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THEORETICAL NOTE

A Formal Analysis of the Standard Operating Processes (SOP) and Multiple Time Scales (MTS) Theories of Habituation

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In this article, we compare two theories of habituation: the standard operating processes (SOP) and the multiple time scales (MTS) models. Both theories propose that habituation is due to a reduction in the difference between actual and remembered stimulation. Although the two approaches explain short-term habituation using a similar nonassociative mechanism based on a time-decaying memory of recent stimulus presentations, their understanding of retention of habituation or long-term habituation differs. SOP suggests that retention of habituation happens through associative retrieval from a long-term memory store, while MTS relies on the differential decay rate of a series of memory units. This essential difference implies that spontaneous recovery, which refers to the return of the response to levels above those reached during habituation, is predominantly a consequence of a mixture of decay and loss of association for SOP and exclusively of decay for MTS. We analyze these mechanisms conceptually and mathematically and demonstrate their functioning with computer simulations of conceptual and published experiments. We evaluate both theories regarding parsimony and explanatory power and propose potential experiments to evaluate their predictions. We provide MATLAB-Simulink and Python codes for the simulations.

Keywords: sometimes opponent processes, priming theory, multiple time scales, habituation, trial spacing

Habituation is a form of learning consisting of a decrease in the magnitude of the response to a stimulus due to its repetition. One of the earliest and most prevalent theoretical ideas to explain this phenomenon is to assume that stimulus repetition leads to forming a "memory" of it that inhibits subsequent reaction to that same stimulus. This notion is embodied at the heart of several early theories under a variety of names such as "reactive inhibition" (Hull, 1943), "satiation" (Glanzer, 1953), "conditioned inhibition" (Stein, 1966), "inhibitory system" (Konorski, 1967), "cholinergic inhibition" (Carlton, 1968), "impulses of extrapolation" (Sokolov, 1969), "priming" (Wagner, 1976), or "inhibitory feedback" (Pfautz et al., 1978). Within this framework, it suffices to posit that such a memory of the stimulus decays spontaneously with time to account for the widespread observation that the shorter the interval between the presentations of the stimulus (hereafter "interstimulus interval [ISI]"), the faster and more pronounced the habituation (e.g., Davis, 1970; Hargitt,1906; Rankin & Broster, 1992; Yerkes, 1906).

However, a unique memory process seems insufficient to embrace a phenomenon known as "rate-sensitive habituation," where the ISI has opposite effects on the development of habituation and its retention (J. Staddon, 1993). For instance, Davis (1970) observed that habituation of the startle response of rats proceeded faster and to a greater extent in a group of animals that received 1,000 stimuli every 2 s than a group that received the same stimulation every 16 s, but remarkably, the response was more diminished in the 16 s group than in the 2 s group when the stimulus was tested 1 min or 24 hr later with ISIs of 2, 8, and 16 s in both groups. In principle, rate-sensitive habituation seems to uncover a more complex type of learning that occurs during stimulus repetition. The most common choice is the assumption that two kinds of memory are involved in habituation: a transitory memory, called short-term habituation (STH), favored by presentations of the stimulus at short intervals, and

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a more durable process, known as long-term habituation (LTH), facilitated by more spaced repetitions. STH would be the principal cause of the progressive decrement in responding within a session, and LTH would be responsible for the retention of habituation across sessions. The rationale of this explanation is that the formation of long-term memories is favored by spaced training because it provides better opportunities for rehearsing the stimulus trace without interference from the memorial effect of the previous trial, which would be responsible for short-term habituation (e.g., Hintzman, 1974; Wagner, 1976). Extensive investigation has been carried out simultaneously at both neurobiological and behavioral levels with several animal models, especially with the mollusk *Aplysia* (see Carew, 1984), the nematode *C. elegans* (see McDiarmid et al., 2019), and the rat (see Davis & File, 1984), which supports the distinction between STH and LTH (McDiarmid et al., 2019).

Although the short- and long-term notion provides a way to describe rate-sensitive habituation at a qualitative level, it only predicts the phenomenon with specific assumptions about how these two mnemonic processes are differentially dependent on the ISI. A conspicuous representant of this approach is Wagner's (1976, 1978, 1979) priming theory and its quantitative elaboration standard operating processes (SOP; Mazur & Wagner, 1982; Wagner, 1981; Whitlow & Wagner, 1984), which proposes that a stimulus's capacity to be represented in active memory and elicit a response is inversely proportional to its level of expectation or priming before presentation. Wagner supposed that one source for this priming is simply the memory of a recent presentation of the same stimulus (self-generated priming). This memory is transient and disappears when sufficient time has elapsed from the last presentation of the stimulus (e.g., with long ISIs or from one session to another). Retention of habituation or long-term habituation, on the other hand, would require a different mechanism that Wagner called "retrieval-generated priming." In this case, the supposition was that the repetition of a stimulus in a context would cause the context to become a conditioned stimulus to develop an association with the habituating stimulus, which plays the role of the unconditioned stimulus. As the association grows over trials, the stimulus becomes gradually more expected in the context or primed by it. The strength of this association depends on the degree of simultaneous activation of the stimulus and context in active memory. Since repetition of the stimulus at short ISIs causes low activation of its representation (due to self-priming), little association develops. On the contrary, more spaced intervals cause less self-priming, allowing for the acquisition of more association with the context in each trial. Therefore, SOP posits that more self-generated priming results in less associatively generated priming (i.e., rate-sensitive habituation; see Uribe-Bahamonde et al., 2019; Vogel et al., 2020).

Alternatively, J. Staddon (1993; J. E. R. Staddon & Higa, 1996) proposed a quantitative model of rate-sensitive habituation, called the multiple time scales model (hereafter MTS model), that does not rely on the distinction between short- and long-term mechanisms. MTS proposes that habituation results from the difference between actual stimulation and a time-decaying memory of the stimulus. This memory is formed every time the stimulus is presented but decays exponentially with time. In that respect, Staddon's approach is conceptually identical to Wagner's self-generated priming since they both explain habituation in terms of a time-decaying memory. MTS differs from priming theory in how it produces rate sensitivity. For this, Staddon proposed that stimulus processing occurs through a serial concatenation of memory units where the output of each unit is the input of the following. The output of each unit is the difference between its input and the memory stored in it. The critical assumption is that the memory of each unit decays at different rates, with "earlier" units decaying faster than "late" units. Thus, with short ISIs, habituation is large because the memory in the early units has not decayed yet when the next stimulus is presented, but retention is little because the slow-decaying late units do not receive enough input from the earlier units, so they remain inactive. On the contrary, with longer ISIs, there is little habituation because the memory in the early unit has fully decayed before the next presentation of the stimulus, but it is more retained because the late units received sufficient input from earlier units and stored it for a more extended period.

Notice that both Wagner's and Staddon's theories assume that habituation results from a mnemonic mechanism by which the accumulated effect of previous stimulus presentations provokes the suppression of the direct behavioral effect of the stimulus. In that respect, the two theories assimilate habituation with a sort of refractorylike effect accumulating over trials. These theories differ, however, in the mechanism they propose for retention of habituation: Staddon's approach relies on a sort of slow leaky integrator. In contrast, Wagner's theory relies on context-stimulus associations. This difference leads to several testable predictions. First, according to SOP, LTH should be context specific and susceptible to extinction in a way that MTS, which assumes no association between the context and the stimulus, should not. Secondly, since Staddon's model explains retention exclusively in terms of memory decay, it predicts that the degree of retention would be inversely proportional to the interval between stimulus exposure and test (i.e., the so-called "retention interval"). On the contrary, SOP predicts that as time passes, the performance in the retention test would be more dependent on LTH than on STH, and therefore, retention would be relatively insensitive to the passage of time, providing that animals spend this interval outside the experimental context. However, if the retention interval happens within the experimental context, SOP predicts the extinction of the association between the context and the stimulus proportional to the duration of this interval.

However, it is not easy to glean further potential differences between SOP and MTS. They are presented with different languages and levels of quantitative details. Wagner and his colleagues were mainly interested in the conceptual implications of SOP for the larger field of associative learning, paying less attention to quantitative implications for any specific data set, including habituation. Thus, only a few time-dependent algebraic functions and a minimal parametric analysis are available regarding SOP. In contrast, Staddon and his colleagues presented the MTS model using very defined discrete-time equations and conducted a relatively exhaustive optimization of parameters for a restricted data set on habituation. As a result, it is not surprising that there is very little cross-citation between the followers of each approach.

In this article, we have gathered information from various sources to present the two models. We first present them with their original language and mathematical notation and then with a set of differential equations. We focus on the conceptual differences between the models, providing readers with a straightforward tool for quantitatively assessing each model's predictions. We then contrast these theoretical aspects with evidence on rate-sensitive habituation that led J. E. R. Staddon and Higa (1996) to set the parameters of their model (i.e., Rankin & Broster, 1992). We also

compare them with the evidence of Davis and Wagner (1968) on stimulus-intensity effects, which have been taken as supportive of the SOP model (e.g., Whitlow & Wagner, 1984). As we will illustrate below, experiments demonstrating ISI and intensity effects highlight the challenge of distinguishing learning from performance in habituation. Based on these experimental designs, we outline future research suggested by this theoretical analysis.

The Models

Standard Operating Processes

The SOP model was introduced in two chapters as "a model of automatic memory processing in animal behavior" (Wagner, 1981) and "an episodic model of associative learning" (Mazur & Wagner, 1982), reflecting an ambitious goal of encompassing the regularities of associative learning. Subsequent extensions of the model were equally concerned with facts and controversies of conditioning (e.g., Brandon & Wagner, 1998; Vogel et al., 2019; Wagner & Brandon, 1989). In spite that the conceptual roots of the SOP model lie in priming theory, which was formulated to account for habituation (Wagner, 1976, 1978, 1979), this phenomenon received less attention among SOP's followers, except for a couple of publications (Donegan & Wagner, 1987; Whitlow & Wagner, 1984) and a few recent simulation exercises (Uribe-Bahamonde et al., 2019, 2021).

According to SOP, environmental stimuli are represented by nodes containing a set of elements that can be in one and only one of three states of activity: inactive (I), primary activity (A1), and secondary or refractory activity (*A*2). Figure 1 presents an example of the theoretical entities of the model, called "nodes," representing a stimulus and the context in which it occurs in a standard habituation experiment. The two events represented in Figure 1 (i.e., target stimulus and context) are each represented by a respective set of theoretical elements. With the onset of any of these events (target stimulus or context), a proportion of its elements moves from an inactivity state to a primary activity state with a probability of $p1_i$. Once in the A1 state, some elements move to a secondary state, with a probability of $pd1_i$, and then back to inactivity, with a probability of $pd2_i$. It is assumed that $p1_i$ is a function of the intensity or salience of the event, taking a value greater than zero in the presence of the event and zero in its absence. The parameters $pd1_i$ and $pd2_i$ are independent of whether the event is on or off (Wagner, 1981). Let I_i be the proportion of elements of event i in the inactive state, $\mathrm{A1}_i$ be the proportion of elements of event i in the primary activity state, and $\mathrm{A2}_i$ be the proportion of elements of event i in the secondary state of activity. It follows that at any moment, t, $I_i + \mathrm{A1}_i + \mathrm{A2}_i = 1$ and a time 0 $I = 1$.

The response to the target stimulus is determined by the proportion of its components in the primary state over time $(\mathrm{A1}_s)$[1]. The theory suggests that the physical presentation of the stimulus is the only way to provoke A1 activity in its node and produce a response. After the first presentation of the stimulus, there is a period where a significant proportion of elements are in the A2 state, so they cannot be promoted to the A1 state upon subsequent presentations of the stimulus. As a result, the second presentation of the stimulus is less effective in eliciting a response than the first presentation, with this effect becoming more pronounced the shorter the ISI. Thus, the observation that habituation is more pronounced with shorter ISIs is explained by SOP through self-generated priming.

The SOP model posits that secondary activity corresponding to the target stimulus, $\mathrm{A2}_s$, is not only influenced by its $\mathrm{A1}_s$ activity but also by contextual cues or other associative sources. Specifically, the context is assumed to behave as a conditioned stimulus that develops an association with the target stimulus (V_{c-s}). This association includes excitatory and inhibitory associations that develop simultaneously depending on the strength of the respective states of activity of the context and the stimulus. The acquisition of an excitatory association is the product of concurrent A1 activity of the context and the stimulus, while the acquisition of an inhibitory context-stimulus association is the product of simultaneous A1 activity of the context and A2 activity of the stimulus. The net association is computed by subtracting inhibitory associations from excitatory associations, weighted by parameters L^+ and L^-, respectively. Formally:

$$\frac{dV_{c-s}}{dt} = A1_c(t)(A1_s(t)L^+ - A2_s(t)L^-). \tag{1}$$

The moment-by-moment acquired associations are accumulated in long-term memory. As shown in Figure 1, the context, via its association with the target stimulus, acquires the ability to promote elements directly from the inactive to the secondary state of activity of the stimulus with a probability of $p2_s$, which is given by:

$$p2_s(t) = \begin{cases} 0, & p2_s(t) \le 0; \\ 1, & p2_s(t) \ge 1; \\ \mathrm{A1}_c(t)V_{c-s}(t), & \text{otherwise.} \end{cases} \tag{2}$$

Thus, the general way of describing the activity of the target stimulus regarding contextual influences is as follows[2]:

$$\frac{dI_s}{dt} = pd2_s(t)\mathrm{A2}_s(t) - (p1_s(t) + p2_s(t))I_s(t). \tag{3}$$

$$\frac{d\mathrm{A1}_s}{dt} = p1_s(t)I_s(t) - pd1_s(t)\mathrm{A1}_s(t). \tag{4}$$

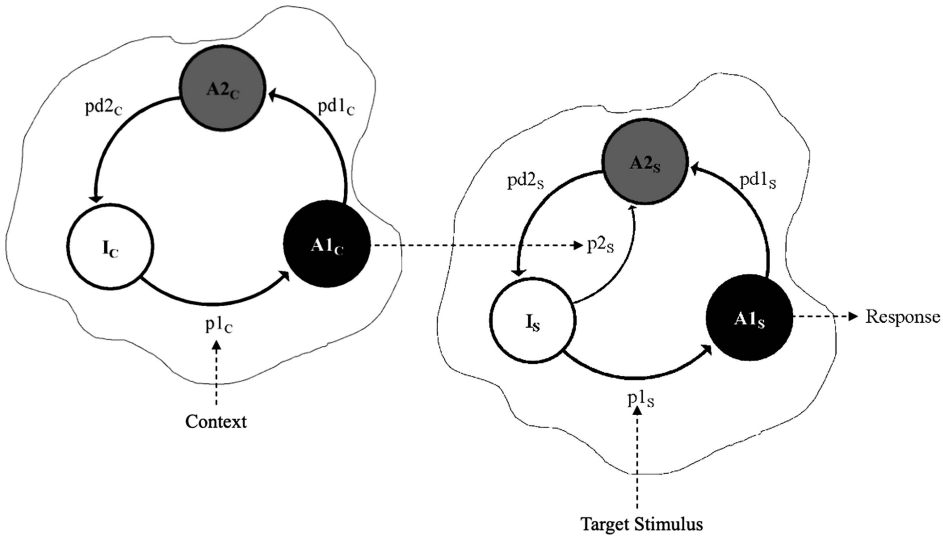
$$\frac{dA2_s}{dt} = pd1_s(t)\mathrm{A1}_s(t) + p2_s(t)I_s(t) - pd2_s(t)\mathrm{A2}_s(t). \tag{5}$$

One crucial parametric assumption of SOP is that the primary activity decays faster than the secondary activity (i.e., $pd1 > pd2$). An important consequence of this assumption is that the acquisition of an association between the context and the stimulus depends on the interval at which the stimulus is presented. If the ISI is short, the $\mathrm{A1}_s$ activity is constantly reduced by self-generated priming, which results in less excitatory association with the context. Moreover, since the $\mathrm{A2}_s$ activity is protracted, it remains at high levels at each stimulus presentation, leading to substantial inhibitory learning.

[1] The original version of the SOP model suggests that the response to a stimulus is influenced by both the primary and secondary states of activity. The second component of the response can be either agonist, antagonist, or unrelated to the first component. Wagner (1981) proposed that the response is a function of the number of elements in both states of activity, weighted by the linear constants $w1$ and $w2$. Although this concept is useful for Pavlovian conditioning, it is quite complex and unnecessary for describing most facts of habituation. Therefore, in this article we assumed that the response of interest is solely dependent on A1 activity.

[2] The theoretical pattern of activity of the context across the three states (i.e., I_c, $\mathrm{A1}_c$, and $\mathrm{A2}_c$) follows the same rules than those for the target stimulus except that $p2_c = 0$ meaning that no stimulus is assumed to signal the context.

Figure 1
A Visual Characterization of the Standard Operating Processes Model



Note. The target stimulus and the context are represented by nodes containing a set of elements that can be in one of three states of activity: inactive (I_s and I_c, respectively), primary ($A1_s$ and $A1_c$, respectively), and secondary ($A2_s$ and $A2_c$, respectively). The context, via its associative link, influences the activity of the target stimulus by moving its elements directly from I to A2 with probability $p2_s$.

Overall, these decreased excitation and increased inhibition effects at short intervals result in a poor net context-stimulus association. On the other hand, longer intervals lead to more excitatory and less inhibitory learning due to the $A1_s$ and $A2_s$ activities having sufficient time to recover and decay, respectively, from trial to trial. Thus, the observation of less retention of habituation with shorter intervals is explained by SOP through associatively generated priming.

Multiple Time Scales

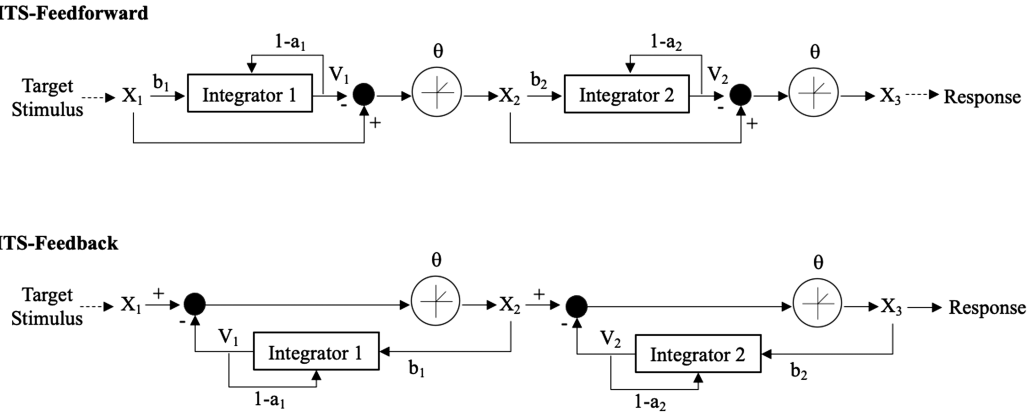
Unlike the SOP model, which was initially proposed as a general theory of conditioning, the MTS model was developed as a specific theory of habituation (Innis & Staddon, 1989; J. Staddon, 1993; J. E. R. Staddon & Higa, 1996) that subsequently was applied to interval timing (e.g., J. E. R. Staddon, 2005; J. E. R. Staddon et al., 2002). The essential aspect of the theory is the use of a discrete-mathematics version of a leaky integrator to describe the relationship between the stimulus (input), the memory (integrator), and the response (output). The model suggests that the response to a target stimulus depends on the difference between the actual stimulation and the memory of that stimulus. This memory results from a series of concatenated memory subunits, where the inputs are converted to outputs according to a leaky integrator algorithm. Two versions of the theory differ in whether each unit's input (feedforward version) or output (feedback version) is used to compute each memory trace. While Staddon and his colleagues have focused mainly on the feedforward version, both versions are presented here for completeness.

The top panel of Figure 2 depicts a feedforward example of two integrators commonly used by Staddon and colleagues to describe rate-sensitive habituation (J. Staddon, 1993; J. E. R. Staddon & Higa, 1996; J. E. R. Staddon et al., 2002). Let's consider the behavior of the first integrator, whose input, X_1, comes directly from the stimulus and its output, $X_2(t)$, results from the subtraction of the input minus the memory in the integrator, that is, $X_2(t) = X_1(t) - V_1(t)$. The parameter θ is used to set a lower limit for the output ($\theta = 0$ in all Staddon's outlets). The memory of the integrator is updated according to a weighted sum of its current input and its memory, that is, $V_1(t + 1) = a_1V_1(t) + b_1X_1(t)$. Since a and b take values between 0 and 1, memory decays over time. Thus, if the stimulus is repeated at a short ISI, the memory of prior stimulations will be substantial, and the response will be diminished. On the contrary, if the ISI is longer, the memory in the integrator will have the opportunity to decay between stimulus repetitions, leading to a greater response. The fact that habituation is more pronounced with shorter intervals is, thus, gracefully accounted for by a single-integrator version of the MTS model. As shown in the bottom plot, the feedback version of the model operates identically to the feedforward version, with the only difference being that the output, instead of the input of each integrator, is used to compute the memory trace of each integrator. The simulation section shows that both versions predict similar results in many circumstances.

