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Interpreting Patterns of Brain Activation in Human Fear Conditioning With an Attentional–Associative Learning Model

Joseph Dunsmoor and Nestor Schmajuk
Duke University

J. E. Dunsmoor, P. A. Bandettini, and D. C. Knight (2007) conducted a neuroimaging study of human fear conditioning and analyzed brain activity under various pairing rates between a conditioned and an unconditioned stimulus. Computer simulations with an attentional-associative model introduced by N. A. Schmajuk, Y. W. Lam, and J. A. Gray (1996) show that activity in the amygdala and anterior cingulate cortex is well described by a variable representing the prediction of the unconditioned stimulus, whereas activity in the dorsolateral prefrontal cortex and insula is well captured by a variable coding the attentional-modulated representation of conditioned stimuli. In addition, the model explains how those variables control behavior and provides a clear framework in which those variables play important roles in the description of numerous classical conditioning paradigms. Also, the model offers a number of predictions related to stimulus novelty for future neuroimaging studies of associative learning.

Keywords: brain, activity, models, reinforcement, novelty

In a recent functional magnetic resonance imaging (fMRI) study by Dunsmoor, Bandettini, and Knight (2007), brain activity was examined during fear learning in humans to three distinct conditioned stimuli (CSs) that coterminated with an aversive unconditioned stimulus (US) on 100%, 50%, or 0% of the trials. A linear pattern of brain activity that increased with the CS–US pairing rate was observed in regions supporting the acquisition and expression of learned fear, such as the amygdala and anterior cingulate cortex. A separate pattern of activity was observed within the dorsolateral prefrontal cortex (dlPFC) and insula; the CS that was intermittently paired with the US evoked the greatest response in these brain regions. Dunsmoor et al. suggested that these separate patterns of neural activity reflected distinct processes involved in Pavlovian fear conditioning. For instance, although the amygdala and anterior cingulate appeared to code for the strength of the CS–US association, the dlPFC and insula responded to the uncertainty for receiving the US.

In this article, we show that variables representing neural activity in a model of classical conditioning presented by Schmajuk, Lam, and Gray (1996) are consistent with the patterns of brain activity reported by Dunsmoor et al. (2007). Computer simulations with the Schmajuk–Lam–Gray (SLG) model demonstrate that activity in the amygdala and anterior cingulate cortex is well described by a variable representing the prediction of the US, whereas activity in the dlPFC and insula can be characterized by a variable coding the attentional-modulated representation of the CS. The results suggest that using a model that incorporates cognitive mechanisms involved in classical conditioning may help illuminate the neural substrates of human associative learning.

The SLG Model

Schmajuk et al. (1996; Schmajuk & Larrauri, 2006; Larrauri & Schmajuk, 2008) introduced a neural network theory of classical conditioning that describes many features that characterize classical conditioning. Figure 1 shows a simplified diagram of the model that illustrates the different stages, or nodes, involved in the generation of a conditioned response (CR) when a given CS is presented.

The output of Node 1 is proportional to $\tau_{CS} + B_{CS}$, where τ_{CS} is a short-term memory trace of the CS and B_{CS} is the prediction of the CS by other stimuli (the context [CX], other CSs, or the CS itself) active at a given time.

The triangle connecting Node 1 to Node 2 represents a synaptic weight proportional to the positive value of attention, z_{CS}. The value of z_{CS} is computed as the association between the output of Node 1 with the value of Novelty′. That is, changes in z_{CS} given by

$$dz_{CS} \sim (\tau_{CS} + B_{CS})\,[\text{Novelty}'\,(1 - z_{CS}) - (1 + z_{CS})], \quad (1)$$

where Novelty′ is proportional to the sum of the novelties of all stimuli present or predicted at a given time. The novelty of a CS, CX, or US is given by the absolute value of the difference between the average observed value of the CS, CX, or US and the average of their corresponding predictions. By Equation 1, z_{CS} increases to 1 when Novelty′ is relatively large and decreases to −1 otherwise.

