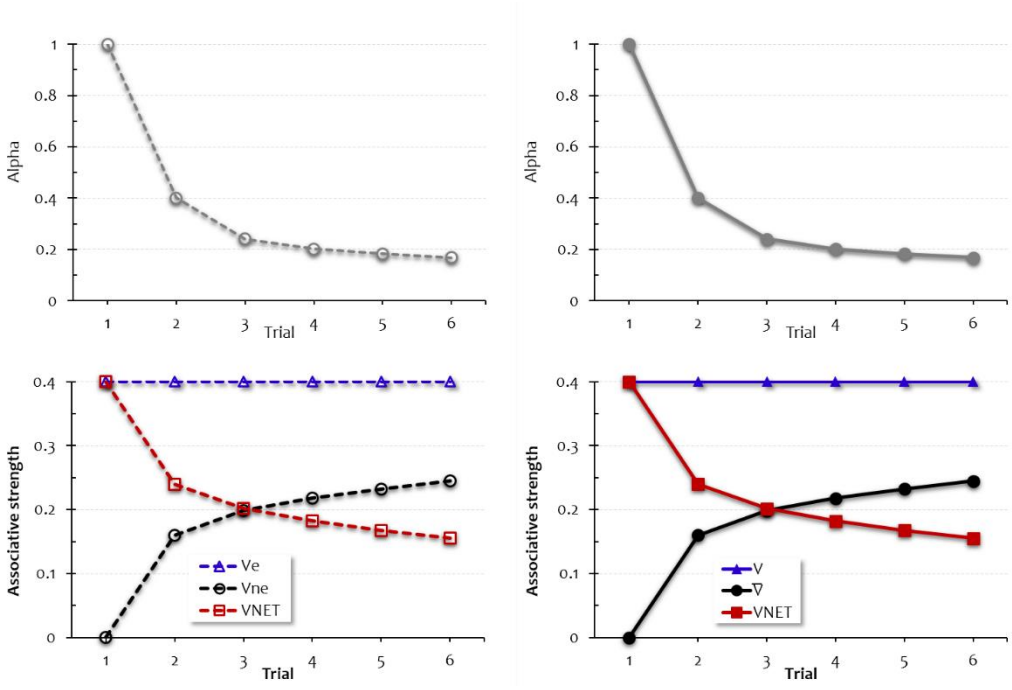

Figure 1. Example of simulations comparing results from Hall and Rodríguez model (left panels), and Pearce and Hall's with an initial excitatory V value (right panels). The top panels show the associability of the CS and the bottom panels the course of associative strength across trials. Parameters in Hall and Rodríguez simulation: $s = 0.4$, $V_e^0 = 0.4$, and $\alpha^0 = 1$. Parameters in Pearce and Hall simulation: $\gamma = 1$, $\beta_I = \beta_E = 0.4$, $V^0 = 0.4$, and $\alpha^0 = 1$. The pattern of results is relatively parameter independent.

Parameters and design were identical to those in (Hall & Rodríguez, 2010, pp. 123; Figure 6.4) from which the example is taken; the right panel displays the corresponding data run setting an initial excitatory V in the Pearce & Hall Simulator[3]. At a glance it is clear that both elaborations make identical predictions.

Hall & Rodríguez (2010) prompted a series of predictions in latent inhibition related phenomena which, it was thought Pearce and Hall's model was unable to foresee. For example, Leung, Killcross and Westbrook (2011) presented a simulation of Hall and Rodríguez's model on the effects of non-reinforced pairings of a stimulus in compound with a previously exposed target on the latent inhibition accrued by the target. The results from this simulation (Figure 2, left panels) advanced a novel prediction of the model: compound preexposure will deepen latent inhibition to the previously exposed target in comparison to the level that the target alone would gain if further exposed alone.