The MTS model posits that to account for the fact that retention of habituation is favored by longer intervals, at least a second integrator must be involved. As shown in Figure 2, the input of the second integrator is the output of the first, so it is not activated unless the first integrator produces an output. The memory trace of the second integrator is determined by a different value of the parameter a_i, which determines how quickly memories decay over time, and b_i, which determines the weight of the stimulus in the formation of the memory. These parameters determine how memories are formed and retained over time. Importantly, rate-sensitive habituation rests in the assumption that the decay rate of the second integrator is slower than that of the first (i.e., $a_1 < a_2$). With very short intervals, the second integrator does not receive much input from the first,

Figure 2

A Visual Characterization of Two-Unit Feedforward (A) and Feedback (B) Versions of the Multiple Time Scales Model



Note. X_1, X_2, and X_3 represent the value of the signal at different stages of the theoretical processing of the stimulus, and θ is the lower limit of these values. The initial value of the signal (X_1) depends entirely on the stimulus's intensity, and the signal's last value, X_3, represents the magnitude of the response. The rectangles represent the integrators of the model in which memory, *Vi*, is updated according to the sum of the respective input and previous memory of the integrator, weighted by the parameters a_i and b_i, respectively.

rendering this assumption irrelevant. However, with longer intervals, the second unit receives some input; thereby, the increased a_2 value lengthens the memory trace. Essentially, the response to the target stimulus hinges entirely on the dynamics of the second integrator, making rate-sensitive habituation dependent on its behavior.

J. E. R. Staddon and Higa (1996) demonstrated that using two integrators is sufficient for describing part of Rankin and Broster's (1992) and Broster and Rankin's (1994) data on rate-sensitive habituation of the nematode with intervals of 10 and 60 s. They indicate, however, that more integrators may be needed for finer temporal sensitivity and longer retention intervals. J. E. R. Staddon (2005) proposed that the output of unit "i" in the general case of *N* integrators is given by:

$$X_i(t) = \begin{cases} \theta, & X_i(t) \leq \theta; \\ X_{i-1}(t) - V_{i-1}(t), & \text{otherwise.} \end{cases} \quad (6)$$

With respect to the activation of the integrators, we transform the equations provided by J. E. R. Staddon and Higa (1996) from discrete to continuous mathematics. In the case of the feedforward version of MTS, we proceed as follows:

$$\frac{dV_i}{dt} = V_i(t+1) - V_i(t) = b_i X_i(t) - (1 - a_i)V_i(t). \quad (7)$$

Following the same procedure, the differential equation for activation of integrator "i" in the feedback version of MTS is given by:

$$\frac{dV_i}{dt} = b_i X_{i+1}(t) - (1 - a_i)V_i(t). \quad (8)$$

As mentioned above, the prediction of rate-sensitive habituation in MTS depends on increasing values of the parameter a_i for the successive integrators. The case of parameter *bi* is less relevant for the predictions of the model, but it is also assumed that it increases exponentially. Specifically, J. E. R. Staddon and Higa (1996) proposed the following equations, where i is the position of the integrator in the series (from 1 to n), and λ_a and λ_b are fixed parameters.

$$a_i = 1 - e^{-\lambda_a \cdot i}. \quad (9)$$

$$b_i = e^{-\lambda_b \cdot i}. \quad (10)$$

Simulations

As shown above, although the SOP and MTS models were formalized using different mathematical strategies, they have some conceptual commonalities. Table 1 shows a few concepts that exemplify these overlaps. For instance, $p1$ in SOP and X_1 in MTS are the input variables activated by the target stimulus and can be taken as representing stimulus intensity. In SOP, $p1$ is a "transition probability," so it varies between 0 and 1. Although Staddon and his colleagues have typically used a value of 1 for the inputs of their simulations, they acknowledge that different values can represent stimulus intensity. We will use 1 for maximal intensity and 0 for the stimulus's absence in both models for consistency.

Table 1

A Comparison of Main Constructs of the Standard Operating Processes and Multiple Time Scale Models

Theoretical concept	SOP	MTS
Input (stimulus intensity)	$p1$	X_1
Output (response magnitude)	A1	X_{n+1}
Memory	A2	V_1, V_2
Rate of memory activation	$p2$, $pd1$	b_1, b_2
Rate of memory decay	$pd2$	a_1, a_2

Note. SOP = standard operating processes; MTS = multiple time scale.

The second concept shown in Table 1 is the response to the target stimulus, which is the primary dependent variable in most studies on habituation. As mentioned in the previous section, the SOP model assumes that the response to the target stimulus depends on $A1_s$, whose value over time is governed by Equation 4. The obtained pattern of activity is quite complex but regular, involving an initial period of recruitment up to a peak proportional to $p1$, followed, if the stimulus remains on, by a period of adaptation proportional to $pd1$, prior to decay toward zero following stimulus termination. Wagner (1981) conceived this pattern of A1 activity to match psychophysical data on stimulus perception (e.g., Marks, 1974) and suggested that specific response functions can be modeled from the value of A1 to produce various parameters such as amplitude, duration, or probability (Donegan & Wagner, 1987). In the case of MTS, Staddon and colleagues assumed that the response was the difference between the input and the memory (Equation 6), so the form of the response is of an exponential decay from the maximal value represented by the input up to a minimal value of theta. Since the input varies between 1 and 0 in the two models, the maximum response also varies between 1 and 0 in the two models.

Memory, designed as $A2_s$ and V_n in the SOP and MTS models, respectively, is the essential theoretical entity that inhibits or subtracts stimulus's input to produce habituation. Both models suggest that memory is activated by recent stimulus presentation (at a rate of $pd1$ for SOP and b_i for MTS) and decays over time at an exponential rate ($pd2$ for SOP and a_i for MTS). However, the two models differ in their assumptions regarding a more complex way of building this memory. SOP assumes that apart from the stimulus, memory can be activated by associative sources like the experimental context (at a rate of $p2$). For MTS, memory involves cascaded units whose activities decay at different rates. Despite the conceptual differences in these two ways of conceiving memory, they have several computational similarities that lead to similar predictions for many experiments.

Table 1 offers only an approximated heuristic for comparing the two models. A more rigorous contrast, of course, must be done with quantitative data. For this, we implemented Equations 1–5 (SOP model) and 6–10 (MTS model) using MATLAB/Simulink (R2023b). The block diagrams for the Simulink version of the SOP and MTS models are shown in Figures 3 and 4, respectively. These diagrams enable interested users to implement their simulations or to reproduce those reported in this article by downloading the MATLAB-Simulink simulators at https://github.com/vogelab/MTSvsSOP. As an open-source alternative, we also provide Python codes for the simulations.

An advantage of presenting the theories in the block diagrams of Simulink is that it allows for intuitively simultaneous exams of concepts, equations, and outcomes of the models. Indeed, by comparing Figure 1 with Figure 3 and Figure 2 with Figure 4, it is evident that the Simulink block diagrams capture each model's central conceptual ideas as their authors formulated them. For instance, the diagrams make apparent the circular nature of the SOP model, contrasting with the serial nature of the MTS model. Likewise, it is evident that the complexity of SOP comes predominantly from the need to represent the stimulus and its context to describe habituation. In contrast, the complexity of MTS comes from the need to represent more than one theoretical unit for the stimulus. We will show that representing these theories through time-varying differential equations and implementing their numerical solutions through a unified platform can be a powerful tool for comparing them conceptually and formally.

To demonstrate how these implementations of the models work, we performed a few simulations of real and hypothetical habituation experiments using MATLAB-Simulink and Python. Since the models were represented using differential equations, they can make predictions in continuous time. However, we utilized Euler's integration method to obtain numerical solutions, which only provides an approximation of continuous time. Additionally, we decided to use a one-moment integration step to maintain consistency with the time unit used in previous SOP and MTS simulations. This means that strictly speaking, the models' equations were discretized into one-moment steps. Exploring other integration methods or different sizes of fixed or variable steps to solve the equations presented in this article can be a matter of future theoretical analysis.

Given the scope of this article, instead of conducting any sort of parameter fitting or optimization, we adopted a straightforward approach of using the parameters most frequently utilized or

Figure 3

Block Diagram for the Simulink Implementation of the Standard Operating Processes Model

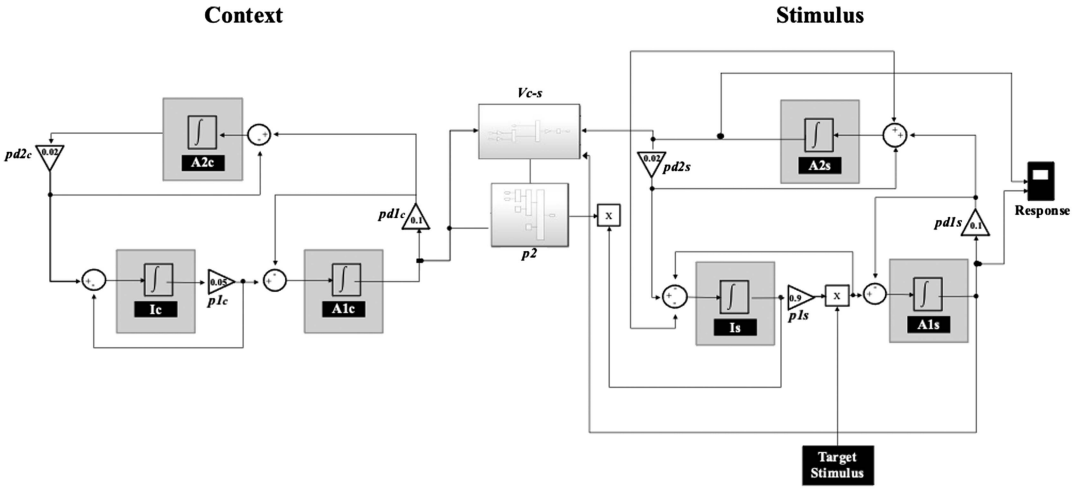
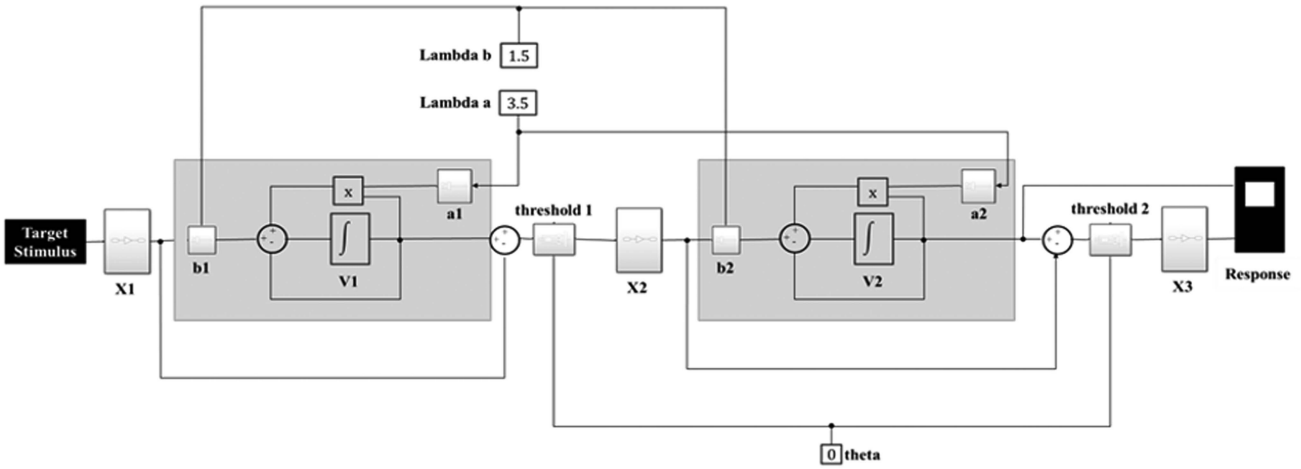


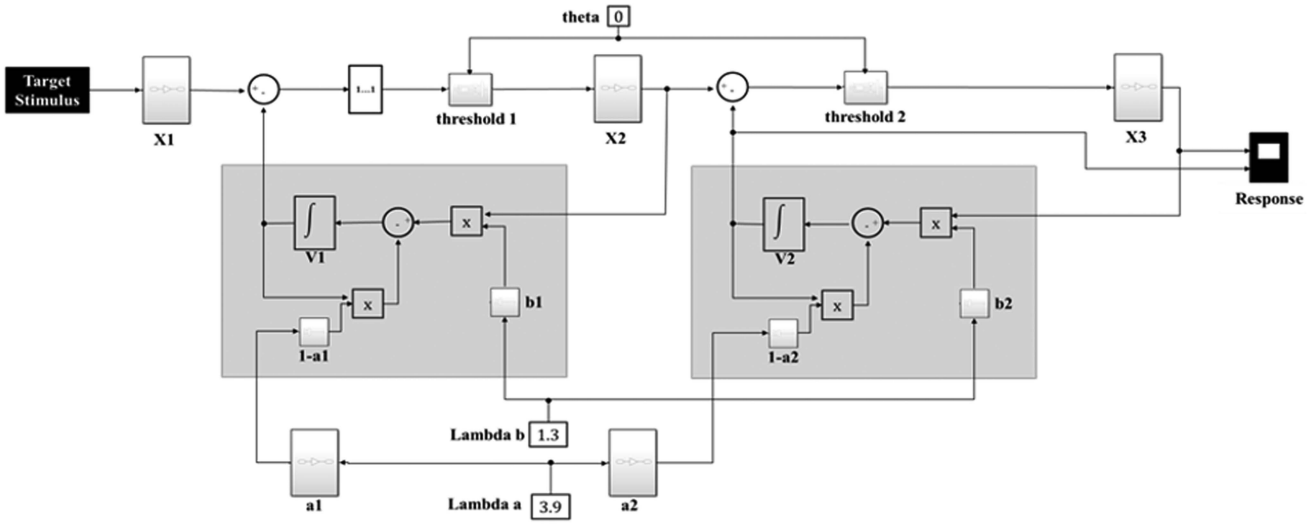
Figure 4

Block Diagram for the Simulink Implementations of the Feedforward (A) and Feedback (B) Versions of the Multiple Time Scale Model

(A) MTS- Feedforward



(B) MTS- Feedback



Note. MTS = Multiple Time Scale.

recommended by the original authors. We preferred this fixed-parameter strategy above optimization because we aim to compare the models conceptually rather than assess their relative fits to data.

In the case of SOP, we mostly follow Vogel et al. (2019) and Uribe-Bahamonde et al. (2019) by setting $p1_s = 0.2$–0.9 to represent stimuli of different intensities; $p1_c = 0.05$; $pd1_s = pd1_c = 0.1$; $pd2_s = pd2_c = 0.2$; $L^+ = 0.015$ and $L^- = 0.0015$. Three aspects of this choice might be important to emphasize. First, we set the intensity of the target stimulus ($p1_s$) at a higher value than that of the context ($p1_c$) to emulate standard habituation experiments in which the context is supposed to be a behaviorally neutral stimulus. Second, the fact that $pd2$ is assumed to be smaller than $pd1$ for both the stimulus and the context ensures that A2 activity lasts longer than A1 activity, which is essential for all priming effects. Finally, L^+ was assumed to be 10 times greater than L^- because this rate allows for the acquisition of an excitatory association between the context and the stimulus. Although, to our knowledge, no parameter optimization of SOP has been conducted to fit empirical data, its qualitative robustness is noticeable with these parameters in explaining many other phenomena of learning beyond habituation (see Vogel et al., 2019).

In the case of the MTS model, we followed J. E. R. Staddon and Higa (1996), who recommended the use of two units, with $\lambda_a = 3.5$ and $\lambda_b = 1.5$ for the feedforward version of the model and $\lambda_a = 3.9$ and $\lambda_b = 1.3$ for the feedback version. These values resulted from a hill-climbing optimization process conducted by the authors to fit the model to Rankin and Broster's (1992) data (see Table 2 in J. E. R. Staddon & Higa, 1996). To keep consistency with SOP, the

intensity, X_1, was varied between 0.2 and 0.9 for stimuli of different intensities.

We conducted simulations on published outcomes of experiments regarding the habituation of the startle response of rats to auditory stimuli, the contraction response of the nematode *C. elegans* to tactile stimulation, and the galvanic skin response in humans to auditory stimuli. The amount of research on these procedures is extensive, and some of its outcomes were explicitly mentioned in the initial formulations of the SOP (Whitlow & Wagner, 1984; predominantly startle in rats) and MTS (J. E. R. Staddon & Higa, 1996; predominantly contraction in nematodes) models. We assumed that the simulations with each model represent the expected or mean values predicted by the models, not individual instances. Therefore, they could be validly compared with the central trends reported in the actual empirical data. Since it has been shown that individual patterns vary significantly around the mean in habituation experiments (e.g., Lane et al., 2013; LaRowe et al., 2006; Peeke & Petrinovich, 1984; Plichta et al., 2014; Poli et al., 2024), examining experiments and models from the perspective of individual differences is a potentially fruitful area of study, which could be pursued in research where relevant individual data is available. However, this would imply adding individual variability into each model, which is beyond the goals of the present article.

The typical stimulus duration for rat experiments ranges from 50 to 100 ms, while the ISI ranges from 2 to 60 s. The typical stimulus duration for nematodes is between 0.6 and 1 s, and the ISI ranges between 2 and 60 s. In the case of humans, there is more variability with ISIs ranging from 1 to 180 s and stimulus durations from 1 to 10 s. Since there is no clear way of determining the behavioral significance of the absolute measured time for these three animals, we assumed that one simulated moment equals 1 s for simulating the ISI of the experiments. Although the stimulus duration plays a crucial role in predicting the models' outcomes, it has not been sufficiently studied in the habituation of the startle response in rats, the skin conductance in humans, or the contraction of *C. elegans*. Therefore, we made the simplest assumption of using a one-moment duration stimulus in all the simulations.

We present the results regarding each model's predicted response. To compare the simulation's outputs with actual data, we redraw published data using the Plot Digitizer software (https://plotdigitizer.com/app).

Simulations of ISI Effects

In the introduction, we mentioned that the MTS and SOP models describe rate-sensitive habituation. To illustrate how they produce this phenomenon, we simulated a thought experiment in which two hypothetical groups of animals received habituation training at short or long ISIs. In the experiment, a 0.9 intensity, one-moment duration stimulus was presented eight times at a short interval of 10 moments or a long interval of 60 moments, followed by a ninth presentation of the stimulus occurring 250 moments after the eighth. Rate-sensitive habituation would mean a greater decrease in the amplitude of the predicted response from trial 1 to 8 in the short-ISI condition than in the long-ISI condition and in a lower amplitude of response in the long-ISI than in the short-ISI condition in the ninth "test" trial.

Figure 5 depicts the essential theoretical processes of four independent simulations of the thought experiment with the SOP model. In simulations A and B, the experimental context was disregarded, so any decrement in response was due exclusively to self-generated priming. In simulations C and D, context-stimulus associations occurred, so the decrement in responding was due to a combination of self- and associatively generated priming. Simulations A and C represent habituation with the short ISI, and simulations B and D with the long ISI. First, consider the self-priming effect common to all four simulations. The first trial produces a rapid and transitory increase in A1 activity, reaching a peak equal to the $p1$ value of 0.9, followed by a rapid decrease toward zero. A2 activity is initiated shortly after A1 activity and decays slowly in all cases. It is evident that when the stimulus is presented again in Trial 2, A2 activity is sufficient to preclude full A1 activation. Thus, since the response depends on A1 activity, the second presentation of the stimulus is less effective in provoking the response than the first presentation. As seen in the four plots, this effect accumulates over time and across trials up to the point that by the eighth trial, the A1 activity occasioned by the stimulus is inhibited by the remnant A2 activity caused by all prior trials. Of course, this self-generated priming is more marked for the short (left-hand plots) than long (right-hand plots) conditions. Notice that self-generated priming loses its effect as the time from the last trial transpires, and the A2 process is allowed to decay almost completely. Thus, in the ninth trial, the amplitude of A1 is considerably recovered relative to the eighth trial but still lower than the first trial in all four simulations.

Let us turn now to the effects of associative priming of SOP, which can be appreciated by comparing the top (no associative priming) with the bottom (associative priming) plots of Figure 5. Generally, the model predicts more recovery in the short than in the long interval when associative priming is operating (Simulations C vs. D) but no difference when it is not (Simulations A vs. B). The effect of priming is due to a more protracted decay of $A2$ activity in the bottom plots compared to the top plots, especially in the case of the long ISI condition (right). In sum, the SOP model requires both self-generated and associatively generated priming to describe rate-sensitive habituation.