The output of Node 2 is the attention-modulated representation of the CS, X_{CS}.

$$X_{CS} \sim z_{CS}\,(\tau_{CS} + B_{CS}), \quad (2)$$

where z_{CS} assumes only positive values. The triangle connecting Node 2 to Node 3 represents a synaptic weight proportional to the excitatory or inhibitory association, $V_{CS,\,US}$, between X_{CS} and the

Joseph Dunsmoor and Nestor Schmajuk, Department of Psychology and Neuroscience, Duke University.

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Correspondence concerning this article should be addressed to Nestor Schmajuk, Department of Psychology and Neuroscience, Duke University, Durham, NC 27708. E-mail: nestor@duke.edu

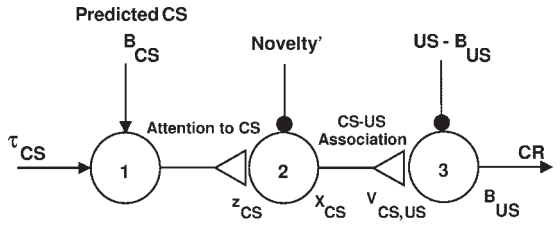

Figure 1. Simplified circuit of the Schmajuk, Lam, and Gray (1996) model. Triangles represent variable connections (associations) between nodes that modulate the activation of the node; arrows represent inputs that control the output of the node; and solid circles represent inputs that modify connections z_{CS} and $V_{CS,\,US}$ without affecting outputs X_{CS} and CR. CS = conditioned stimuli; US = unconditioned stimulus; τ_{CS} = trace of CS; B_{CS} = predicted CS; z_{CS} = attention to CS; X_{CS} = internal representation of the CS; $V_{CS,\,US}$ = X_{CS}–US association; B_{US} = predicted US; CR = conditioned response; Novelty′ = detected novelty.

US. Changes in the association between the CS (or the CX) and the US, V_{CS}, are given by

$$dV_{CS} = K\,X_{CS}\,(\lambda_{US} - B_{US})\,(1 - |V_{CS}|), \tag{3}$$

where K determines the learning rate, X_{CS} is the internal representation of the CS, λ_{US} is the intensity of the US, B_{US} is the aggregate prediction of the US by all Xs active at a given time, $\lambda_{US} - B_{US}$ is the error term, and the term $1 - |V_{CS}|$ constrains V_{CS}, $-1 < V_{CS} < 1$. Associations of a CS and the CX with the US increase when the $\lambda_{US} - B_{US}$ term is positive and decrease when it is negative. Although not shown in Figure 1, we assume that a similar error term, $\lambda_{CS} - B_{CS}$, controls the formation of associations between X_{CS} and other CSs (between-CS associations).

The output of Node 3 is the aggregate prediction of the US by all CSs with representations active at a given time, B_{US}, which is given by

$$B_{US} = \Sigma\, X_{CS} \times V_{CS}, \tag{4}$$

where V_{CS} is the association of X_{CS} with the US. Figure 1 shows only one CS activating $V_{CS,\,US}$. B_{US} is used to compute dV_{CS} in Equation 3 and determines the magnitude of the CR through a sigmoid function, CR = f(B_{US}).

More important, because by Equation 3 the rate of change of every association is directly proportional to X_{CS}, X_{CS} controls learning of the associations. Because by Equation 4 B_{US} is proportional to the product $X_{CS} \times V_{CS,US}$, X_{CS} also controls the retrieval of those associations. Because attentional memory z_{CS} controls the magnitude of the internal representation X_{CS}, attention controls learning and retrieval of CS–CS and CS–US associations. Because B_{US} controls the strength of the CR, attentional memory z_{CS} also controls performance.

In the simulations that follow, we show that (a) activity in the amygdala and anterior cingulate is well characterized by the prediction of the US by the CS and the CX, B_{US}; (b) activity in the dlPFC and anterior insula is well described by the representation of the CS, $X_{CS,}$ and (c) the skin conductance response (SCR) is a nonlinear function of B_{US}. It is important to notice that the variables B_{US} and X_{CS} represent neural activities (see Figure 1) and not the strength of their related synaptic associations, $V_{CS,\,US}$ and z_{CS}, which cannot be appreciated by fMRI methods.