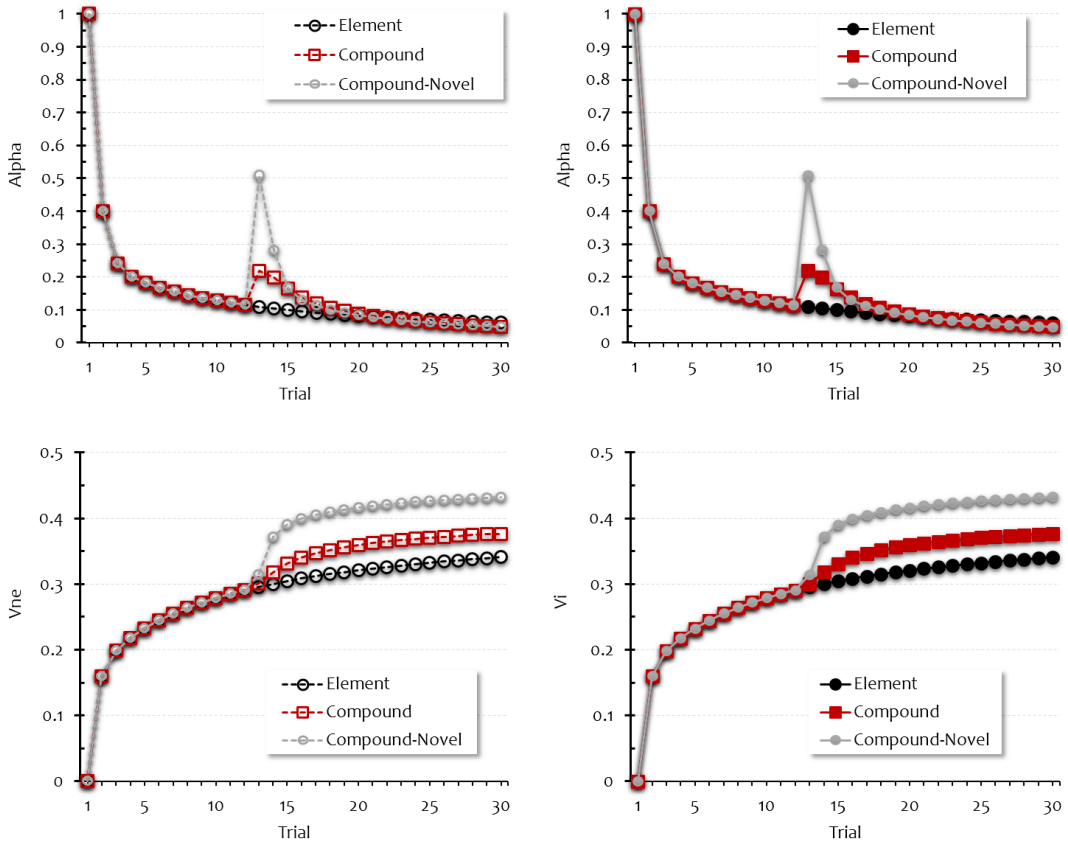

Figure 2. Simulations comparing results from Hall and Rodríguez model (left panels), and Pearce and Hall's with an initial excitatory V value (right panels). The top panels show the associability, and the bottom panels the course of the non-event associative strength (Vne) in the Hall and Rodríguez simulation and the course of the inhibitory associative strength (Vi) in the Pearce and Hall simulation. Parameters in Hall and Rodríguez simulation: $s = 0.4$, $V_e^0 = 0.4$; and $\alpha^0 = 1$. Parameters in Pearce and Hall simulation : $\gamma = 1$ $\beta_I = \beta_E = 0.4$, $V^0 = 0.4$, and $\alpha^0 = 1$.

[3] See (Mondragón, Alonso, & Grikietis, 2016) for how to use the different model elaborations in the simulator.

This effect would be more marked if the paired stimulus were novel. Figure 2 also displays mirror predictions obtained simulating the Pearce and Hall model with an initial excitatory value. Top panels show the progress of the stimulus associability, and bottom panels the growth of the no-event associative value in Hall and Rodriguez's simulation, and the equivalent increase in the inhibitory associative value in Pearce and Hall's model across trials.

It is apparent from inspection of Figure 2 that both models predict identical results for non-reinforced stimulus exposure, both for the stimulus associability and when considering equivalent nodes associative strength.

It seems thus that Hall and Rodriguez's model can indeed be reformulated as Pearce and Hall's provided that an initial excitatory associative strength is set to a value other than 0. But, unlike Hall and Rodríguez's elaboration, the Pearce and Hall model can also simulate results when a reinforcer is introduced, offering more accurate predictions which parallel the real experimental conditions under which animals are usually trained. In other words, Hall and Rodríguez model is formally equivalent to Pearce and Hall's during pre-exposure conditions, and otherwise subsumed by Pearce and Hall's, a model with a broader scope and simulation range.

One would expect that if the models are, after all, equivalent –as we have shown formally and in simulations, allowing an initial value in Pearce and Hall's model—, the results of ensuing experiments would be replicated by this elaboration of Pearce and Hall's model. The following is a simulation of Experiment 3 in Leung et al. (2011), one of the procedures in which the prediction described above was tested. Specifically, the experiment (see Table 3) assessed whether compound preexposure would enhance latent inhibition to a target stimulus which had already undergone non-reinforced exposure, in comparison to the latent inhibition accrued by a comparable stimulus which was equally exposed in isolation. Two groups of subjects received non-reinforced random exposure to three stimuli A, B and C in which each stimulus was presented for a total of 11 trials; next, all animals received 10 non-reinforced trials of a compound AB randomly intermixed with 10 further non-reinforced C trials. Following this exposure regime, animals in Group Compound A received a single conditioning trial in which A was paired with a US (shock), and animals in Group Element C received identical conditioning training to C. During the final test phase, all animals received 8 non-reinforced trials to stimulus A in Group Compound A, and to C in Group Element C. If, as predicted by Hall and Rodríguez's model, compound exposure would deepen latent inhibition, conditioning to A in Group Compound A would be poorer than conditioning to C in Group Element C when tested in extinction test trials.