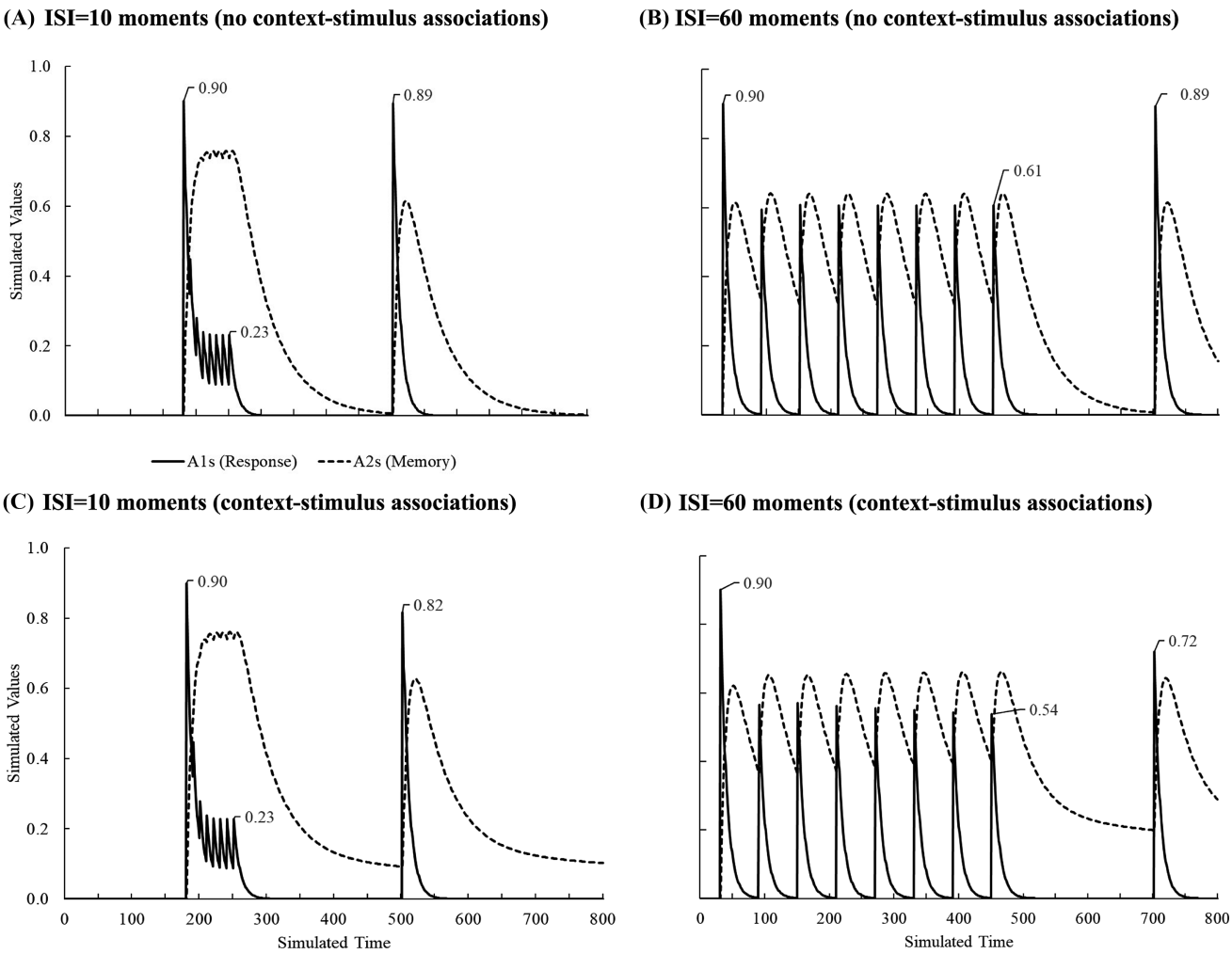
Figure 6 depicts the essential theoretical processes of four independent simulations with the feedback version of the MTS model.[3] Simulations A and B were conducted with only one integrator, while C and D were conducted with two. Simulations A and C represent habituation with the short interval, and simulations B and D with the long interval. The graphs depict the value of the memory, V_1 (A and B) and V_2 (C and D), and the value of the response or output, X_2 (A and B) and X_3 (C and D). The value of the response at each moment is the difference between the input provided by the stimulus of intensity 0.9 and the memory. In Trial 1, since there is no previous memory of the stimulus (i.e., $V_1 = 0$), the response reaches the maximal amplitude of 0.9 in all simulations. Subsequently, the presentation of the stimulus in each trial creates a memory of the stimulus proportional to b_1 in simulations A and B (corresponding to Integrator 1) and to b_1 and b_2 in Simulations C and D (corresponding to Integrators 1 and 2, respectively), leading to a decrement in response strength from Trial 1 to 8 in all simulations.

In the single-unit simulations of Figure 6 (top plots), memory decays in time exclusively in proportion to the fast a_1 parameter, so habituation in the first eight trials is larger for the short (Panel A)

[3] We conducted equivalent simulations with the feedforward version of the MTS model and obtained results that were ordinally identical to those of the feedback version.

Figure 5

Simulations of the Theoretical Processes Involved in Rate-Sensitive Habituation According to the Standard Operating Processes Model



Note. Each plot displays the results of a simulation in which a one-moment stimulus was presented eight times at a short interval of 10 moments (Panels A and C) or a long interval of 60 moments (Panels B and D), followed by a ninth presentation of the stimulus occurring 250 moments after the fourth. The top plots display the simulations without representing the experimental context. In the bottom plots, the context is assumed to be on throughout the simulation. The numbers in the plots represent the peak A1 activity (i.e., the peak response) at trials 1, 8, and 9. ISI = interstimulus interval.

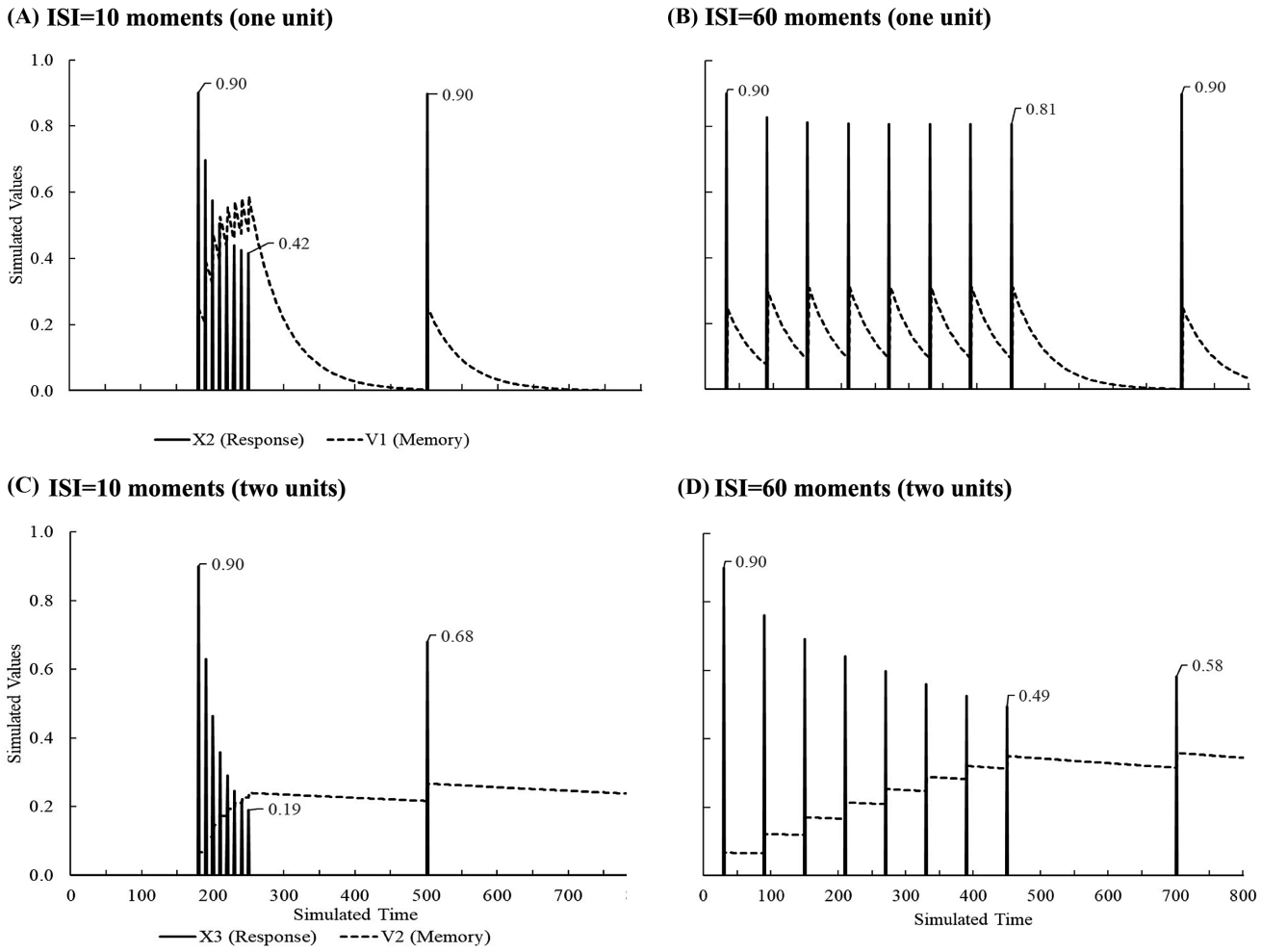
than for the long (Panel B) ISI, but no difference is observed at Trial 9 when the memory has equally decayed for the two ISI conditions. On the contrary, in Simulations C and D, memory is a compound of fast- and slow-decaying units (proportional to a_1 and a_2, respectively, where $a_1 < a_2$). Thus, in the long ISI condition (D), the early-fast unit, V_1, decays substantially from trial to trial, so the response in Trial 9 depends almost exclusively on the values of the late-slow unit, V_2. Conversely, in the short-ISI condition (C), the early-fast unit does not decay as much during the ISI, precluding the formation of a strong memory in the late-slow unit, V_2. In this case, since the response in Trial 9 depends almost exclusively on the values of the early-fast unit, V_2, recovery is larger in Simulation C than D. In sum, the MTS model requires at least two integrators with different decay rates to describe rate-sensitive habituation.

The simulations of Figures 5 and 6 reveal that the SOP and MTS theories are alike in several ways. The primary similarity between them is that they both involve a form of dual-processes memory where one is favored by stimuli presented at short intervals and the other by long intervals. In the case of SOP, memory is equivalent to A2 activity activated by intrinsic (self-generated priming) and extrinsic (associatively generated priming) processes. In the case of MTS, memory is a composite of at least one fast- and one slow-decaying unit. Unsurprisingly, when using comparable parameters, the numerical results of simulations with the two models are very similar.

Now, we present simulations of actual experimental data reported by Rankin and Broster (1992, Experiment 1) on the habituation of the contraction responses of the nematode *C. elegans* to tactile stimulation (taps). We chose this experiment because J. E. R. Staddon and Higa (1996) used it as an empirical basis for setting the parameters of the MTS model. Since there has not been a similar exercise with the SOP model, here we present a comparative analysis

Figure 6

Simulations of the Theoretical Processes Involved in Rate-Sensitive Habituation According to the Feedback Version of the Multiple Time Scale Model



Note. Each plot displays the results of a simulation in which a one-moment stimulus was presented eight times at a short interval of 10 moments (Panels A and C) or a long interval of 60 moments (Panels B and D), followed by a ninth presentation of the stimulus occurring 250 moments after the fourth. The top plots display the results of simulations conducted with a single-unit model and the bottom with a two-unit model. The numbers in the plots represent the peak response at trials 1, 8, and 9. ISI = interstimulus interval.

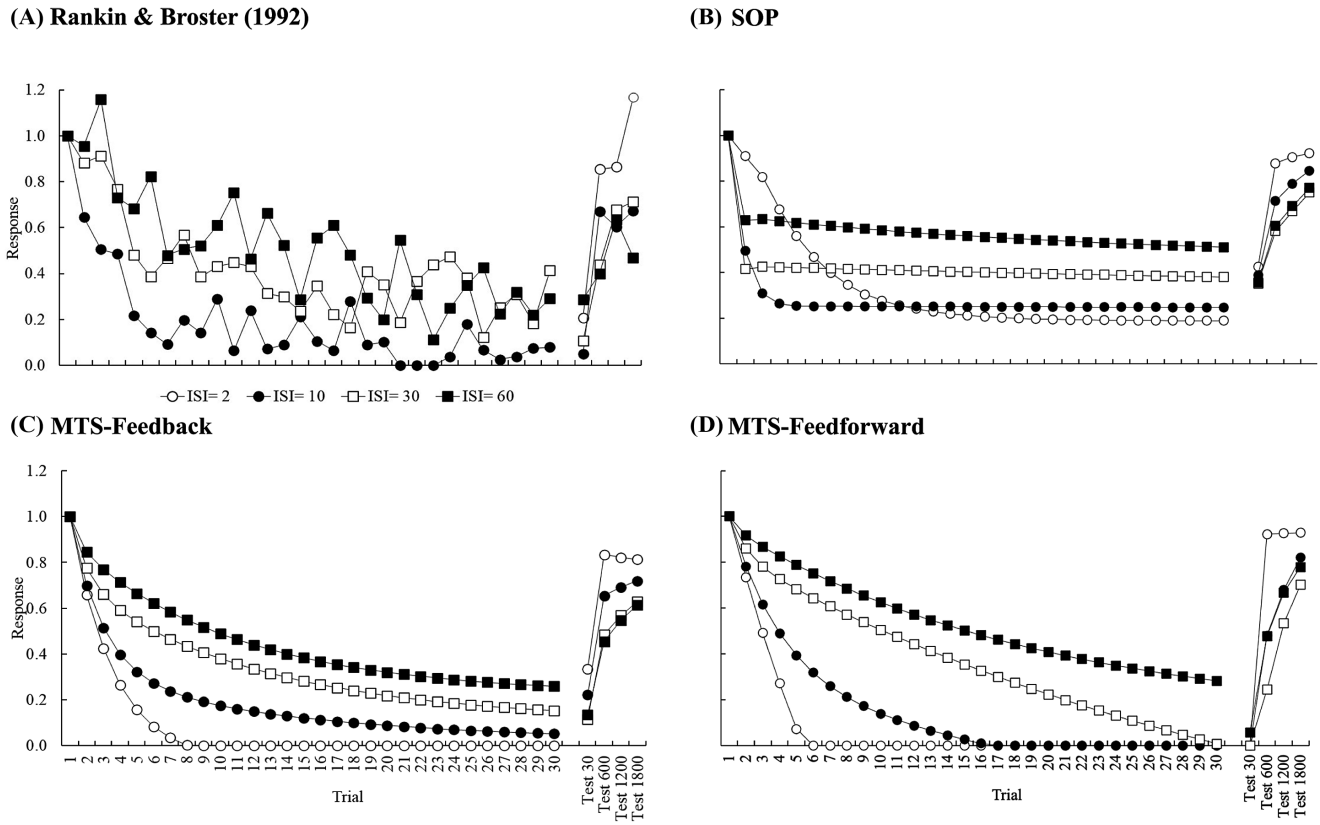
of the two models with respect to these data. In the experiment, four groups of animals received habituation training consisting of the presentation of 60 taps at intervals of 2, 10, 30, or 60 s. The amplitude of the contractile response was recorded in every habituation trial and four subsequent test trials occurring 30, 600, 1,200, and 1,800 s after habituation. To emulate the experimental conditions of Rankin and Broster (1992), the simulations comprised 60 presentations of a one-moment stimulus at intervals of 2, 10, 30, or 60 moments in the habituation phase followed by four test trials occurring 30, 600, 1,200, and 1,800 moments after habituation. The response was quantified as the maximal $A1_s$ (SOP) or X_3 (MTS) value in each trial. In this simulation and hereafter, we will use the full version of SOP (i.e., with context learning) and the two-unit version of MTS.

Panel A of Figure 7 shows the results from Rankin and Broster's (1992) study, while Panels B, C, and D show the outcomes of simulations with the SOP, MTS-feedback, and MTS-feedforward models, respectively. Overall, the figure suggests that all three models did well in predicting that shorter intervals led to more habituation during the first 30 trials and more recovery over the four testing trials. However, there were also some differences between the models. For instance, while both versions of the MTS model predict that the response decreases at a faster rate for shorter ISIs, SOP produces a more complex outcome in which habituation proceeded faster for intermediate ISIs (10 and 30 s) than for the shorter (2 s) and longest ISIs (60 s). Although the empirical data of Panel A seemed to align better with MTS's predictions, indicating that habituation proceeded faster for 10-s ISI, no data for the 2-s condition are available, which was the case where the SOP and MTS mainly differed with respect to habituation rate.

Figure 7 reveals that the models also differ in whether they predict the same or different asymptotic decrements for all ISIs. The SOP and the MTS-feedback models predict an inverse relationship

Figure 7

Data and Simulations of Rankin and Broster's (1992) Experiment 1



Note. The experiment comprised 60 habituation trials at 2-, 10-, 30-, or 60-s ISIs and four testing trials at 30, 600, 1,200 and 1,800 s after habituation. Panel A presents the approximated values of the mean standardized percent of response reported by Rankin and Broster (1992). Panels B, C, and D display the maximal predicted response according to simulations of the SOP (A), MTS-feedback (B), and MTS-feedforward models (D). The figure displays only the results of the first 30 habituation trials. SOP = standard operating processes; MTS = multiple time scales; ISI = interstimulus interval. Empirical data adapted from "Factors Affecting Habituation and Recovery From Habituation in the Nematode Caenorhabditis Elegans," by C. H. Rankin and B. S. Broster, 1992, *Behavioral Neuroscience*, *106*(2), p. 241 (https://doi.org/10.1037/0735-7044.106.2.239). Copyright 1992 by the American Psychological Association.

between the asymptotic response and the ISI. In contrast, the feedforward model predicts a trend toward equal asymptotes for all ISIs except the longest 60 s-ISI. Although the empirical data in Panel A of the figure are not conclusive concerning the asymptote, it is worth examining why the models produced such different outcomes.

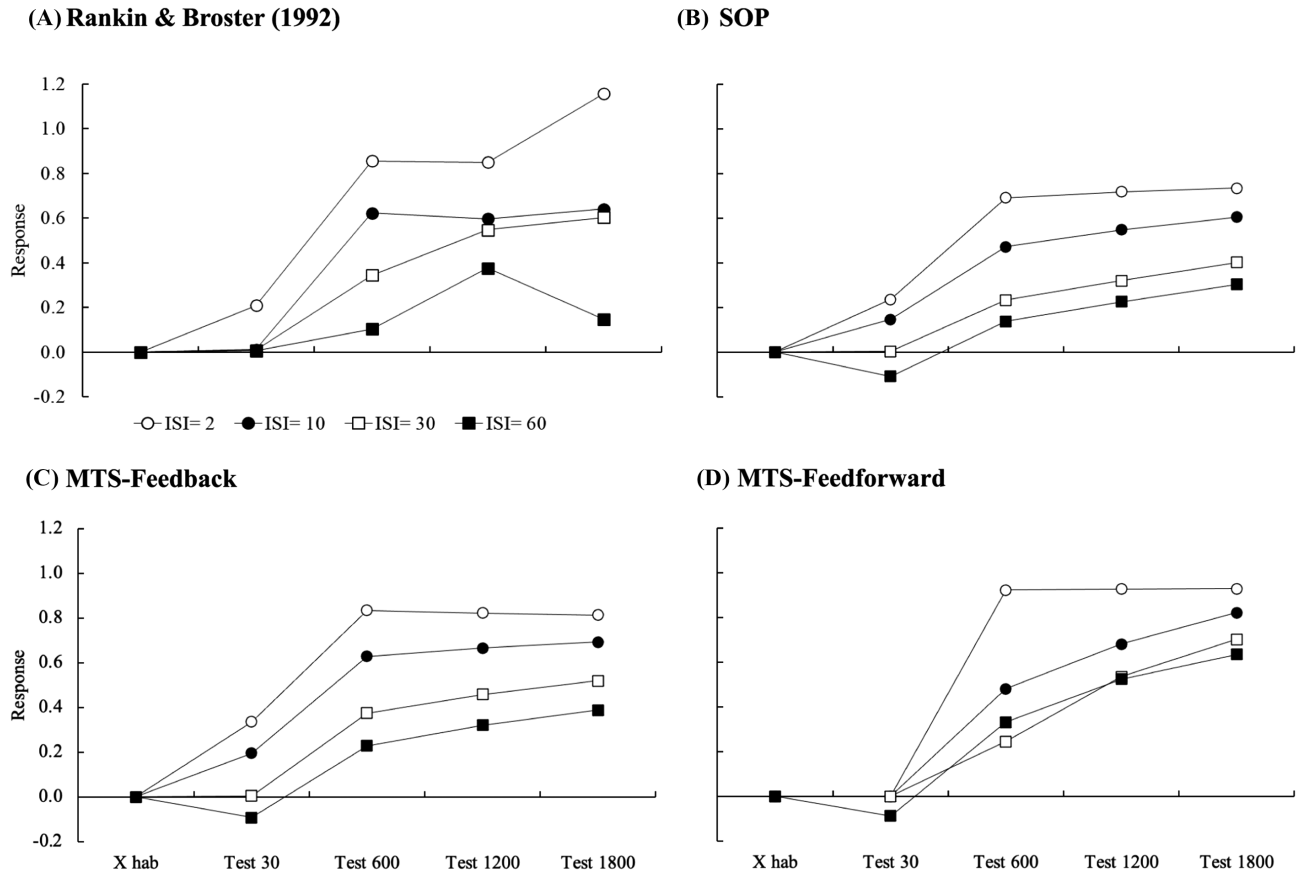
In the feedforward model, the response in each trial is the suprathreshold value of the difference between the intensity of the stimulus and the cumulative time-decaying effect of the memory of all previous trials. Thus, in the feedforward model, the memory grows without constraint in each trial, causing all ISIs to eventually develop enough memory to reduce the response to an identical threshold value. On the contrary, in the feedback version of MTS, the response in the current trial is added (or fed back) to the memory of all previous trials to compute memory. This ensures that memory growth is constrained by its own development until an asymptote is above the threshold in this model. In the case of SOP, its prediction rests on the fact that A1 and A2 activities have reciprocal influences: The more A1 activity (response), the more the subsequent A2 activity (memory), which, in turn, leads to less A1 activity on the subsequent trial. Thus, except for the first trial when A1 is large due to no previous A2 activity, the nonassociative influence of A1 on A2 and vice versa stabilizes after a few repetitions of the stimulus, producing an asymptotic response inversely proportional to the ISI.

As for the retention test of Figure 7, the panorama is also congruent between simulations and data. That is, there is a general trend toward more responding to the shorter ISIs and progressive recovery in responding as the habituation-test interval increases from 30 to 1,800 moments. This congruency can be seen more clearly in Figure 8, which displays the difference between the amplitude of response in each test minus the mean response in the last three trials of the habituation phase.