Results

Experimental data. Dunsmoor et al. (2007) demonstrated patterns of learning-related activity within several brain regions in a fear-conditioning task that varied the CS–US pairing rate. While in fMRI, participants were presented with three auditory CSs of 10 s each, which coterminated with a 500-ms 100-dB aversive white noise on 100% (CS100), 50% (CS50), or 0% (CS−) of trials. The conditioning session included 40 trials of each CS (120 total). Fear conditioning was evaluated by the SCR and ratings of expectancy for receiving the US.

Dunsmoor et al. (2007) reported two distinct patterns of brain activity to CSs that varied as a function of the CS–US pairing rate. The magnitude of activity in the amygdala and anterior cingulate cortex was greatest to a CS that coterminated with the US on 100% of trials (CS100), whereas activity to a partially paired CS (CS50) fell at an intermediate level between the CS100 and an unpaired control stimulus (CS−; see Figure 2, top panel). Activity observed within these regions was suggested by Dunsmoor et al. as reflecting the strength of the CS–US association. A separate pattern of

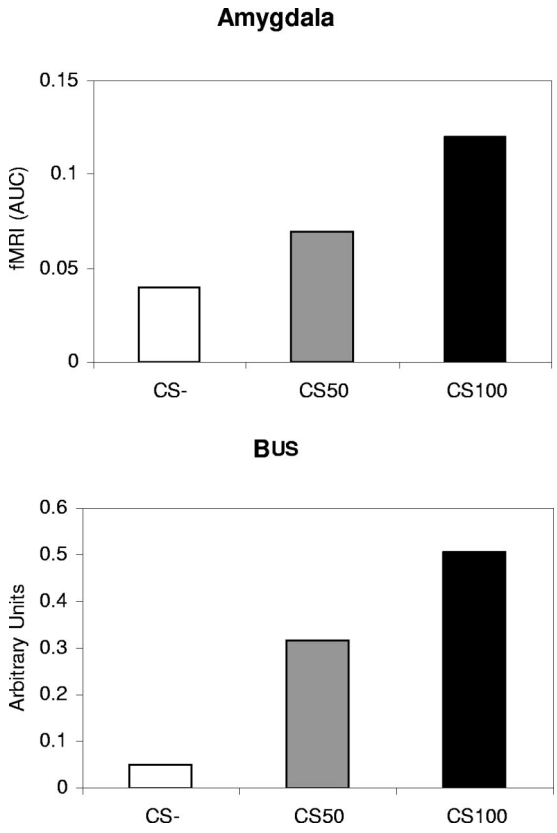

Figure 2. Hemodynamic response in the amygdala. Top: Percent area under the hemodynamic response curve (AUC) within the bilateral amygdala. Data from Dunsmoor et al. (2007). Bottom: Computer simulations of the B_{US} variable for CS−, CS50, and CS100, with the Schmajuk, Lam, and Gray (1996) model. B_{US} = predicted US; CS− = conditioned stimulus (CS) that coterminated with an aversive unconditioned stimulus (US) on 0% of the trials; CS50 = CS that coterminated with an aversive US on 50% of the trials; CS100 = CS that coterminated with an aversive US on 100% of the trials; fMRI = functional MRI.

activity was observed within the dlPFC and insula. The magnitude of activity within these regions was greatest to the partially paired CS50 than to either the CS100 or the CS−. Heightened activity to the CS50 within the dlPFC and insula was suggested as reflecting the uncertainty for receiving the US (see Figure 3, top panel). Finally, Dunsmoor et al. reported that the magnitude of the SCR was proportional to the probability of reinforcement (see Figure 4, top panel).

Simulation. Computer simulations with the SLG model consisted of presentations of three different CSs paired with the US on 0% (CS−), 50% (CS50), or 100% of the trials (CS100). The CS salience was 1, and CSs were 20 time units in duration; the US had strength 2 and overlapped with the last five time units of the CSs; the context salience was 0.1. Generalization between CSs was achieved by including an additional CS, CSg, with salience 0.1, representing elements common to all CSs. The duration of the intertrial interval was set to 3,000 time units to ensure that the traces of the different CSs became associated with their corresponding level of Novelty′, independent of the Novelty′ corresponding to other CSs. The model received 64 CS−, 64 CS100, and 64 CS50 alternated trials. Parameter values used in all the simulations were identical to those used in previous articles (Larrauri & Schmajuk, 2008; Schmajuk & Larrauri, 2006). As mentioned, we hypothesized that (a) activity in the amygdala and anterior cingulate can be characterized by the prediction of the US by the CS and the CX, B_{US}; (b) activity in the dlPFC and insula can be described by the representation of the CS, X_{CS}; and (c) the SCR is a nonlinear function of B_{US}.