Table 2
Experiment 3 design (Leung et al., 2011)

	Pre-exposure 1	Pre-exposure 2	Conditioning	Extinction Test
Compound-A	A- ; B- ; C-	AB- ; C-	A+	A-
Element-C	A- ; B- ; C-	AB- ; C-	C+	C-

Data in Figure 3 shows predictions for the final test obtained when simulating Pearce and Hall's model with an initial V excitatory setting (right hand side panel) in comparison to the predictions from the original model with zero initial excitatory value (middle panel). The left hand panel displays an extract of the empirical results (adapted from Leung et al., 2011). Although no statistical test was conducted, visual inspection of the data reveals that the original version of the model renders it unable to predict the observed difference between the experimental conditions. However, by allowing the model to set an initial excitatory value under the assumptions above described, the prediction pattern in the net associative strength of the stimuli is analogous to that found in the experiment during test, that is, higher conditioning to stimulus C (Group element C) than to stimulus A in Group Compound A.

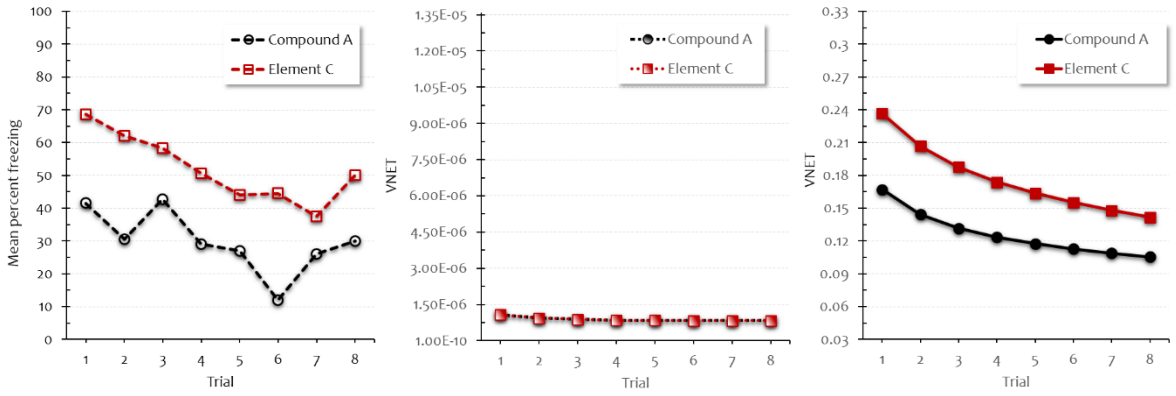

Figure 3. Empirical results during an extinction test adapted from Leung et al., 2011, Experiment 3 (left panel), and equivalent simulated results (right panel) as predicted by the original Pearce and Hall model (middle panel) and by Pearce and Hall model with the provision of an initial excitatory value (right panel). Parameters : $\gamma = 0.5$; $\alpha^0 = \beta_E = 0.75$; $\beta_I = 0.25$. Initial excitatory value $V^0 = 0.75$.

4. Conclusions

Pearce and Hall's is one of most influential models of classical conditioning in that, among other things, can account for pre-exposure effects such as latent inhibition on the basis of the decline in the stimulus associability. However, as formulated in (Pearce and Hall, 1980; Pearce et al., 1982), latent inhibition arises as a by-product rather than as a learning process in itself by which the stimulus becomes an accurate predictor of precise consequences, a true parallel to conditioning phenomena. Hall and Rodríguez's elaboration of the model proposed a dual mechanism to account for latent inhibition. According to their suggestion, latent inhibition will be the result of, on the one hand, the loss in associability of a stimulus undergoing pre-exposure, and on the other, and critically so, to the establishment of a stimulus—no-event association. Such an association might be expected to compete with other similar links formed during an exposure regime and to interfere with the formation or retrieval of stimulus—outcome associations. Hall and Rodríguez's model has thus triggered a wave of experimental activity in the area.

In this technical report we have proved that, provided Pearce and Hall's model takes an initial V value higher than 0, both models are formally equivalent when applied to non-reinforced trials. Indeed, simulations of Pearce and Hall's model under such provision render exactly the same results than Hall and Rodríguez's and accurately replicate its predictions, that lay outside the scope of the original Pearce and Hall's model. Moreover, unlike Hall and Rodríguez's model, the elaboration of Pearce and Hall model introduced here can cope with both reinforced and non-reinforced conditions – enabling it to simulate empirical results at their actual learning stage.

Hall and Rodríguez's model should be acknowledged nevertheless as a modification that has spurred the refinement of Pearce and Hall's model, and that has in turn deepened our understanding of latent inhibition and related phenomena.

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