In the simulations of Rankin and Broster's data with the MTS model, we used the parameters J. E. R. Staddon and Higa (1996) found optimal for this specific data set in a two-unit model. To examine the generality of the goodness of fit of the MTS, we conducted simulations of a different data set but using the same parameters as those of Figures 7 and 8. The chosen study is the classic experiment of Davis (1970), who examined the acoustic

Figure 8

Degree of Recovery From Habituation in the Data and Simulations of Rankin and Broster's (1992) Experiment 1



Note. The magnitude of recovery was computed as the difference between the response amplitude in each test minus the mean response in the last three trials of the habituation phase (Trials 58, 59, and 60). Panel A presents the approximated values reported by Rankin and Broster in Figure 4. The simulations with the SOP (Panel B), MTS-feedback (Panel C), and MTS-feedforward (Panel D) models were the same as those reported in Figure 7. SOP = standard operating processes; MTS = multiple time scales; ISI = interstimulus interval. Empirical data adapted from "Factors Afecting Habituation and Recovery From Habituation in the Nematode Caenorhabditis Elegans," by C. H. Rankin and B. S. Broster, 1992, *Behavioral Neuroscience*, *106*(2), p. 242, (https://doi.org/10.1037/0735-7044.106.2.239). Copyright 1992 by the American Psychological Association.

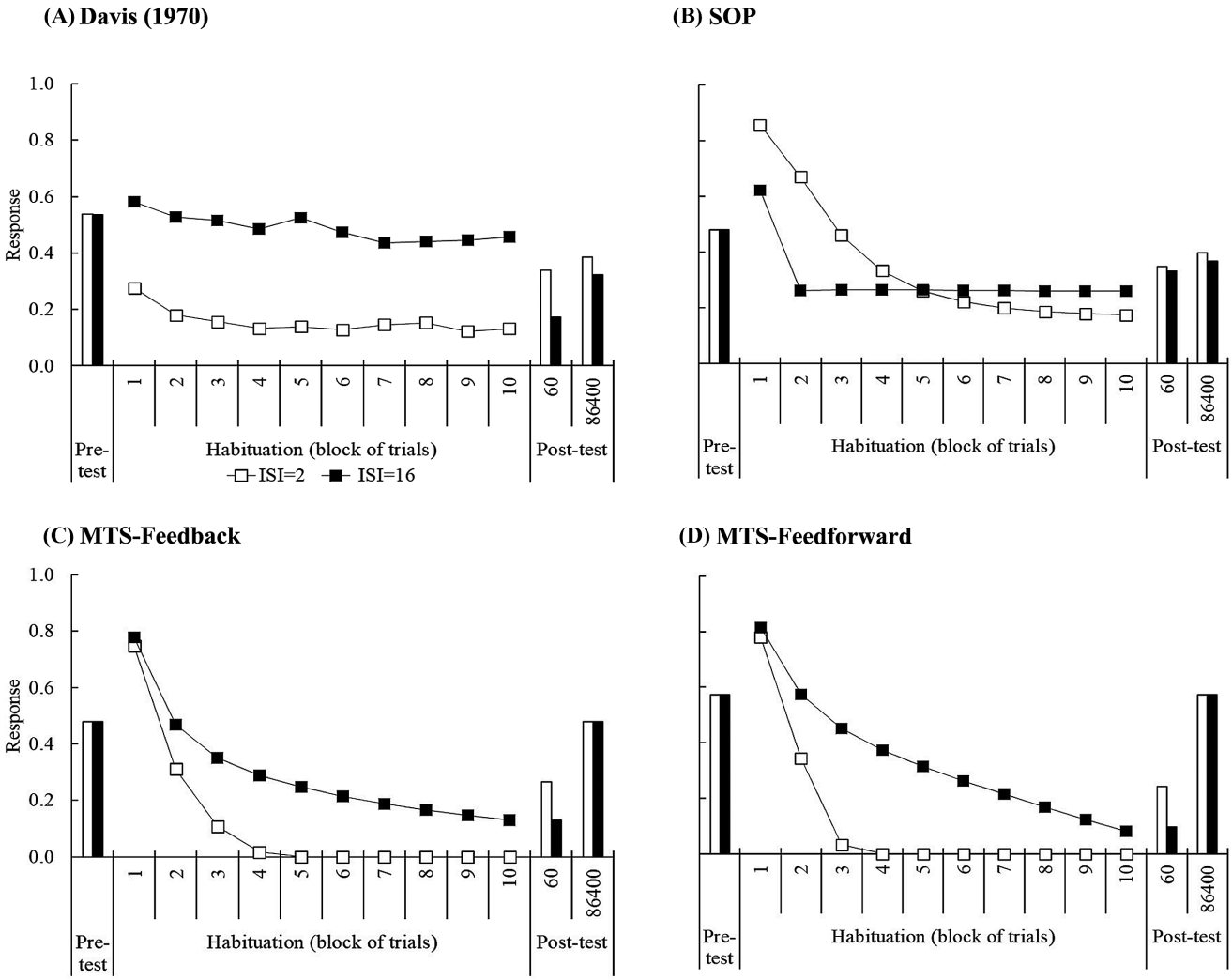
startle response to a tone in two groups of rats that received 1,000 tones with ISIs of 2 s or 16 s. The experiment began with a pretest phase in which the rats received 75 tones at each of 2-, 8-, 16-, and 32-s ISI presented in a pseudorandom sequence, totalizing 300 stimuli. The habituation phase occurred 24 hr after the pretest and consisted of the presentation of 1,000 tones with ISIs of 2 s or 16 s. One minute or 24 hr after habituation, separated groups of animals received a posttest phase that was identical to the pretest. The duration of the stimulus was 50 ms, and its intensity was 120 dB in all phases. Panel A of Figure 9 summarizes the main results of this experiment, which showed more decrement in responding in the 2-s group than in the 16-s group during the habituation phase. However, it also reveals less retention of habituation in the former than in the latter group when the test was conducted after 1 min or 24 hr.

The design of Davis's (1970) experiment is quite complex to implement in a simulation protocol. Thus, we made some simplifications. First, instead of simulating four ISIs in the test phases, we used only two, the same as the two habituating ISIs (2 and 16). Second, instead of simulating 75 trials with each ISI in the pre- and posttest phases, we simulated two trials with each. Third, to avoid order effects, we conducted six simulations with each possible combination of the two ISIs across the five trials of the pre- and posttest phases. The reported values are the mean across these simulations. Third, since we used two instead of 300 trials in the test phases, we used 20 trials in the habituation phase, representing approximately the same proportion of test to habituation trials. To keep consistency with the simulations of Figures 5–8, the stimulus duration was one moment, and the stimulus intensity ($p1$ in the case of SOP and X_1 in the case of MTS) was 0.9.

Panel B of Figure 9 shows that the SOP model appears to be accurate in predicting more decrement in the amplitude of response at asymptote in the 2-s group and more recovery in this group than in the 16-s group when the test was conducted after 1 min or 24 hr. However, the 2-s group seemed to reach its asymptote later than the 16-s group, which appears to depart from the actual data depicted in Panel A. Panels C and D demonstrate that the two versions of the MTS model correctly predict more decrement for the short than for the long interval during habituation and more retention of

Figure 9

Data (Panel A) and Simulations With the Standard Operating Processes, Multiple Time Scale-Feedback, and Multiple Time Scale-Feedforward Models (Panels B, C, and D, Respectively) of Davis's (1970) Experiment 1



Note. In the simulations, the pre-and posttest phases comprised two presentations of the stimulus at each of two different ISIs (2 and 16), totalizing five trials, and the habituation phase comprised 20 trials at either 2- or 16-moment ISIs. For each of the two habituating ISI conditions, there was one simulation in which the interval between habituation and posttest was 60 moments and another 84,400 moments. In the pre-and posttest phases, the outcome of the simulations is expressed as the maximal predicted response averaged across the five trials for each of the two habituating ISI conditions. In the habituation phase, the plots depict the maximal predicted response averaged in blocks of two trials. ISI = interstimulus interval; SOP = standard operating processes; MTS = multiple time scales. Empirical data adapted from "Effects of Interstimulus Interval Length and Variability on Startle-Response Habituation in the Rat," by M. Davis, 1970, *Journal of Comparative and Physiological Psychology*, *72*(2), p. 180 (https://doi.org/10.1037/h0029472). Copyright 1970 by the American Psychological Association.

habituation for the long interval than for the short when the test is conducted 1 min after habituation. However, the MTS model differs from the data in that habituation progresses toward equal asymptotes—especially in the feedforward version—in the two groups and in the critical fact that in the test occurring 24 hr later, retention of habituation has dissipated completely.

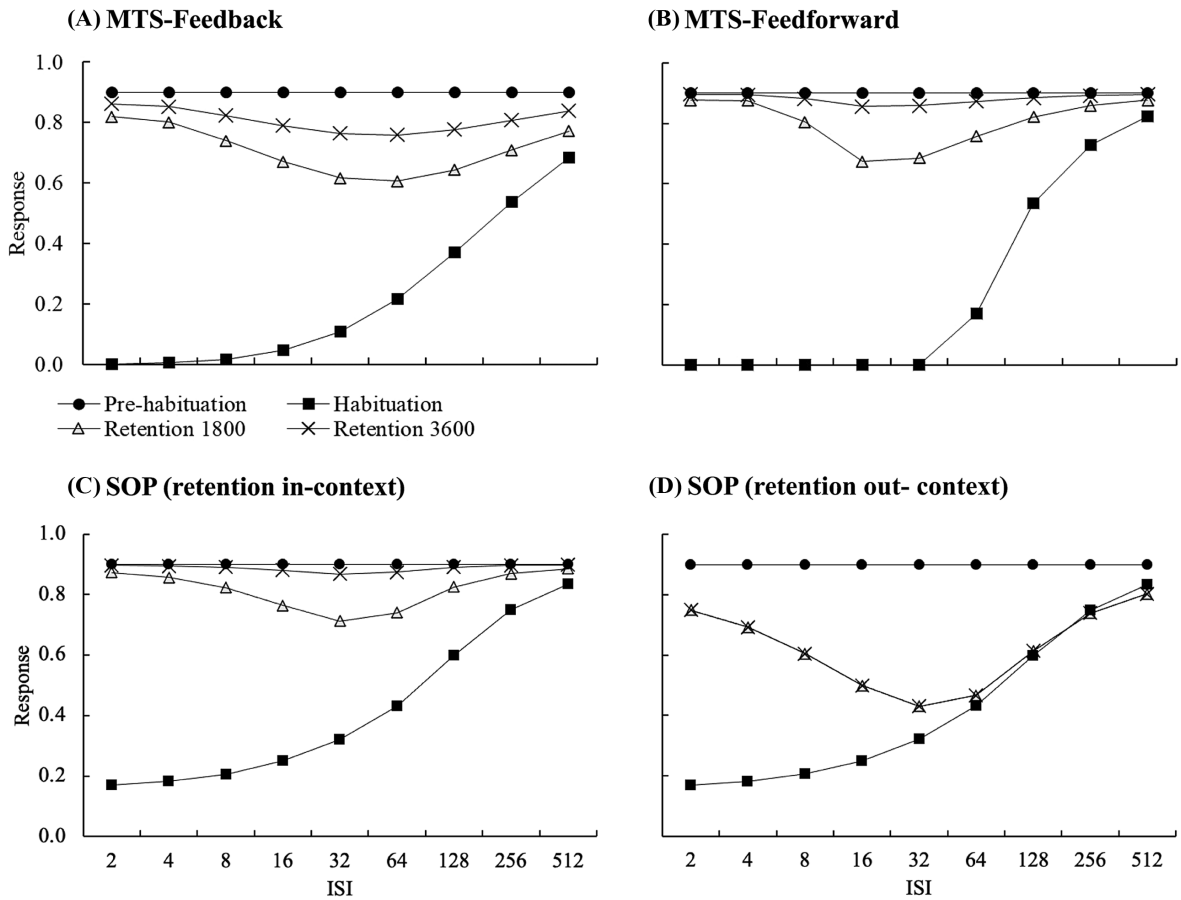
To summarize, the MTS and SOP models explain rate sensitivity habituation data. They agree that habituation is inversely proportional to the ISI during stimulus exposure but differ in aspects of habituation involved in this proportionality. According to the SOP model, the habituation's asymptote, not its rate, is inversely proportional to the ISI. On the other hand, the MTS model predicts that mainly the rate, but not as much the asymptote of habituation (especially for the feedforward version), is inversely proportional to the ISI. Both models agree that retention of habituation is directly proportional to the ISI and that it fades over time if the animals remain in the same context. However, one key difference between the models is that the amount of retention of habituation is insensitive to long retention intervals if the animals stay outside the context during the intervening time, according to SOP, but not MTS.

Further research is needed to clarify these differences. For example, if the group of animals that were tested 24 hr after habituation in Davis's (1970) experiment had spent this time in the same context as that of habituation, SOP predicts that the association between the context and the stimulus would be extinguished, and the response would be recovered at the time of posttest. The SOP and MTS models would now make similar predictions for this hypothetical case.

Figure 10 presents a general panorama of the similarities and differences between the theories regarding ISI effects. The simulations comprised 60 presentations of a one-moment duration, 0.9 intensity stimulus at 2, 4, 8, 16, 32, 64, 128, 256, or 512 moments. A single test trial occurred 1,800 or 3,600 moments after the last habituation trial. We simulated two retention periods to illustrate a critical conceptual and computational difference between the MTS and SOP models. For the MTS model, since retention depends on the rate at which memory decays in time, it would be higher for the 1,800—than for the 3,600—moment test. For the SOP model, retention of habituation depends on a context-stimulus association, which is susceptible to extinction if the animal stays in the context without the stimulus. Of course, the longer the interval (e.g., 3,600 vs. 1,800 moments), the greater the extinction. Alternatively, SOP predicts no such extinction if the retention interval occurs outside the experimental context. The outcome of the simulations corroborates these points. First, all simulations are similar in showing that the terminal level of response at Trial 60 is an increasing S-shaped function of ISI and that the level of response in the retention test (Trial 61) is a U-shaped function of ISI. Second, the feedback (Panel A) and feedforward (Panel B) versions of MTS and the SOP model with the retention interval occurring in the experimental context (Panel C) indicate more retention of habituation for the 1,800- than for the 3,600-moment retention test. On the contrary, when the retention interval happens outside the experimental context, SOP predicts identical retention levels in the 1,800 and 3,600 conditions. Thus, the differences between the MTS and SOP models regarding retention of habituation are reminiscent of the differences between decay and loss of association.

Figure 10

Simulations of a Conceptual Experiment Involving 60 Presentations of a One-Moment Duration, 0.9 Intensity Stimulus at 2, 4, 8, 16, 32, 64, 128, 256, or 512 Moments ISI



Note. Each plot depicts the predicted peak response at Trial 1 (prehabituation), 60 (habituation), and 61(retention). Retention 1,800 and retention 3,600 stand for simulations where Trial 61 occurred 1,800 or 3,600 moments after Trial 60. Simulations of the SOP model in Panels C and D differ in whether the context stayed on (i.e., $p1c = 0.05$) throughout the retention interval (Panel C) or it was turned off (i.e., $p1c = 0$) during this period. SOP = standard operating processes; MTS = multiple time scales; ISI = interstimulus interval.

The comparison between MTS and SOP concerning the hypothetical experiment presented in Figure 10 indicates that there is still room for empirical assessment of ISI effects. For example, no reports have tested SOP and MTS's predictions that there is an S-shaped function relating the ISI and STH and an optimal ISI of intermediate duration for LTH. The effects of different retention intervals occurring inside versus outside the experimental context are still awaiting further scrutiny.

Simulations of Stimulus Intensity Effects

In addition to the ISI, stimulus intensity is another aspect of repeated stimulation commonly mentioned as essential for habituation. Groves and Thompson (1970) stated a straightforward and somehow obvious relationship: "The weaker the stimulus, the more rapid and/or more pronounced is habituation. Strong stimuli may yield no significant habituation." However, Davis and Wagner (1968) and others (e.g., Colwill et al., 2023) have emphasized that this relationship holds only when the habituation and test stimuli have the same intensity. Davis and Wagner observed that when animals are habituated with either high or low intensity but tested with both intensities, the test revealed more retention of habituation in animals habituated with higher intensity. To encompass these sorts of observations, Thompson (2009) suggested that the stimulus intensity is inversely proportional to relative habituation and directly proportional to absolute habituation. Colwill et al. (2023) pointed out that this distinction is not very useful and proposed a distinction between performance and learning (i.e., habituation) instead. We use the Colwill et al. distinction hereafter.

We conducted a simulated experiment to examine what the SOP and MTS models say regarding the effects of stimulus intensity on performance and habituation. Two hypothetical groups of animals received habituation training consisting of eight presentations of a one-moment stimulus of high (0.9) or low (0.5) intensity at an interval of 60 moments. Two hundred fifty moments after the eighth trial, both groups received a single stimulus presentation at an intermediate intensity of 0.7. We used the same parameters as Figures 4C (SOP model) and 5C (MTS model). A 60-moment ISI was used instead of a 10-moment ISI, as the latter produces less retention of habituation.

According to Figure 11, both the SOP model (Panels A and B) and the MTS-feedback model (Panels C and D) resulted in similar outcomes. They showed reduced response during stimulus exposure but more recovery during testing in the low-intensity condition (Panels B and D) compared to the high-intensity condition (Panels A and C). Both theories predicted that a more intense stimulus (Panels A and C) would elicit higher responses (represented by the values of $A1_s$ and X_3 for the SOP and MTS models, respectively) than a less intense stimulus (Panels C and D) throughout the eight habituation trials. However, memory (represented by the values of A2s and V_2 for the SOP and MTS models, respectively) increased more across trials in the high-intensity condition (Panels A and C) compared to the low-intensity condition (Panels C and D), resulting in a lower response in the high-intensity condition at the common-intensity test.

In the SOP model, $p1_s$ and $pd1_s$ are the critical parameters for explaining the results of Figure 11. The more intense the stimulus (i.e., higher $p1$), the more elements will be promoted to the A1 state, leading to a response of greater amplitude (Equation 4). The parameter $pd1$, which determines the rate at which A1 activity decays toward A2 activity, is assumed to be independent of $p1$ in the model. So, the amount of A1 activity depends exclusively on $p1_s$ in the current simulations. Thus, the increased A1 activity of the higher-intensity stimulus would provide a better opportunity to develop an association between the context and the stimulus (Equation 1), leading to increased associative priming (Equation 2). In the case of the MTS model, the critical parameters are X_1 and b_1, which are the approximate equivalent to $p1$ and $pd1$ of SOP (see Table 1). In the current simulations, X_1 was set to 0.9 or 0.5, producing responses of different amplitude according to Equation 6. On the other hand, the parameters b_1 and b_2 weigh the contribution of each of these intensities to memory development in their respective units, V_1 and V_2 (Equation 8). Since b_1 and b_2 are independent of X_1 in the model, memory development in MTS depends entirely on X_1. Thus, for both theories, the stronger the stimulus, the greater the response during exposure and the more habituation when the response is evaluated at the same intensity for all conditions.