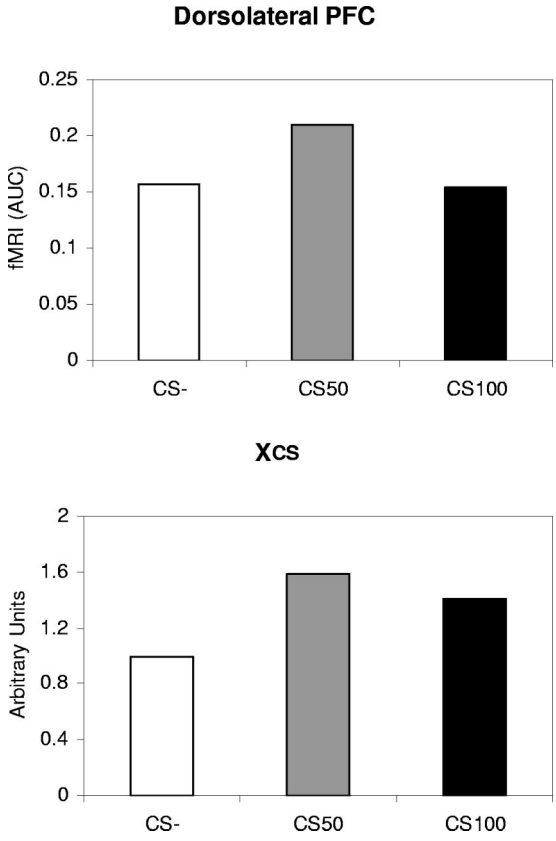

Figure 3. Hemodynamic response in the dorsolateral prefrontal cortex. Top: Percent area under the hemodynamic response curve (AUC) within the dorsolateral prefrontal cortex. Data from Dunsmoor et al. (2007). Bottom: Computer simulations of the X_{CS} variable for CS−, CS50, and CS100, with the Schmajuk, Lam, and Gray (1996) model. B_{US} = predicted US; CS− = conditioned stimulus (CS) that coterminated with an aversive unconditioned stimulus (US) on 0% of the trials; CS50 = CS that coterminated with an aversive US on 50% of the trials; CS100 = CS that coterminated with an aversive US on 100% of the trials; fMRI = functional MRI.

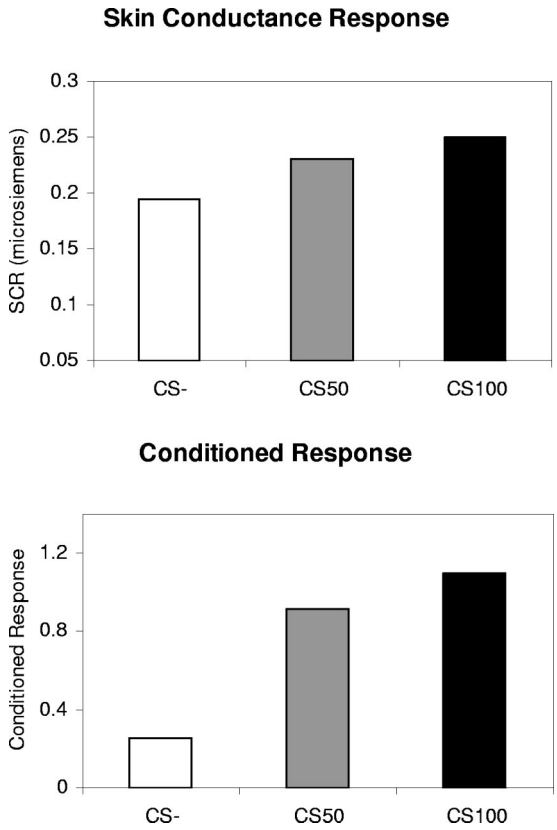

Figure 4. Conditioned skin conductance response (SCR). Top: Data from Dunsmoor et al. (2007). Bottom: Computer simulations of the conditioned response for CS−, CS50, and CS100, with the Schmajuk, Lam, and Gray (1996) model. CS− = conditioned stimulus (CS) that coterminated with an aversive unconditioned stimulus (US) on 0% of the trials; CS50 = CS that coterminated with an aversive US on 50% of the trials; CS100 = CS that coterminated with an aversive US on 100% of the trials.