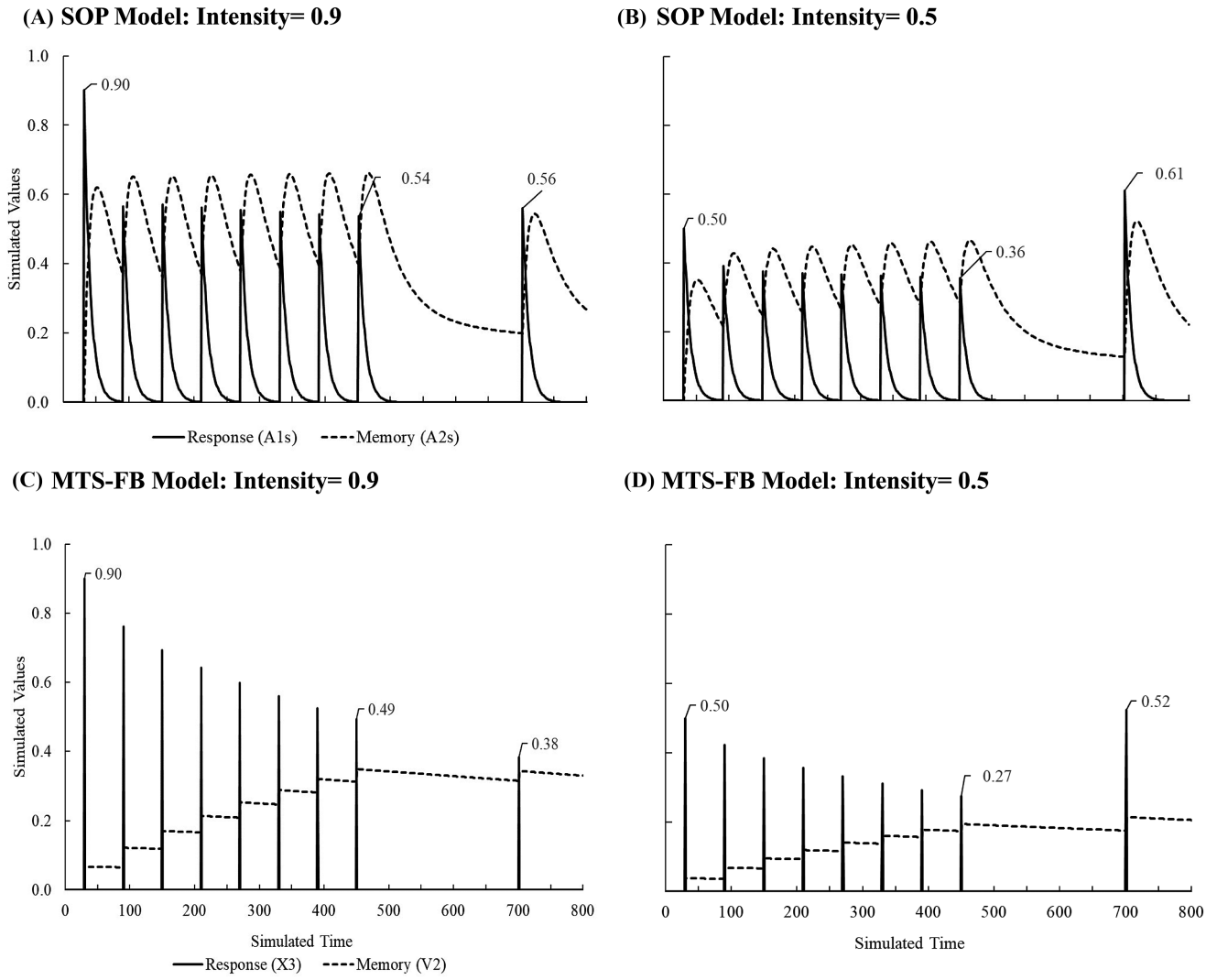
As we did in the case of rate-sensitive habituation in the previous section, we now examine the behavior of the models against the actual data. Davis and Wagner (1968) reported data on the habituation of acoustic startle responses in rats that might be very informative in this respect. We chose this experiment because it is a rare example where the intensity of the stimulus during habituation and the intensity at the test were unconfounded. The experiment comprised three phases: pretest, habituation, and posttest. During the pretest and posttest phases, the rats received 20 tones of each 96, 102, 108, 110, and 120 dB presented in a pseudorandom sequence. During the intervening habituation phase, separated by 1 min from the pretest and posttest phases, different groups of rats received 300 or 700 tones at 108 or 120 dB. The duration of the stimulus was 50 ms, and the ISI was 8 s in all phases. The significant finding was that the response amplitude to each of the five intensities was lower in the posttest relative to the pretest in the groups habituated with the 120 dB tones than in the groups habituated with the 96 dB tone. Panel A of Figure 12 depicts part of the results reported by Davis and Wagner.

To simulate Davis and Wagner's (1968) data, we made the same sort of simplifications as we did for the simulations of Davis (1970). First, instead of simulating five test intensities, we used only two, the same as the habituating intensities. Second, to avoid order effects during the pre- and posttest, we conducted two simulations with each possible order of the two intensities. The reported values are the mean across these simulations. Third, since we used two instead of 100 trials in the test phases, we used six or 14 trials in the habituation phase, which, like in the actual experiment, represents three and seven times the number of trials of the test phases. To keep consistency with previous simulations, the duration of the stimulus was one moment, and the tested intensities ($p1$ in the case of SOP and X_1 in the case of MTS) were 0.5 and 0.9. The ISI and the interphase interval were 8 and 60 moments, respectively.

Panel A of Figure 12 reproduces the data of interest from Davis and Wagner's (1968) experiment, and Panels B–D show the results of the simulations. As expected from the analysis of Figure 11, the two models replicate the experimental data reasonably well, although the effects are more pronounced in the two versions of the MTS model than in the SOP model. The effect in SOP is not very large because self-priming is substantial for the stimulus presented

Figure 11

Simulations of the Theoretical Processes Involved in Intensity-Sensitive Habituation According to the Standard Operating Processes Model (A and B) and the Feedback Version of the Multiple Time Scale Model (C and D)



Note. Each plot displays the results of a simulation in which a one-moment stimulus was presented eight times at an interval of 60 moments with an intensity of 0.9 (A and C) or 0.5 (B and D). A ninth presentation of a 0.7-intensity stimulus occurred 250 moments after the eighth presentation. The values of A1 and X_3 represent the response, and the values of A2 and V_2 represent the memory in the SOP and MTS models, respectively. The numbers in the plots represent the peak response at trials 1, 8, and 9. SOP = standard operating processes; MTS = multiple time scales; MTS-FB = multiple time scale feedback.

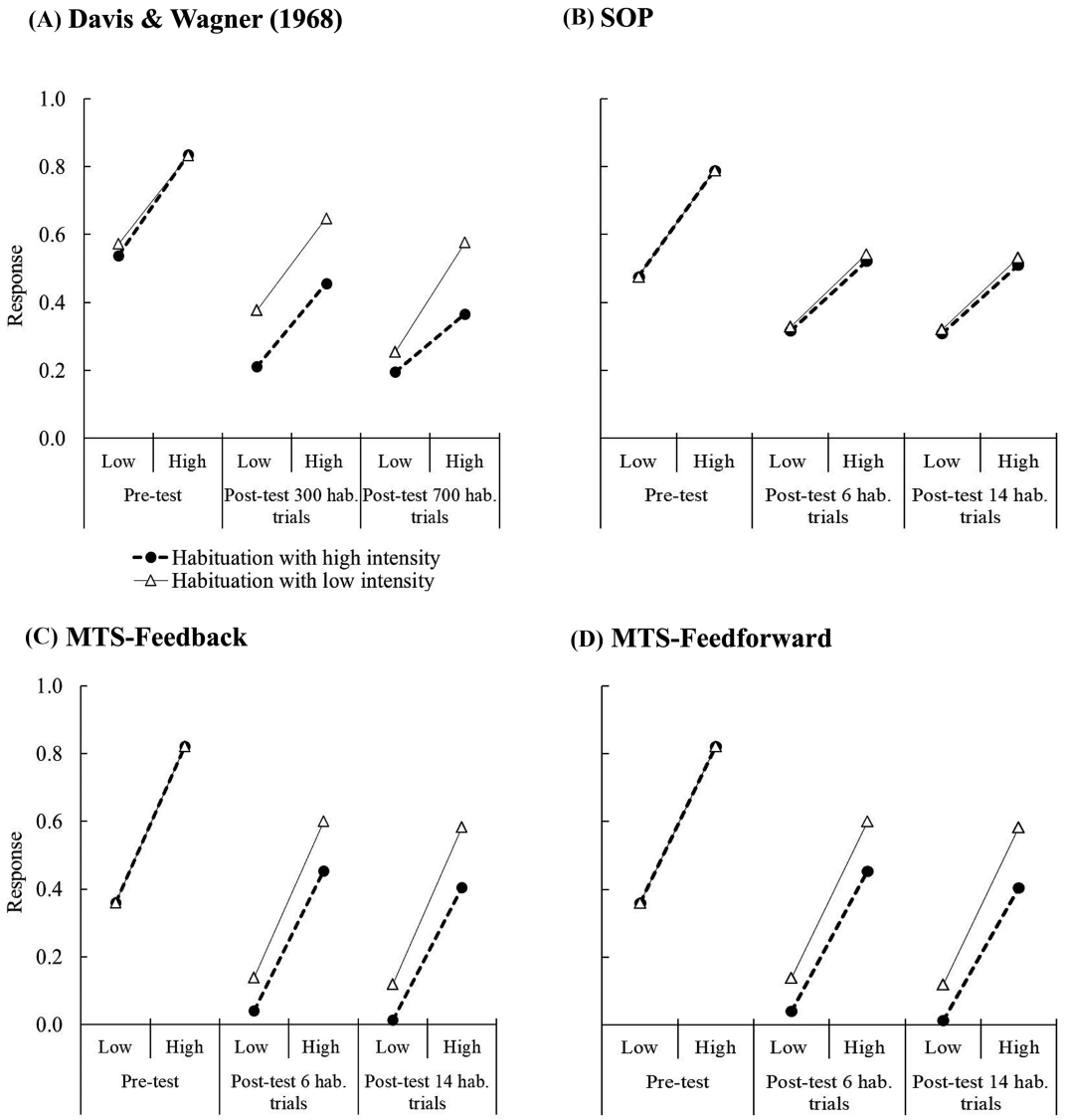
at eight-moment ISI during the test, so such a nonassociative influence somewhat obscures the differences in associative priming caused by habituation with different intensities. This smallish effect is not necessarily a wrong prediction since, in other response systems, no effects of stimulus intensity on the retention of habituation have been shown (e.g., Lozada et al., 1990).

Unfortunately, the study by Davis and Wagner has not been replicated, nor has it been further explored with different ISIs. However, James and Hughes (1969) offered some data on the habituation of the human skin conductance response, which could provide extra information to assess the models. In their experiment, participants received 12 trials with a 2-s noise at a mean ISI of 60 s. Half of the participants were exposed to the first eight stimuli at 60 dB, while the other half experienced them at 76 dB. For Trials 9–12, half of the participants in each group were subjected to stimuli at either 70 or 73 dB. Figure 13A illustrates the main findings, showing that although the participants who received the initial eight trials with a lower intensity of 67 dB responded less than those who experienced noise at 76 dB during the first eight trials, the pattern reversed in the last four trials when both groups were tested at intermediate intensities. Thus, James and Hughes essentially reproduced the findings of Davis and Wagner (1968).

Figure 13 presents the results of James and Hughes (1969) along with simulations of the SOP model and the two versions of the MTS model. The figure shows that data and simulations are very consistent at an ordinal level in all cases. During the first eight trials,

Figure 12

Data (Panel A) and Simulations With the Standard Operating Processes, Multiple Time Scale-Feedback, and Multiple Time Scale-Feedforward Models (Panels B, C, and D, Respectively) of Davis and Wagner (1968)



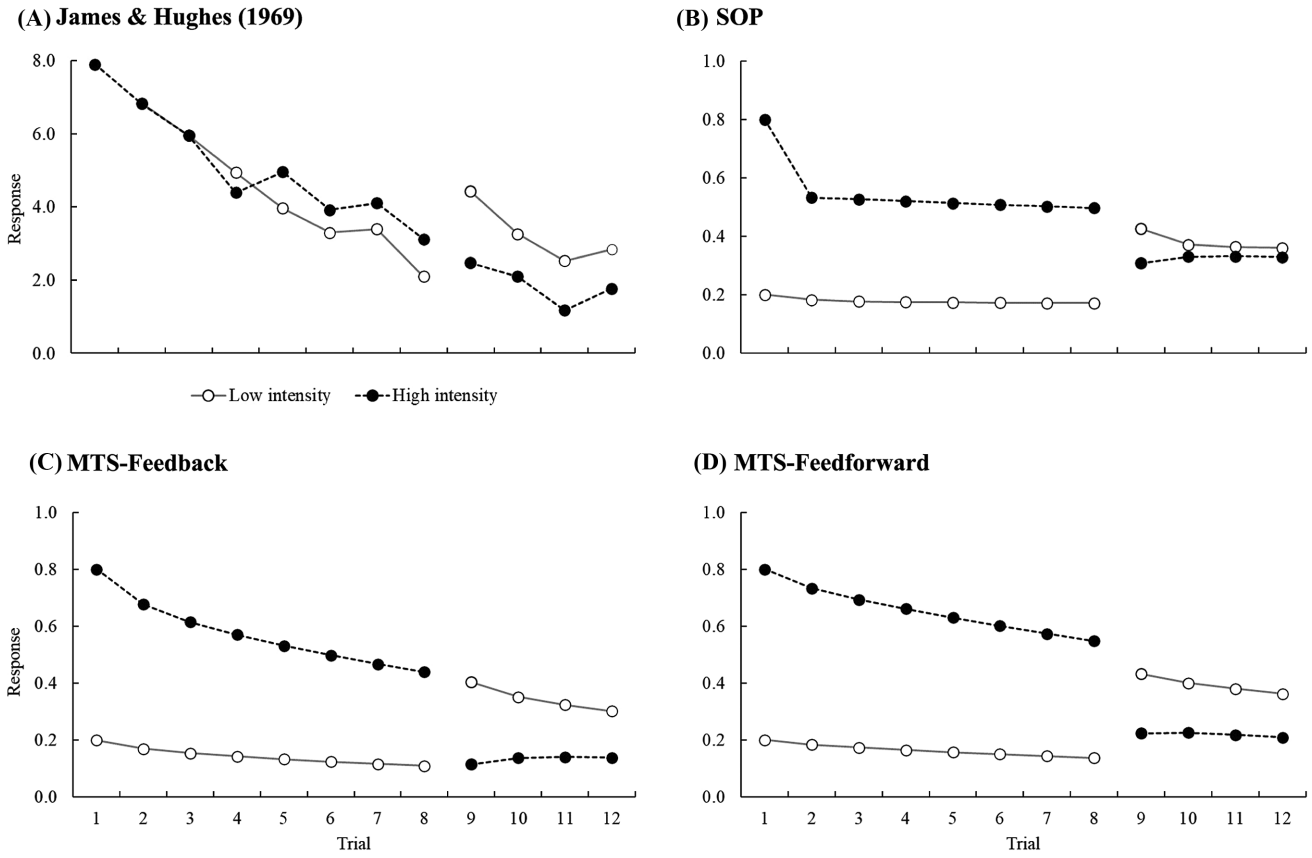
Note. In the simulations, the pre-and posttest phases comprised one stimulus presentation at each of two different intensities (0.5 and 0.9), totalizing two trials, and the habituation phase comprised either six or 14 trials. The interval between habituation and pre-and posttest was 60 moments. In the pre-and posttest phases, the outcome of the simulations is expressed as the maximal predicted response averaged across the two trials for each of the four conditions. SOP = standard operating processes; MTS = multiple time scales. Empirical data adapted from Figure 1 of "Startle Responsiveness After Habituation to Different Intensities of Tone," by M. Davis and A. R. Wagner, 1968, *Psychonomic Science*, *12*(7), p. 337 (https://doi.org/10.3758/BF03331339). Copyright 1968 by Springer Nature. Adapted with permission.

there was more response in the high-intensity condition than in the low-intensity condition, but the opposite was observed when the groups were compared with identical intensities in Trials 9–12. In agreement with the simulations of Davis and Wagner (1968), the two versions of the MTS models seem to produce more pronounced effects than the SOP for James and Hughes's (1969) data.

Comparing Davis and Wagner's (1968) and James and Hughes's (1969) studies is challenging due to using different species and response systems. However, two procedural differences are worth noting. Unlike Davis and Wagner, James and Hughes did not conduct a pretest with the tested intensities, preventing potential learning during this stage. Secondly, James and Hughes (1969) used a more prolonged ISI than Davis and Wagner (60 vs. 8 s), which may create a more favorable condition for observing the effect of associative versus self-priming in the SOP model. Given the scarcity of data on this topic (we are aware of only one further study with a design of this type by Jackson, 1974), one should exercise caution when interpreting the theoretical significance of the simulations of Figures 12 and 13.

Figure 13

Data (Panel A) and Simulations With the Standard Operating Processes, Multiple Time Scale-Feedback, and Multiple Time Scale-Feedforward Models (Panels B, C, and D, Respectively) of James and Hughes (1969) Experiment



Note. The experiment involved eight habituation trials at 60-s intervals (data) or moments (simulation) ISI. In one condition, the first eight trials were at a low intensity of 67 dB (data) or 0.2 $X_1/p1$ (simulations) and in the other at a high intensity of 76 dB (data) or 0.8 $X_1/p1$ (simulations). In Trials 4 to 12, each of these groups was subdivided into groups that received the trials at one of two intermediate intensities of 70 dB (data) or 0.4 $X_1/p1$ (simulations) or 73 dB (data) or 0.6 $X_1/p1$ (simulations). SOP = standard operating processes; MTS = multiple time scales; ISI = interstimulus interval. Empirical data adapted from "Generalization of Habituation of the GSR to White Noise of Varying Intensities," by J. P. James and G. R. Hughes, 1969, *Psychonomic Science*, *14*(4), p. 163 (https://doi.org/10.3758/BF03332767). Copyright 1969 by Springer Nature. Adapted with permission.

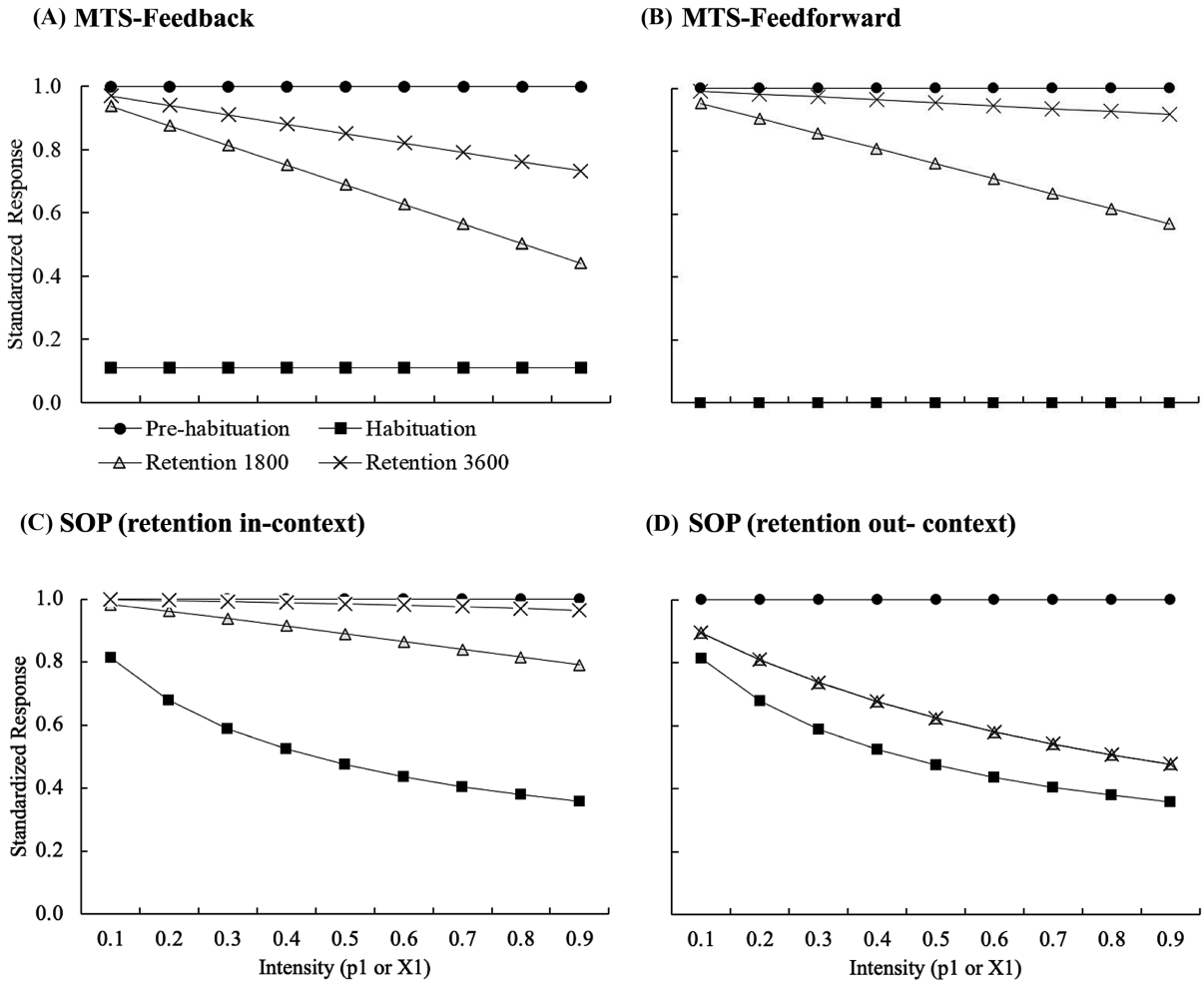
In the previous section, we emphasized the fact that the ISI effects predicted by the MTS and SOP models are sensitive to the retention interval, that is, the intervening time between habituation and test, and that this sensitivity can be considerably reduced in the case of the SOP, but not the MTS model, if the retention interval is spent outside the experimental context. Given that similar mechanisms are involved in the predictions of each model for the intensity effects, the same sort of differential sensitivity should hold for the intensity effects. To illustrate this, we conducted computer simulations with a range of intensities. The simulations comprised 60 presentations of a one-moment stimulus at an ISI of 30 moments. Separated simulations were conducted with one of each nine intensities ranging from 0.1 to 0.9. A single test trial occurred 1,800 or 3,600 moments after the last habituation trial. In the test trial, the intensity was 0.5 in all simulations. In the simulation in Figure 14, the two versions of the MTS model (Panels A and B) predict that the standardized level of response at the last habituation trial is identical for all habituating intensities. In contrast, the SOP model (Panels C and D) predicts an inverse relationship between the habituating intensity and the standardized response. Secondly, all models agree in predicting that at the retention test, when the tested intensity is the same for all conditions, the level of response is an inverse function of the ISI, which is consistent with Davis and Wagner's (1968) and James and Hughes's (1969) findings. This effect is, however, attenuated when the retention interval is increased from 1,800 to 3,600 moments in the two versions of the MTS models. In the case of SOP, this attenuation happens when the retention interval is conducted in the experimental context (Panel C) but not when it occurs outside the context (Panel D).

Models Fit With a Larger Parametric Space

In this article, we did not optimize the parameters of each model for each simulation, which may raise doubts about how well the models perform under different combinations of their respective parameters. To provide a preliminary assessment of the robustness of each model fitting to the empirical data, we conducted 81 versions of each simulation of Figure 7 (Rankin & Broster, 1992),

Figure 14

Simulations of a Conceptual Experiment Involving 60 Presentations of a One-Moment Stimulus at 30-Moments ISI With Intensity Ranging From 0.1 to 0.9



Note. Each plot depicts the predicted peak response at Trial 1 (prehabituation), 60 (habituation), and 61(retention). Retention 1,800 and retention 3,600 are simulations where Trial 61 occurred 1,800 or 3,600 moments after Trial 60. Simulations of the SOP model in Panels C and D differ in whether the context stayed on (i.e., $p1c = 0.05$) throughout the retention interval (Panel C) or it was turned off (i.e., $p1c = 0$) during this period. MTS = multiple time scale; SOP = standard operating processes; ISI = interstimulus interval.

Figure 9 (Davis, 1970), Figure 12 (Davis & Wagner, 1968), and Figure 13 (James & Hughes, 1969) using meaningful combinations of the most critical patterns of these models.

Our focus for the SOP model was on the parameters *pd*1 and *pd*2, which play a crucial role in controlling the decay probabilities of A1 and *A*2 activity. To explore this, we examined nine different values of *pd*1, ranging from 0.1 to 0.9, and nine *pd*1/*pd*2 ratios ranging from 1 to 9. These choices were not arbitrary but based on A. R. Wagner's (1981) suggestion that the decay from A1 to A2 must be faster than from A2 to *I* to produce substantial priming in the temporal range of his demonstrations. Thus, although we included the case in which $pd1 = pd2$, we dismiss the possibility of $pd2 > pd1$ as it would not produce meaningful priming effects.

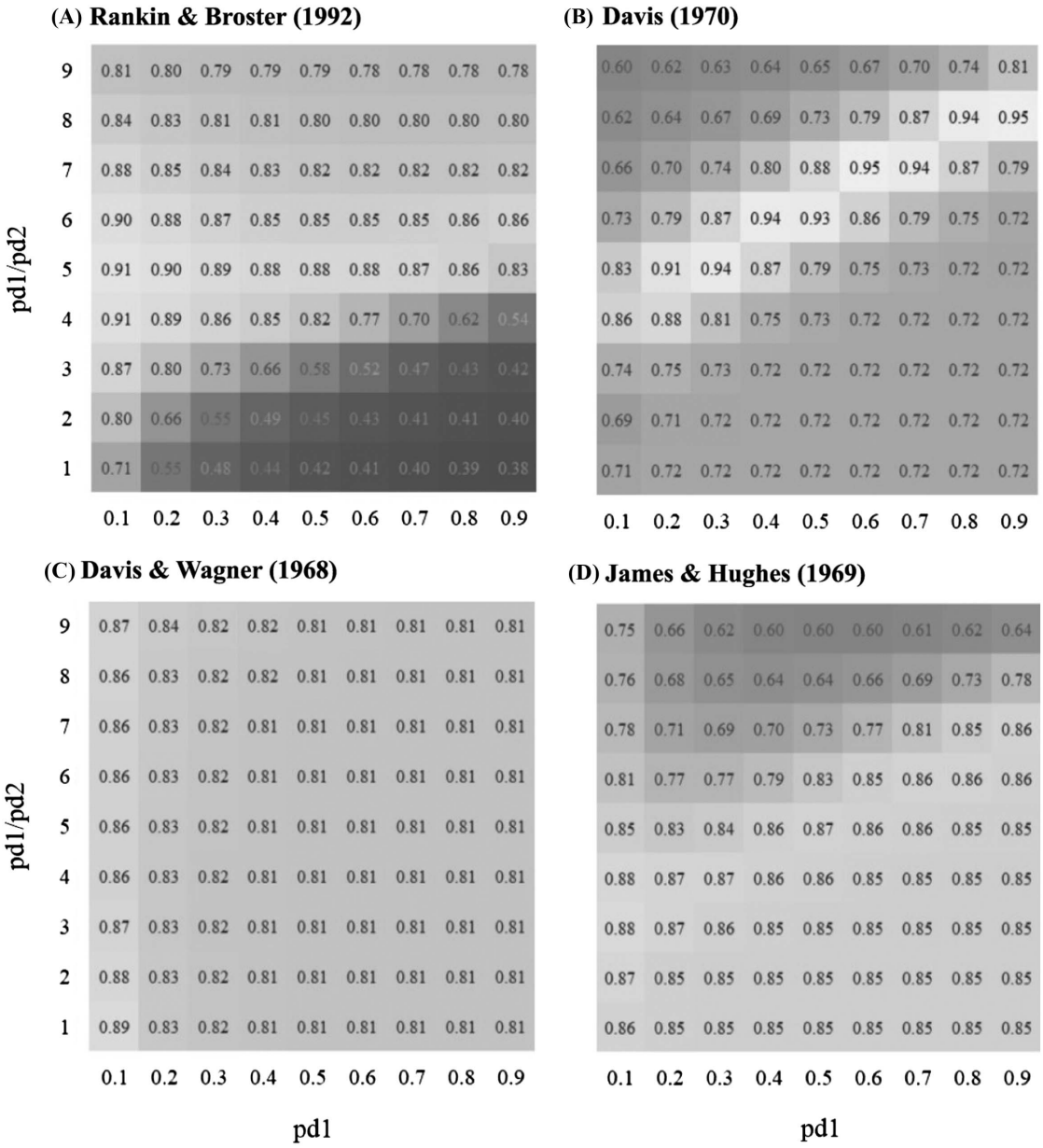
Regarding the MTS model, as mentioned in the introduction of this article, J. E. R. Staddon and Higa (1996) examined the models' fit to Rankin and Broster's (1992) data by varying λ_a from 0.1 to 5 in steps of 0.1 and λ_b from 0.1 to 3 also in steps of 0.1. Although they let both parameters vary independently, they found a better fit when $\lambda_a > \lambda_b$. To examine a parametric space like that of Staddon and Higa while obtaining some comparability with the parametric space explored in SOP, we examined nine different values of λ_a, ranging from 0.6 to 5 and nine λ_a/λ_b ratios ranging from 1 to 9.

Figures 15 and 16 provide a visual representation of the correlation coefficients of predicted-observed test values for each set of parameters for the data of Rankin and Broster (1992), Davis (1970), Davis and Wagner (1968), and James and Hughes (1969) according to the SOP and MTS-feedback models, respectively.[4] Each cell is color coded according to its values, with lighter shades indicating higher correlation and darker shades indicating lower

[4] For brevity, no simulations with the feedforward version of the MTS model are presented in this section.

Figure 15

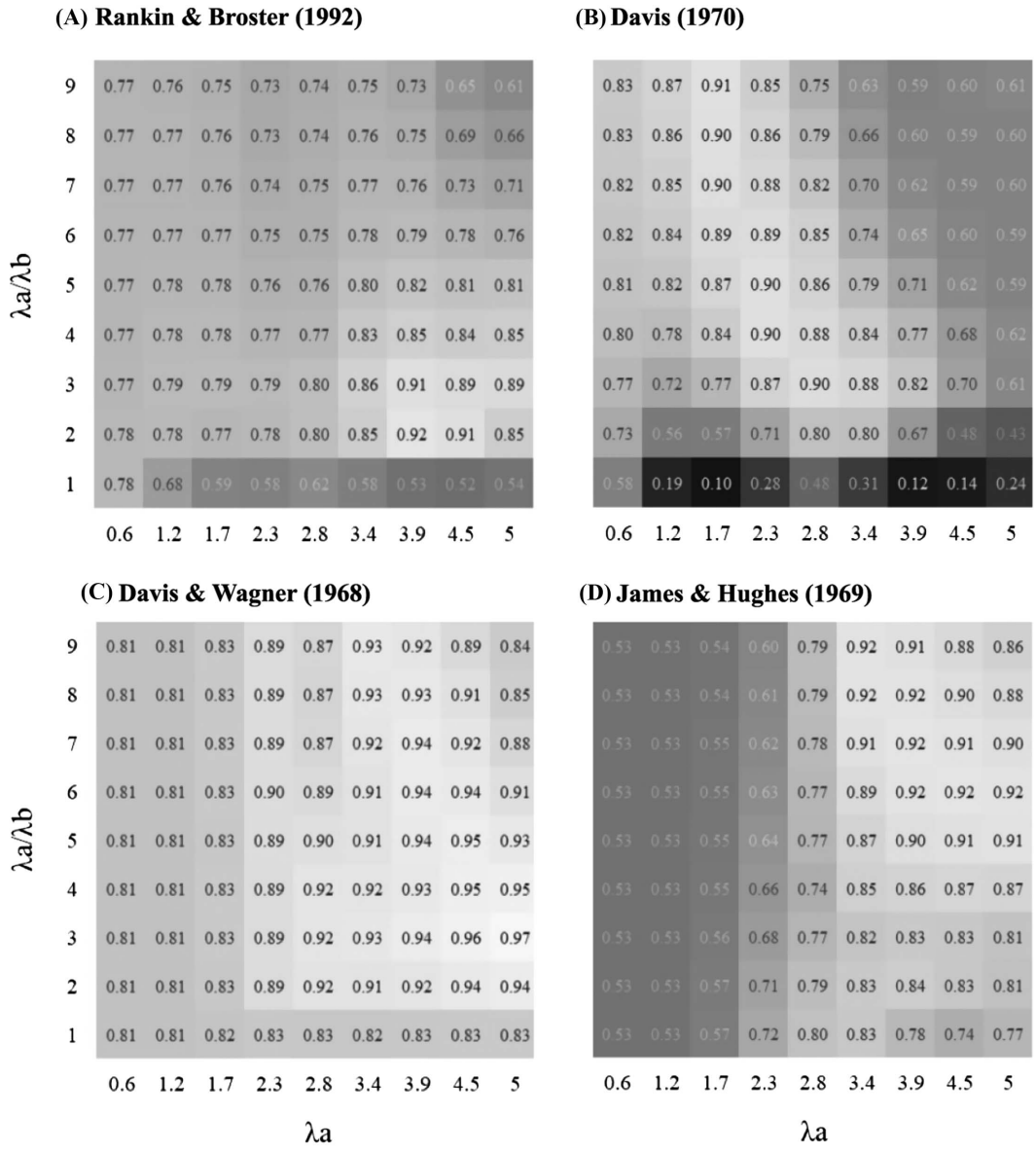
Correlation Coefficients Between Predicted and Experimental Data for 81 Different Combinations of pd1 and pd1/pd2 Values of the Standard Operating Processes Model



Note. Each cell is color coded based on its values, with lighter shades indicating higher correlation and darker shades indicating lower correlation (computed with Microsoft's Visual Basic, assuming black = 0 and white = 1). In Panel A, the correlation coefficients were based on 16 pairs of values corresponding to the four test trials for each of the four ISI conditions of data and simulations of Rankin and Broster (1992). In Panel B, the correlation coefficients were based on four pairs of values corresponding to the mean value in the test trials for each of the two ISI conditions and the two retention intervals of data and simulations of Davis (1970). In Panel C, the correlation coefficients were based on eight pairs of values corresponding to the two tested intensities for each of the two habituated intensities and the two number of habituation trials of data and simulations of Davis and Wagner (1968). In Panel D, the correlation coefficients were based on eight pairs of values corresponding to the four test trials for each of the two intensities conditions of data and simulations of James and Hughes (1969). ISI = interstimulus interval.

Figure 16

Correlation Coefficients Between Predicted and Experimental Data for 81 Different Combinations of λ_a and λ_a/λ_b Values of the Multiple Time Scale-Feedback Model



Note. Each cell is color coded based on its values, with lighter shades indicating higher correlation and darker shades indicating lower correlation (computed with Microsoft's Visual Basic, assuming black = 0 and white = 1). In Panel A, the correlation coefficients were based on 16 pairs of values corresponding to the four test trials for each of the four ISI conditions of data and simulations of Rankin and Broster (1992). In Panel B, the correlation coefficients were based on four pairs of values corresponding to the mean value in the test trials for each of the two ISI conditions and the two retention intervals of data and simulations of Davis (1970). In Panel C, the correlation coefficients were based on eight pairs of values corresponding to the two tested intensities for each of the two habituated intensities and the two number of habituation trials of data and simulations of Davis and Wagner (1968). In Panel D, the correlation coefficients were based on eight pairs of values corresponding to the four test trials for each of the two intensities conditions of data and simulations of James and Hughes (1969). ISI = interstimulus interval.

correlation. This visual aid makes comparing the performance of different models and parameter combinations easier.

The analysis of SOP's correlations shown in Figure 15 leads to the following observations: First, the values of $pd1 = 0.1$ and $pd1/pd2 = 5$ used in this article provided good fits of 0.91, 0.83, 0.86, and 0.85 for Rankin and Broster's (1992), Davis's (1970), Davis and Wagner's (1968), and James and Hughes's (1969) simulations, respectively. Second, several other values also fit the

four simulated experiments well, especially those near the original parameters (e.g., $pd1 = 0.2$, 0.3, or 0.4, and $pd1/pd2 = 4$, 5, or 6). Third, for the Rankin and Broster experiment, good fits can be obtained with most of the explored parameter space, except when $pd1 > 0.1$ and $pd1/pd2 < 5$ (darker area in Panel A). Fourth, for the Davis (1970) experiment, the best fits are obtained when the $pd1/pd2$ ratios increase approximately proportionally to $pd1$ (for instance, a good fit of 0.95 is obtained when $pd1 = 0.9$ and $pd1/pd2 = 9$ and of 0.94 when $pd1 = 0.3$ and $pd1/pd2 = 5$). Fifth, the fits of SOP to the intensity effects (bottom matrices), which range between 0.81 and 0.89 for Davis and Wagner's data and between 0.88 and 0.60 for James and Hughes's data, are more homogeneous than the fits to the ISI effects (top matrices), which range between 0.91 and 0.38 for Broster and Rankin's data and between 0.60 and 0.95 for Davis's data. This pattern is not surprising since intensity effects primarily depend on the differential value of the peak A1 activity caused by stimuli of different intensities, which is proportional to $p1$ and relatively independent of $pd1$ and $pd2$ when the duration of the stimulus is one moment, as assumed in this article.

Figure 16 shows the results of the correlations obtained with the MTS-feedback model. The values of $\lambda_a = 3.9$ and $\lambda_a/\lambda_b = 3$ used in this article provided good fits of 0.91, 0.82, 0.94, and 0.83 for Rankin and Broster's (1992), Davis's (1970), Davis and Wagner's (1968), and James and Hughes's (1969) simulations, respectively. Other values near the original parameters (e.g., $\lambda_a = 3.4$, and $\lambda_a/\lambda_b =$ 3 or 4) also fit the four simulated experiments well. However, except for the predictions of Davis and Wagner's (1968) data, in which the correlations are consistent (ranging from 0.81 to 0.97), the correlations vary substantially across the parametric space. Specifically, the correlations vary between 0.52 and 0.92 for Rankin and Broster's data, from 0.10 to 0.91 for Davis's (1970) data, and from 0.53 to 0.92 for James and Hughes's data. In general, across all four experiments, it was observed that the poorest correlations are obtained when $\lambda_a/\lambda_b = 1$, especially with lower λ_a values.

Further Predictions on ISI and Intensity Effects

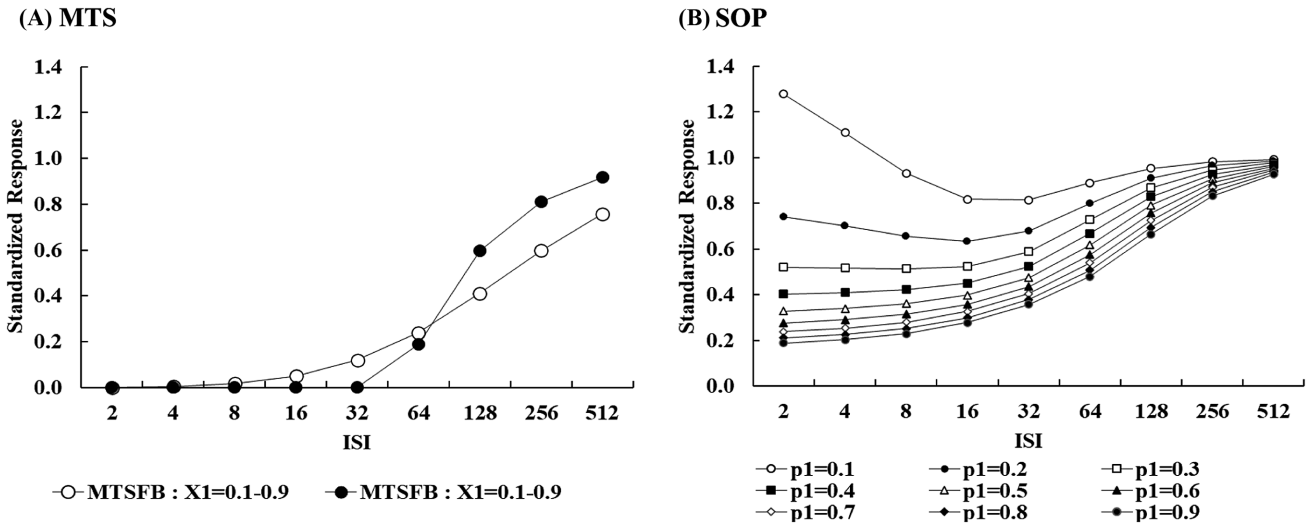
In the previous sections, we show that, at least at the ordinal level, the two theories account for ISI and intensity effects on habituation equally well. However, the predictions of the theories, either those in which they agree or disagree, go beyond the available empirical information. Consider an imagined experiment in which different groups of animals received 60 trials with one of the 81 ISI × Intensity conditions that result from the combination of nine ISIs (2, 4, 8, 16, 32, 64, 128, 256, and 512) and nine intensities (0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, and 0.9).

Concerning the course of response decrement across stimulus repetition, the simulations of Figure 17 indicate that models generally agree in predicting a higher terminal response (i.e., less habituation) for longer ISIs. The only exception to this trend is the prediction of SOP of an increase rather than a decrease in responding over trials to low-intensity stimuli ($p1 = 0.1$) presented at short ISIs (2 and 4). This effect is attributed to the temporal summation of the $A1$ process in SOP, which MTS does not predict with the current parameters. Another noteworthy difference between the models is that when the response is standardized based on the initial level of response, the relative decrement in response is the same for all intensities in MTS, but it increases with intensity in SOP. These subtle but essential differences have yet to be tested.

Regarding retention of habituation, the two theories agree that the relationship between the intensity of the stimulus and the response in a retention test is inverse and that the relationship between the ISI and the response is a U-shaped function. Figure 18 illustrates that this is a general prediction across all 81 simulations; as commented before, the essential difference emerges when the retention interval

Figure 17

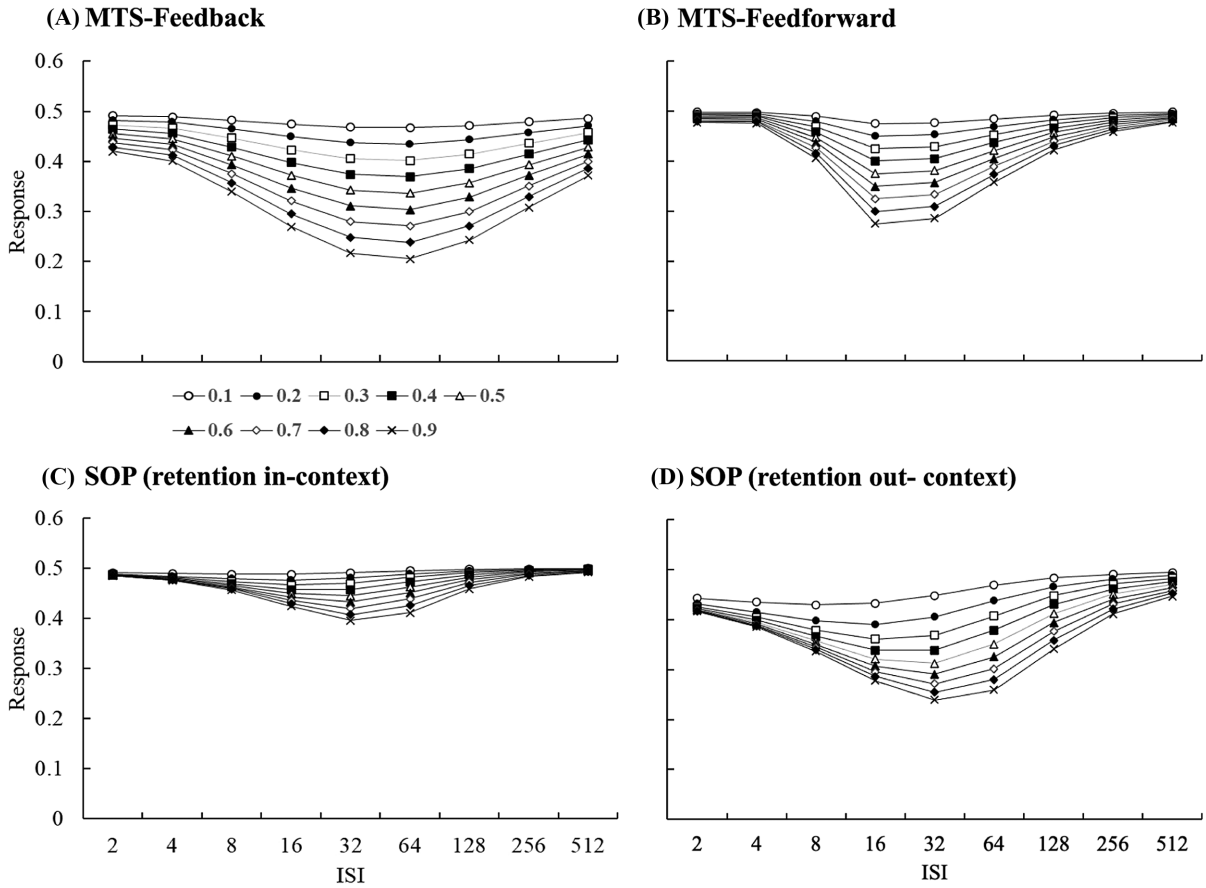
Simulations of a Conceptual Experiment Involving 60 Presentations of a One-Moment Stimulus at ISIs Ranging From 2 to 512 Moments With Intensity Ranging From 0.1 to 0.9



Note. Each plot depicts the standardized response at Trial 60. The remaining parameters are the same as those of Figures 5–14. MTS = multiple time scale; SOP = standard operating processes; MTSFB = multiple time scale-feedback; MTSFF = multiple time scales-feedforward.

Figure 18

Simulations of a Conceptual Experiment Involving 60 Presentations of a One-Moment Stimulus at ISIs Ranging From 2 to 512 Moments With Intensity Ranging From 0.1 to 0.9



Note. Each plot depicts the predicted peak response in a retention trial 1,800 moments after Trial 60. Simulations of the SOP model in Panels C and D differ in whether the context stayed on (i.e., $p1c = 0.05$) or off (i.e., $p1c = 0$) during the retention interval, respectively. The remaining parameters are the same as those of Figures 5–14. MTS = multiple time scale; SOP = standard operating processes; ISI = interstimulus interval.

occurs in or outside the context. For the MTS model, the length of time since the last stimulus determines the amount of habituation retained at the test, irrespective of whether the time transpires in or out of the experimental context. On the contrary, for the SOP model, the critical aspect determining the amount of retention is the amount of time in the context.

As stated in the introduction, the SOP model suggests that the retention of habituation depends on the association between the context and the stimulus. As a result, it predicts that the retention of habituation should be specific to the context. However, the evidence for this point is controversial since different measures of retention of habituation have shown variable sensitivity to changes in context. For instance, Jordan et al. (2000) measured startle and lick suppression in rats that were exposed to several tones and found that only lick suppression showed substantial recovery with a shift in context, although habituation was observed in both measures. Similarly, Pinto et al. (2014) found context specificity of the habituation of the cardiac response but not of the eyeblink response in the same experiment with humans. There have been reports of a lack of contextual control of the habituating startle response in rats (Marlin & Miller, 1981), in contrast to reports of contextual control in a range of other response systems, such as the escape response of crabs (Tomsic et al., 1998) and scape in invertebrates (Rankin, 2000). Refer to Dissegna et al.'s (2021) article for a comprehensive review.

An alternative is the distinction between decay and loss of association. The MTS model is essentially a "decay model" because it posits that spontaneous recovery should increase as the retention interval increases. SOP predicts that this increase happens for a limited range of intervals, after which the level of recovery stabilizes unless context-stimulus associations are allowed to extinguish by exposing the animals to the context alone. The fact that retention of habituation is susceptible to decay in the case of MTS and to loss of association in the case of SOP is one of the few aspects in which the models make contrasting and testable predictions. A few habituation protocols tend to agree with the decay hypothesis (Beck & Rankin, 1997; Menzel, 2001; Moyer, 1963; Hermitte et al., 1999; Rankin & Carew, 1987), but there is also evidence of null effects of the retention interval in the degree of spontaneous recovery (Black et al., 1964; Leaton, 1974). However, we are unaware of studies

comparing the effects of different retention intervals when animals spend those intervals in and out of the context, so this issue must wait for further assessments.

Dishabituation and Sensitization According to Standard Operating Processes and Multiple Time Scale

In this article, we preferred to keep the quantitative analysis as simple as possible to contrast the fundamentals of the MTS and SOP theories. Of course, our simulations of the ISI and intensity effects do not exhaust the field's empirical wealth. Conspicuous examples are observations of "dishabituation" and "sensitization," which refer to situations where the response increases rather than decreases during stimulus-repetition protocols.

Dishabituation consists of a transient recovery in a habituated response due to the interposition of an innocuous stimulus or "dishabituator" between two presentations of the target stimulus (e.g., Whitlow, 1975). Humphrey (1933) speculated that this was due to habituation being fragile, reversible, and sensitive to global changes in stimulation conditions. However, several researchers, including Humphrey, quickly found evidence that response increases could be far more complicated. For example, it is possible to increase the magnitude or frequency of a response when presenting a novel stimulus, even before habituation to the target stimulus has occurred. This transient increase in response can also occur "spontaneously," that is, without the presentation of a dishabituator (Thompson & Spencer, 1966).

Moreover, under certain circumstances, the repetition of the stimulus could produce a relatively enduring increase in reactivity (Davis, 1974). An eventual fragility of habituation did not explain these results alone, and a new concept emerged: sensitization (Davis & Wagner, 1969; Groves & Thompson, 1970; Hilgard & Marquis, 1940). Thus, sensitization refers to the observation that the behavioral decrement that typically follows stimulus repetition is sometimes delayed, reduced, restored, or even replaced by a transient increment in the response (Thompson, 2009). Unlike dishabituation, the demonstration of sensitization might or might not involve the presentation of an additional stimulus and does not require that habituation has occurred. Despite the principled similarity between sensitization and dishabituation and some debate concerning their interdependence, there is substantial behavioral and neurobiological evidence indicating different underlying processes (Bristol & Carew, 2005; Hochner et al., 1986; Marcus et al., 1988; Steiner & Barry, 2014). In line with this distinction, Wagner and colleagues (e.g., Mazur & Wagner, 1982; Wagner, 1981; Wagner & Vogel, 2010; Whitlow & Wagner, 1984) sustain that dishabituation might occur at the level of the mnemonic processing of the stimulus (through the parameters *pd*1 and *pd*2 of SOP) and sensitization at the level of the input (through the parameter *p*1).

To explain dishabituation, Wagner proposed that animals have a limited capacity for processing stimuli. This means that a maximum A1 and A2 activity can be generated anytime across all stimulus representations. The model represents this limit as an increase in the decay rates (*pd*1 and *pd*2) proportional to the summed A1 and A2 activity across all stimuli at a given moment. Therefore, if an extraneous stimulus or distractor is presented while the target stimulus is being processed, the decay rates *pd*1 and *pd*2 of the target stimulus will increase, leading to a faster return of its elements to inactivity. As a result, the elements of the target stimulus will be released from self-generated priming and will be available for activation sooner due to the distractor.

Wagner's distractor rules led to several predictions about the effectiveness of distractors. For example, since the distractor affects the decay rates of other cue representations, it would only influence the activity of already activated cues. Thus, dishabituation should be specific to a just-presented stimulus, as Whitlow (1975) demonstrated. Additionally, the effectiveness of dishabituation depends on the distractor's capacity to generate its own A1 and A2 activity, so it will vary based on the distractor's intensity, duration, and temporal locus. Moreover, since contextual cues can also prime the distractor, the phenomenon of "habituation of dishabituation" (Thompson & Spencer, 1966) is straightforwardly predicted by the SOP model.

The concept of competition for a limited processing capacity, as A. R. Wagner (1981) suggested to explain dishabituation, does not contradict the principles of MTS. In fact, J. Staddon (1993) proposed that dishabituation could be achieved through a temporary reduction in the threshold (θ) or by resetting the memory (V_1 and V_2). The second alternative is more consistent with the idea of limited capacity. It can be imagined that the rate of memory decay, a_1 and a_2, is temporarily increased by the presentation of a distractor. However, without a formal implementation of these possibilities, it is difficult to determine whether the MTS model will make predictions like SOP's predictions regarding whether dishabituation is stimulus specific, temporally sensitive, and susceptible to habituation.

Concerning sensitization, Wagner and colleagues explored the possibility of explaining it through nonassociative and associative influences on the *p*1 parameter of the SOP model. For instance, Wagner and Brandon (1989) proposed an extension of the model, which suggests that a stimulus can elicit two types of responses: sensory-motor and emotive. While the sensory-motor response is the response of interest in most habituation experiments, the emotive response is not typically measured. They assumed that the target stimulus activates two sets of A1/A2 units, with one representing the sensory-motor aspect and the other representing the emotional-arousing aspect. Any stimulus can provoke emotive tendencies and act to potentiate the activation parameter, *p*1, of any other stimuli, including itself. Therefore, the efficacy with which a stimulus would provoke its response, *p*1, would be an increasing function of the emotive state of the organism.

This assumption to incorporate sensitization to SOP leads to different possibilities of response-potentiating tendencies, ranging from transient and nonassociative to durable associative sensitization. For example, Wagner and Vogel (2010) suggested that habituation training with an aversive stimulus, such as a loud noise, can lead to the context controlling long-term habituation to that specific stimulus. Furthermore, it can produce a conditioned emotional response that would enhance the response to any other stimulus, including the habituated one. If long-term habituation is seen as relatively specific to the exposed stimulus, and long-term sensitization is viewed as more globally influential, then even if they are both associatively mediated and context dependent, one should be able to distinguish the influences by tests, not only of the exposed stimulus but of other potentially effective stimuli.

Innis and Staddon (1989) proposed, in turn, that differential sensitization could be achieved through temporal summation of the stimulus input in the MTS model. To achieve this, they suggested varying the weight of the parameter "b_i", which determines the contribution of the stimulus to memory formation and response

(see and Equations 7 and 8).[5] They also suggested that variation in this parameter across different species might explain why some organisms show an initial increase in response before habituation while others do not. As Wagner and Brandon (1989) did with the SOP model, it is conceivable in the MTS model to let the b_1 parameter vary as a function of the emotive state of the organism. However, the computational implications of these assumptions are yet to be evaluated.

Final Remarks

In this article, we compared two distinct quantitative instantiations of the Sokolovian idea that habituation depends on an inhibitory relationship between memory and perception of the eliciting stimulus: Allan Wagner's SOP model (Mazur & Wagner, 1982; A. R. Wagner, 1981; Whitlow & Wagner, 1984) and John Staddon's multiple time scale model (Innis & Staddon, 1989; J. Staddon, 1993; J. E. R. Staddon & Higa, 1996). We chose these models among the various theories available, as they provide enough quantitative details to make testable predictions and propose different mechanisms to explain habituation.

Despite being several decades old, it is surprising that there has been almost no cross-citation between the SOP and MTS's main articles, suggesting that they might have reached different audiences. This lack of interaction could be due to language and implementation differences rather than conceptual divergences. Additionally, the models might be difficult to implement for users without advanced programming training, so their predictions cannot be easily derived. In this article, we expressed both theories using a common conceptual framework and comparable mathematical approaches and parameters to address these issues.

Our analysis revealed striking similarities between MTS and SOP, resulting in identical predictions for several well-known empirical data. At first sight, one may wonder where this is an example of theoretical underdetermination attributed to theories of learning (e.g., Soto, 2019). However, we were able to derive contrasting predictions by doing a fine-grained analysis of the sort of functional mechanism that each theory offers to explain habituation and its retention in time. A fertile field for future assessment of these two views of habituation is in the rich domain of decay, forgetting, and extinction, whose major procedures and theoretical analysis (e.g., Bouton, 1994; de Oliveira Alvares & Do-Monte, 2021) have been underused in habituation.

Given the precedent analysis and the existing literature, we cannot recommend one theory over the other with certainty. The MTS model is, in some respect, more parsimonious, explaining short- and long-term habituation through a single, nonassociative mechanism. On the other hand, the SOP model is more complex, as it involves associative and nonassociative mechanisms to explain habituation. However, the SOP model can accommodate context-specific habituation, which is not allowed by the MTS model. Furthermore, the SOP's stimulus processing rationale naturally accommodates sensitization and dishabituation. In conclusion, both theories are valuable and should be compared against each other more often.

Soto (2019) recommended including neurobiological constraints in the computational models to solve theoretical underdetermination (e.g., Soto, 2019). Although such constraints were not explicitly stated in the formulation of SOP and MTS, there have been some speculations on their biological plausibility. For instance, Hawkins and Kandel (1984) noticed a striking similarity in the reciprocal relationship between facilitatory and inhibitory neurons involved in the learning circuit of the aplysia and the A1 and A2 processes of SOP. Likewise, J. E. R. Staddon et al. (2002) pointed out a correspondence between the cascaded structure of the MTS model and the sequentiallike pattern of cortical activation in people subjected to stimulus repetition protocols (Uusitalo et al., 1996). These examples of structural isomorphism between the neural circuit in the aplysia and the SOP model and between the sequence of cortical processes in the human brain and the MTS model are more inspirational than validatory in the sense of Soto's claim. Furthermore, we showed in this article that beyond the differences in the layout of the two theories, they have commonalities in the proposed functional entities involved in habituation.

The current trend in the neurobiology of habituation suggests that multiple mechanisms are involved (McDiarmid et al., 2019; Randlett et al., 2019). Thus, it is conceivable, for instance, that retention of habituation occurs through simple decay in some species or response systems, as proposed by MTS, and through a combination of decay and loss of association in others, as proposed by SOP. This multiple-mechanism view provides opportunities for further behavioral studies, especially those conducted in inter- and intraspecies research programs.

Research on habituation across various species has been ongoing for over a century, resulting in significant empirical information. Several theories have been proposed to encompass this empirical body, differing in their level of formalization, empirical focus, and level of analysis. Most of these theories are rooted in two primary approaches. First is Sokolov's (1963, 1969) comparator theory, which suggests that repeated stimulation leads the animal to develop "impulses signaling the operation of an expected stimulus," also referred to as "impulses of extrapolation." These impulses are then compared to subsequent stimulation to determine the immediate response and succeeding stimulus processing. The second approach is Thompson and Spencer's (1966) dual process theory, which posits that the presentation of a stimulus triggers two independent processes—a decremental process known as habituation, intrinsic to the stimulus–response circuit, and an incremental process called sensitization, which serves as an extrinsic modulator. Although these processes would depend on different neural substrates, they combine to produce the net behavioral effect of stimulus repetition.

Dual process theory emphasizes the influences of short-term changes in behavior and supposes that these changes occur at the level of reflex pathways. As a result, it has gained significant acceptance among those interested in habituation in simple organisms such as nematodes (Kepler et al., 2020) and mollusks (Bristol & Carew, 2005) and in the neurobiological mechanisms responsible for incremental and decremental effects of stimulus repetition (Ardiel et al., 2017; Glanzman, 2009; Kandel, 1976, 1978). In this tradition, several authors have proposed quantitative theories aimed at modeling the underlying cellular mechanisms (Byrne & Gingrich, 1989; Hawkins, 1989; Gluck & Thompson, 1987; Poon & Young, 2006; Santos et al., 2007) or at describing mathematically the functions of response decrement and increment

[5] Note that the model proposed by Innis and Staddon (1989) is a preliminary version of MTS and that the letters "a" and "b" refer to different parameters than those used in subsequent presentations of the theory and in the current article.

over trials (e.g., Del Rosal et al., 2006; Stanley, 1976; Wang, 1993; Wang & Arbib, 1992).

The comparator theory emphasizes long-term changes in behavior due to changes in mnemonic representations of events and has been particularly cited in human research (O'Gorman & Jamieson, 1975; Spinks & Siddle, 1976; Siddle et al., 1978), especially on the cortical correlates of habituation (Barry et al., 2016, 2020; MacDonald et al., 2015). However, associative memory of the stimulus is also central to theories where the cortex does not necessarily play a central role in habituation. Some of these theories propose that the response to a stimulus weakens with repetition because its internal representation becomes predicted by the formation of Pavlovian conditioning links. For some theories, the Pavlovian association is established between the stimulus and external cues (Konorski, 1967; Wagner, 1976, 1978, 1981), and for others, with internal aspects of the stimulus itself (Hall & Rodríguez, 2019; McLaren & Mackintosh, 2000; Ratner, 1970; Stein, 1966).

It is important to note that many well-articulated theories combine elements of both the comparator and dual-process theories. For example, the SOP model considers habituation as involving intrinsic influences in the stimulus–response pathways (self-generated priming), like the dual-process theory, and extrinsic associative influences (associative priming), like the comparator theory. Another example is Barry's (2006, 2009) preliminary process theory, which explains the habituation of the orienting response in humans through a combination of the dual-process theory and a "cortical set" that modulates response based on stimulus significance (an approximation of Sokolov's view). Additionally, in the context of individual differences in visual habituation in infants, there's a growing interest in "hybrid" theories of habituation, such as those proposed by Hunter and Ames (1988), Sirois and Mareschal (2002), Thomas and Gilmore (2004), and Schöner and Thelen (2006). Some of these theories characterize individuals' responses as a dynamic function of immediate stimulation and the history of the organism's encounters with similar situations. Like the dual process theory, these approaches rely on the interplay of activation and inhibition processes that determine whether individuals will respond to the stimulus.

New theories of habituation are regularly being developed, with a specific focus (e.g., Poli et al., 2024) or a general focus (e.g., Gershman, 2024; Ramaswami, 2014). Due to their increasing complexity and formal differences, determining their novelty or advantages compared to previous theories can be challenging. In our article, we examined two theories, SOP and MTS, and found that they are more similar than previously thought despite minimal cross-citation. This exercise suggests that perhaps only a few mechanisms may be necessary to explain habituation. We propose that, nowadays, theoretical integration and empirical comparison are more needed than new theories in habituation research.

References

Ardiel, E. L., Yu, A. J., Giles, A. C., & Rankin, C. H. (2017). Habituation as an adaptive shift in response strategy mediated by neuropeptides. *npj Science of Learning*, *2*(1), Article 9. https://doi.org/10.1038/s41539-017-0011-8

Barry, R. J. (2006). Promise versus reality in relation to the unitary orienting reflex: A case study examining the role of theory in psychophysiology. *International Journal of Psychophysiology*, *62*(3), 353–366. https://doi.org/10.1016/j.ijpsycho.2006.01.004

Barry, R. J. (2009). Habituation of the orienting reflex and the development of Preliminary Process Theory. *Neurobiology of Learning and Memory*, *92*(2), 235–242. https://doi.org/10.1016/j.nlm.2008.07.007

Barry, R. J., Steiner, G. Z., & De Blasio, F. M. (2016). Reinstating the novelty P3. *Scientific Reports*, *6*(1), Article 31200. https://doi.org/10.1038/srep31200

Barry, R. J., Steiner, G. Z., De Blasio, F. M., Fogarty, J. S., Karamacoska, D., & MacDonald, B. (2020). Components in the P300: Don't forget the Novelty P3! *Psychophysiology*, *57*(7), Article e13371. https://doi.org/10.1111/psyp.13371

Beck, C. D., & Rankin, C. H. (1997). Long-term habituation is produced by distributed training at long ISIs and not by massed training or short ISIs in *Caenorhabditis elegans*. *Animal Learning & Behavior*, *25*(4), 446–457. https://doi.org/10.3758/BF03209851

Black, R. W., Fowler, R. L., & Kimbrell, G. (1964). Adaptation and habituation of heart rate to handling in the rat. *Journal of Comparative and Physiological Psychology*, *57*(3), 422–425. https://doi.org/10.1037/h0043044

Bouton, M. E. (1994). Conditioning, remembering, and forgetting. *Journal of Experimental Psychology: Animal Behavior Processes*, *20*(3), 219–231. https://doi.org/10.1037/0097-7403.20.3.219

Brandon, S. E., & Wagner, A. R. (1998). Occasion setting: Influences of conditioned emotional responses and configural cues. In N. A. Schmajuk & P. C. Holland (Eds.), *Occasion setting: Associative learning and cognition in animals* (pp. 343–382). American Psychological Association. https://doi.org/10.1037/10298-012

Bristol, A. S., & Carew, T. J. (2005). Differential role of inhibition in habituation of two independent afferent pathways to a common motor output. *Learning & Memory*, *12*(1), 52–60. https://doi.org/10.1101/lm.83405

Broster, B. S., & Rankin, C. H. (1994). Effects of changing interstimulus interval during habituation in *Caenorhabditis elegans*. *Behavioral Neuroscience*, *108*(6), 1019–1029. https://doi.org/10.1037/0735-7044.108.6.1019

Byrne, J. H., & Gingrich, K. J. (1989). Mathematical model of cellular and molecular processes contributing to associative and nonassociative learning in Aplysia. In J. H. Byrne & W. O. Berry (Eds.), *Neural models of plasticity* (pp. 58–72). Academic Press. https://doi.org/10.1016/B978-0-12-148955-7.50008-4

Carew, T. (1984). An introduction to cellular approaches used in the analysis of habituation and sensitization in Aplysia. In H. V. S. Peeke & L. Petrinovich (Eds.), *Habituation, sensitization, and behavior* (pp. 205–249). Academic Press. https://doi.org/10.1016/B978-0-12-549860-9.50013-4

Carlton, P. L. (1968). Brain acetylcholine and habituation. *Progress in Brain Research*, *28*, 48–60. https://doi.org/10.1016/S0079-6123(08)64542-2

Colwill, R. M., Lattal, K. M., Whitlow, J. W., Jr., & Delamater, A. R. (2023). Habituation: It's not what you think it is. *Behavioural Processes*, *207*, Article 104845. https://doi.org/10.1016/j.beproc.2023.104845

Davis, M. (1970). Effects of interstimulus interval length and variability on startle-response habituation in the rat. *Journal of Comparative and Physiological Psychology*, *72*(2), 177–192. https://doi.org/10.1037/h0029472

Davis, M. (1974). Sensitization of the rat startle response by noise. *Journal of Comparative and Physiological Psychology*, *87*(3), 571–581. https://doi.org/10.1037/h0036985

Davis, M., & File, S. E. (1984). Intrinsic and extrinsic mechanisms of habituation and sensitization: Implications for the design and analysis of experiments. In H. V. S. Peeke & L. Petrinovich (Eds.), *Habituation, sensitization and behavior* (pp. 287–323). Academic Press. https://doi.org/10.1016/B978-0-12-549860-9.50015-8

Davis, M., & Wagner, A. R. (1968). Startle responsiveness after habituation to different intensities of tone. *Psychonomic Science*, *12*(7), 337–338. https://doi.org/10.3758/BF03331339

Davis, M., & Wagner, A. R. (1969). Habituation of startle response under incremental sequence of stimulus intensities. *Journal of Comparative and Physiological Psychology*, *67*(4), 486–492. https://doi.org/10.1037/h0027308

de Oliveira Alvares, L., & Do-Monte, F. H. (2021). Understanding the dynamic and destiny of memories. *Neuroscience and Biobehavioral Reviews*, *125*, 592–607. https://doi.org/10.1016/j.neubiorev.2021.03.009

del Rosal, E., Alonso, L., Moreno, R., Vázquez, M., & Santacreu, J. (2006). Simulation of habituation to simple and multiple stimuli. *Behavioural Processes*, *73*(3), 272–277. https://doi.org/10.1016/j.beproc.2006.06.007

Dissegna, A., Turatto, M., & Chiandetti, C. (2021). Context-specific habituation. *Animals: An Open Access Journal From MDPI*, *11*(6), Article 1767. https://doi.org/10.3390/ani11061767

Donegan, N. H., & Wagner, A. R. (1987). Conditioned diminution and facilitation of the UR: A sometimes opponent-process interpretation. In I. Gormezano, W. F. Prokasy, & R. F. Thompson (Eds.), *Classical conditioning* (3rd ed., pp. 339–369). Lawrence Erlbaum Associates.

Gershman, S. J. (2024). *Habituation as optimal filtering*. https://doi.org/10.31234/osf.io/ystw2

Glanzer, M. (1953). Stimulus satiation: An explanation of spontaneous alternation and related phenomena. *Psychological Review*, *60*(4), 257–268. https://doi.org/10.1037/h0062718

Glanzman, D. L. (2009). Habituation in Aplysia: The Cheshire cat of neurobiology. *Neurobiology of Learning and Memory*, *92*(2), 147–154. https://doi.org/10.1016/j.nlm.2009.03.005

Gluck, M. A., & Thompson, R. F. (1987). Modeling the neural substrates of associative learning and memory: A computational approach. *Psychological Review*, *94*(2), 176–191. https://doi.org/10.1037/0033-295X.94.2.176

Groves, P. M., & Thompson, R. F. (1970). Habituation: A dual-process theory. *Psychological Review*, *77*(5), 419–450. https://doi.org/10.1037/h0029810

Hall, G., & Rodríguez, G. (2019). Attention to perceive, to learn and to respond. *The Quarterly Journal of Experimental Psychology*, *72*(2), 335–345. https://doi.org/10.1080/17470218.2017.1339719

Hargitt, C. W. (1906). Experiments on the behavior of tubicolous annelids. *The Journal of Experimental Zoology*, *3*(2), 295–320. https://doi.org/10.1002/jez.1400030204

Hawkins, R. D. (1989). A biologically based computational model for several simple forms of learning. *Psychology of Learning and Motivation*, *23*, 65–108. https://doi.org/10.1016/S0079-7421(08)60109-7

Hawkins, R. D., & Kandel, E. R. (1984). Is there a cell-biological alphabet for simple forms of learning? *Psychological Review*, *91*(3), 375–391. https://doi.org/10.1037/0033-295X.91.3.375

Hermitte, G., Pedreira, M. E., Tomsic, D., & Maldonado, H. (1999). Context shift and protein synthesis inhibition disrupt long-term habituation after spaced, but not massed, training in the crab Chasmagnathus. *Neurobiology of Learning and Memory*, *71*(1), 34–49. https://doi.org/10.1006/nlme.1998.3858

Hilgard, E. R., & Marquis, D. G. (1940). *Conditioning and learning*. Appleton-Century-Crofts.

Hintzman, D. L. (1974). Theoretical implications of the spacing effect. In R. L. Solso (Ed.), *Theories in cognitive psychology: The Loyola Symposium* (pp. 77–99). Lawrence Erlbaum Associates.

Hochner, B., Klein, M., Schacher, S., & Kandel, E. R. (1986). Additional component in the cellular mechanism of presynaptic facilitation contributes to behavioral dishabituation in Aplysia. *Proceedings of the National Academy of Sciences of the United States of America*, *83*(22), 8794–8798. https://doi.org/10.1073/pnas.83.22.8794

Hull, C. L. (1943). *Principles of behavior: An introduction to behavior theory*. Appleton-Century-Crofts.

Humphrey, G. (1933). *The nature of learning in its relation to the living system*. Routdledge.

Hunter, M. A., & Ames, E. W. (1988). A multifactor model of infant preferences for novel and familiar stimuli. In C. Rovee-Collier & L. P. Lipsitt (Eds.), *Advances in infancy research* (Vol. 5, pp. 69–95). Ablex Publishing.

Innis, N. K., & Staddon, J. E. R. (1989). What should comparative psychology compare? *International Journal of Comparative Psychology*, *2*(3), 145–156. https://doi.org/10.46867/C4F01X

Jackson, J. C. (1974). Amplitude and habituation of the orienting reflex as a function of stimulus intensity. *Psychophysiology*, *11*(6), 647–659. https://doi.org/10.1111/j.1469-8986.1974.tb01133.x

James, J. P., & Hughes, G. R. (1969). Generalization of habituation of the GSR to white noise of varying intensities. *Psychonomic Science*, *14*(4), 163–164. https://doi.org/10.3758/BF03332767

Jordan, W. P., Strasser, H. C., & McHale, L. (2000). Contextual control of long-term habituation in rats. *Journal of Experimental Psychology: Animal Behavior Processes*, *26*(3), 323–339. https://doi.org/10.1037/0097-7403.26.3.323

Kandel, E. (1976). *Cellular basis of behavior: An introduction to behavioral neurobiology*. W.H. Freeman.

Kandel, E. R. (1978). *A cell-biological approach to learning*. Society for Neuroscience.

Kepler, L. D., McDiarmid, T. A., & Rankin, C. H. (2020). Habituation in high-throughput genetic model organisms as a tool to investigate the mechanisms of neurodevelopmental disorders. *Neurobiology of Learning and Memory*, *171*, Article 107208. https://doi.org/10.1016/j.nlm.2020.107208

Konorski, J. (1967). Some new ideas concerning the physiological mechanisms of perception. *Acta Biologiae Experimentalis*, *27*(2), 147–161. https://rcin.org.pl/Content/4767/PDF/WA488_4260_P180-T27-z2_ABE.pdf

Lane, S. T., Franklin, J. C., & Curran, P. J. (2013). Clarifying the nature of startle habituation using latent curve modeling. *International Journal of Psychophysiology*, *88*(1), 55–63. https://doi.org/10.1016/j.ijpsycho.2013.01.010

LaRowe, S. D., Patrick, C. J., Curtin, J. J., & Kline, J. P. (2006). Personality correlates of startle habituation. *Biological Psychology*, *72*(3), 257–264. https://doi.org/10.1016/j.biopsycho.2005.11.008

Leaton, R. N. (1974). Long-term retention of the habituation of lick suppression in rats. *Journal of Comparative and Physiological Psychology*, *87*(6), 1157–1164. https://doi.org/10.1037/h0037596

Lozada, M., Romano, A., & Maldonado, H. (1990). Long-term habituation to a danger stimulus in the crab *Chasmagnathus granulatus*. *Physiology & Behavior*, *47*(1), 35–41. https://doi.org/10.1016/0031-9384(90)90039-7

MacDonald, B., Barry, R. J., & Bonfield, R. C. (2015). Trials and intensity effects in single-trial ERP components and autonomic responses in a dishabituation paradigm with very long ISIs. *International Journal of Psychophysiology*, *98*(3 Part 1), 394–412. https://doi.org/10.1016/j.ijpsycho.2015.08.002

Marcus, E. A., Nolen, T. G., Rankin, C. H., & Carew, T. J. (1988). Behavioral dissociation of dishabituation, sensitization, and inhibition in Aplysia. *Science*, *241*(4862), 210–213. https://doi.org/10.1126/science.3388032

Marks, L. E. (1974). On scales of sensation: Prolegomena to any future psychophysics that will be able to come forth as science. *Perception & Psychophysics*, *16*(2), 358–376. https://doi.org/10.3758/BF03203956

Marlin, N. A., & Miller, R. R. (1981). Associations to contextual stimuli as a determinant of long-term habituation. *Journal of Experimental Psychology: Animal Behavior Processes*, *7*(4), 313–333. https://doi.org/10.1037/0097-7403.7.4.313

Mazur, J. E., & Wagner, A. R. (1982). An episodic model of associative learning. In M. Commons, R. Herrnstein, & A. R. Wagner (Eds.), *Acquisition. Quantitative analyses of behavior* (pp. 3–39). Ballinger.

McDiarmid, T. A., Yu, A. J., & Rankin, C. H. (2019). Habituation is more than learning to ignore: Multiple mechanisms serve to facilitate shifts in behavioral strategy. *BioEssays*, *41*(9), Article e1900077. https://doi.org/10.1002/bies.201900077

McLaren, I. P. L., & Mackintosh, N. J. (2000). An elemental model of associative learning: I. Latent inhibition and perceptual learning. *Animal Learning & Behavior*, *28*(3), 211–246. https://doi.org/10.3758/BF03200258

Menzel, R. (2001). Searching for the memory trace in a mini-brain, the honeybee. *Learning & Memory*, *8*(2), 53–62. https://doi.org/10.1101/lm.38801

Moyer, K. E. (1963). Startle response: Habituation over trials and days, and sex and strain differences. *Journal of Comparative and Physiological Psychology*, *56*(5), 863–865. https://doi.org/10.1037/h0045861

O'Gorman, J. G., & Jamieson, R. D. (1975). The incremental stimulus intensity effect and habituation of autonomic responses in man. *Physiological Psychology*, *3*(4), 385–389. https://doi.org/10.3758/BF03326846

Peeke, H. V. S., & Petrinovich, L. (1984). *Habituation, sensitization, and behavior*. Academic Press.

Pfautz, P. L., Donegan, N. H., & Wagner, A. R. (1978). Sensory preconditioning versus protection from habituation. *Journal of Experimental Psychology: Animal Behavior Processes*, *4*(3), 286–295. https://doi.org/10.1037/0097-7403.4.3.286

Pinto, J. A., Becerra, S. A., Ponce, F. P., & Vogel, E. H. (2014). Especificidad contextual diferencial en la habituación de las respuestas de parpadeo y aceleración cardiaca en humanos [Differential context-specificity in the habituation of the eyeblink and cardiac responses in humans]. *Universitas Psychologica*, *13*(4), 1245–1254. https://doi.org/10.11144/Javeriana.UPSY13-4.ecdh

Plichta, M. M., Grimm, O., Morgen, K., Mier, D., Sauer, C., Haddad, L., Tost, H., Esslinger, C., Kirsch, P., Schwarz, A. J., & Meyer-Lindenberg, A. (2014). Amygdala habituation: A reliable fMRI phenotype. *NeuroImage*, *103*, 383–390. https://doi.org/10.1016/j.neuroimage.2014.09.059

Poli, F., Ghilardi, T., Beijers, R., de Weerth, C., Hinne, M., Mars, R. B., & Hunnius, S. (2024). Individual differences in processing speed and curiosity explain infant habituation and dishabituation performance. *Developmental Science*, *27*(3), Article e13460. https://doi.org/10.1111/desc.13460

Poon, C. S., & Young, D. L. (2006). Nonassociative learning as gated neural integrator and differentiator in stimulus–response pathways. *Behavioral and Brain Functions*, *2*(1), Article 29. https://doi.org/10.1186/1744-9081-2-29

Ramaswami, M. (2014). Network plasticity in adaptive filtering and behavioral habituation. *Neuron*, *82*(6), 1216–1229. https://doi.org/10.1016/j.neuron.2014.04.035

Randlett, O., Haesemeyer, M., Forkin, G., Shoenhard, H., Schier, A. F., Engert, F., & Granato, M. (2019). Distributed plasticity drives visual habituation learning in larval zebrafish. *Current Biology*, *29*(8), 1337–1345.e4. https://doi.org/10.1016/j.cub.2019.02.039

Rankin, C. H. (2000). Context conditioning in habituation in the nematode *Caenorhabditis elegans*. *Behavioral Neuroscience*, *114*(3), 496–505. https://doi.org/10.1037/0735-7044.114.3.496

Rankin, C. H., & Broster, B. S. (1992). Factors affecting habituation and recovery from habituation in the nematode *Caenorhabditis elegans*. *Behavioral Neuroscience*, *106*(2), 239–249. https://doi.org/10.1037/0735-7044.106.2.239

Rankin, C. H., & Carew, T. J. (1987). Development of learning and memory in Aplysia. II. Habituation and dishabituation. *The Journal of Neuroscience*, *7*(1), 133–143. https://doi.org/10.1523/JNEUROSCI.07-01-00133.1987

Ratner, S. C. (1970). Habituation: Research and theory. In J. H. Reynierse (Ed.), *Current issues in animal learning* (pp. 55–84). University of Nebraska Press.

Santos, A., Porto, A., Romero, J., Albó, A., & Pazos, A. (2007). Study of classical conditioning in Aplysia through the implementation of computational models of its learning circuit. *Journal of Experimental & Theoretical Artificial Intelligence*, *19*(2), 119–158. https://doi.org/10.1080/09528130601052177

Schöner, G., & Thelen, E. (2006). Using dynamic field theory to rethink infant habituation. *Psychological Review*, *113*(2), 273–299. https://doi.org/10.1037/0033-295X.113.2.273

Siddle, D. A. T., Kyriacou, C., & Heron, P. A. (1978) Effects of change in stimulus duration on amplitude of the electrodermal orienting response. *Psychobiology*, *6*, 121–125. https://doi.org/10.3758/BF03326705

Sirois, S., & Mareschal, D. (2002). Infant habituation: A review of current computational models and a new proposal. In J. Bullinaria (Ed.), *Proceedings of the seventh neural computation and psychology workshop (NCPW7)* (pp. 90–103). World Scientific Press.

Sokolov, E. N. (1963). *Perception and the condition reflex*. Pergamon.

Sokolov, E. N. (1969). Modeling properties of the nervous system. In I. Maltzman & N. Coile (Eds.), *Handbook of contemporary soviet psychology* (pp. 671–704). Basic books.

Soto, F. A. (2019). Beyond the "conceptual nervous system": Can computational cognitive neuroscience transform learning theory? *Behavioural Processes*, *167*, Article 103908. https://doi.org/10.1016/j.beproc.2019.103908

Spinks, J. A., & Siddle, D. A. (1976). Effects of stimulus information and stimulus duration on amplitude and habituation of the electrodermal orienting response. *Biological Psychology*, *4*(1), 29–39. https://doi.org/10.1016/0301-0511(76)90028-4

Staddon, J. (1993). On rate-sensitive habituation. *Adaptive Behavior*, *1*(4), 421–436. https://doi.org/10.1177/105971239300100402

Staddon, J. E. R. (2005). Interval timing: Memory, not a clock. *Trends in Cognitive Sciences*, *9*(7), 312–314. https://doi.org/10.1016/j.tics.2005.05.013

Staddon, J. E. R., Chelaru, I. M., & Higa, J. J. (2002). Habituation, memory and the brain: The dynamics of interval timing. *Behavioural Processes*, *57*(2–3), 71–88. https://doi.org/10.1016/S0376-6357(02)00006-2

Staddon, J. E. R., & Higa, J. J. (1996). Multiple time scales in simple habituation. *Psychological Review*, *103*(4), 720–733. https://doi.org/10.1037/0033-295X.103.4.720

Stanley, J. C. (1976). Computer simulation of a model of habituation. *Nature*, *261*(5556), 146–148. https://doi.org/10.1038/261146a0

Stein, L. (1966). Habituation and stimulus novelty: A model based on classical conditioning. *Psychological Review*, *73*(4), 352–356. https://doi.org/10.1037/h0023449

Steiner, G. Z., & Barry, R. J. (2014). The mechanism of dishabituation. *Frontiers in Integrative Neuroscience*, *8*, Article 14. https://doi.org/10.3389/fnint.2014.00014

Thomas, H., & Gilmore, R. O. (2004). Habituation assessment in infancy. *Psychological Methods*, *9*(1), 70–92. https://doi.org/10.1037/1082-989X.9.1.70

Thompson, R. F. (2009). Habituation: A history. *Neurobiology of Learning and Memory*, *92*(2), 127–134. https://doi.org/10.1016/j.nlm.2008.07.011

Thompson, R. F., & Spencer, W. A. (1966). Habituation: A model phenomenon for the study of neuronal substrates of behavior. *Psychological Review*, *73*(1), 16–43. https://doi.org/10.1037/h0022681

Tomsic, D., Pedreira, M. E., Romano, A., Hermitte, G., & Maldonado, H. (1998). Context-US association as a determinant of long-term habituation in the crab Chasmagnathus. *Animal Learning & Behavior*, *26*(2), 196–209. https://doi.org/10.3758/BF03199212

Uribe-Bahamonde, Y. E., Becerra, S. A., Ponce, F. P., & Vogel, E. H. (2019). A quantitative account of the behavioral characteristics of habituation: The sometimes opponent processes model of stimulus processing. *Frontiers in Psychology*, *10*, Article 504. https://doi.org/10.3389/fpsyg.2019.00504

Uribe-Bahamonde, Y. E., Jorquera, O. E., Becerra, S. A., & Vogel, E. H. (2021). SOP- habituation laboratory: An interactive tool for simulating the basic behavioral features of habituation. *Behavior Research Methods*, *53*(5), 2120–2126. https://doi.org/10.3758/s13428-021-01548-y

Uusitalo, M. A., Williamson, S. J., & Seppä, M. T. (1996). Dynamical organisation of the human visual system revealed by lifetimes of activation

traces. *Neuroscience Letters*, *213*(3), 149–152. https://doi.org/10.1016/0304-3940(96)12846-9

Vogel, E. H., Ponce, F. P., & Brandon, S. E. (2020). Can the stimulus processing assumptions of the sometimes-opponent-process (SOP) model explain instances of contextual learning? *Journal of Experimental Psychology: Animal Learning and Cognition*, *46*(3), 205–214. https://doi.org/10.1037/xan0000251

Vogel, E. H., Ponce, F. P., & Wagner, A. R. (2019). The development and present status of the SOP model of associative learning. *The Quarterly Journal of Experimental Psychology*, *72*(2), 346–374. https://doi.org/10.1177/1747021818777074

Wagner, A., & Vogel, E. (2010). Associative modulation of US processing: Implications for understanding of habituation. In N. Schmajuk (Ed.), *Computational models of conditioning* (pp. 150–185). Cambridge University Press. https://doi.org/10.1017/CBO9780511760402

Wagner, A. R. (1976). Priming in STM: An information-processing mechanism for self-generated or retrieval-generated depression in performance. In T. J. Tighe & R. N. Leaton (Eds.), *Habituation: Perspectives from child development, animal behavior and neurophysiology* (pp. 95–128). Erlbaum.

Wagner, A. R. (1978). Expectancies and the priming of STM. In S. H. Hulse, H. Fowler, & W. K. Honig (Eds.), *Cognitive processes in animal behavior* (pp. 177–209). Erlbaum.

Wagner, A. R. (1979). Habituation and memory. In A. Dickinson & R. A. Boakes (Eds.), *Mechanisms of learning and motivation: A memorial volume for Jerzy Konorski* (pp. 53–82). Erlbaum Associates.

Wagner, A. R. (1981). SOP: A model of automatic memory processing in animal behavior. In N. E. Spear & R. R. Miller (Eds.), *Information processing in animals: Memory mechanisms* (pp. 5–48). Erlbaum.

Wagner, A. R., & Brandon, S. E. (1989). Evolution of a structured connectionist model of Pavlovian conditioning (AESOP). In S. B. Klein & R. R. Mowrer (Eds.), *Contemporary learning theories: Pavlovian conditioning and the status of traditional learning theory* (pp. 149–189). Lawrence Erlbaum Associates.

Wang, D. (1993). A neural model of synaptic plasticity underlying short-term and long-term habituation. *Adaptive Behavior*, *2*(2), 111–129. https://doi.org/10.1177/105971239300200201

Wang, D., & Arbib, M. A. (1992). Modeling the dishabituation hierarchy: The role of the primordial hippocampus. *Biological Cybernetics*, *67*(6), 535–544. https://doi.org/10.1007/BF00198760

Whitlow, J. W. (1975). Short-term memory in habituation and dishabituation. *Journal of Experimental Psychology: Animal Behavior Processes*, *1*(3), 189–206. https://doi.org/10.1037/0097-7403.1.3.189

Whitlow, J. W., & Wagner, A. R. (1984). Memory and habituation. In H. V. S. Peeke & L. Petrinovich (Eds.), *Habituation, sensitization and behavior* (pp. 103–153). Academic Press. https://doi.org/10.1016/B978-0-12-549860-9.50010-9

Yerkes, A. W. (1906). Modifiability of behavior in Hydroides dianthus V. *Journal of Comparative Neurology & Psychology*, *16*(6), 441–449. https://doi.org/10.1002/cne.920160604

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