Computer simulations using the SLG model show that the prediction for the US, B_{US}, increases linearly with the CS–US pairing rate, such that the continuously paired CS elicits the strongest association; the partially paired CS, an intermediate amount of association; and the unpaired control stimulus, the lowest amount of association (Figure 2, bottom panel). These results derive from the process of acquisition to CSs of variable CS–US reinforcement. That is, acquisition to a continuously paired CS increases in associative strength on each paired trial, whereas a partially paired CS alternates between gaining and losing associative strength over the course of paired and unpaired trials. Notably, although the CS− received no reinforcement with the US, B_{US} is still greater than zero for this stimulus because CSg and

the CX provide excitatory associations with the US. Overall, the pattern of responses revealed by the simulation is in line with those obtained from the amygdala and anterior cingulate cortex (Dunsmoor et al., 2007).

In addition, the internal representation of each CS (X_{CS}) is proportional to the level of Novelty′ detected at the time the CS trace is active (Figure 3, bottom panel). For instance, Novelty′ is minimal when the CS predicts that the US will be present on either 100% of the trials or 0% of the trials, and the US is consistently either present or absent (the participant is never surprised). In contrast, Novelty′ is maximal when the CS predicts that the US will be present on 50% of the trials, and the US is alternatively present or absent (the participant is always surprised). This pattern of simulated results seems to be consistent with the pattern of brain activity obtained from the dlPFC and insula (Dunsmoor et al., 2007).

Finally, Figure 4 (bottom panel) demonstrates a pattern of simulated CRs in line with the SCRs and US expectancy exhibited by human participants during fear conditioning. Because the CR in the present model is a nonlinear function of B_{US}, CR = f(B_{US}), where f represents a sigmoid function, the CRs obtained by the model show a pattern similar to that of B_{US} (see Figure 2).

Discussion

We showed that variables in a model of classical conditioning are capable of describing (a) activity in the amygdala and anterior cingulate cortex, (b) activity in the dlPFC and insula, and (c) the SCR during fear conditioning in humans (Dunsmoor et al., 2007).

The amygdala is an important area for conditioned fear learning and is consistently implicated in (a) forming the CS–US association (LeDoux, 2000) and (b) producing the CR (Knight et al., 2005). The simulations shown in Figure 2 appear to capture both these functions of the amygdala: (a) predicting the US, B_{US}, based on X_{CS}–US associations, $V_{CS, US}$, and (b) using this prediction to generate the CR. Likewise, the anterior cingulate has been shown to respond more to paired CSs than to unpaired CSs in brain imaging studies of classical fear conditioning (Buchel, Morris, Dolan, & Friston, 1998). Reciprocal connections between the amygdala and the anterior cingulate may facilitate heightened responses to stimuli associated with an aversive outcome (Devinsky, Morrell, & Vogt, 1995). However, the overall pattern of simulated results is not exclusive to the SLG model because most associative learning models (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981) predict that differential reinforcement schedules affect the associative value of a CS.

In contrast to the responses in the amygdala and anterior cingulate, Dunsmoor et al. (2007) characterized the pattern of responses in the dlPFC and insula as reflecting uncertainty for receiving the US because activity was greater to the partially paired CS50 than to the CSs with more predictable outcomes (CS− and CS100). The simulations, shown in Figure 3, support this suggestion. Specifically, the internal representation, X_{CS}, and attention, z_{CS}, were greatest for the CS partially paired with the US. These results are also in line with the Pearce–Hall model (1980) of associative learning, which predicts that attention is greater to CSs with more uncertain outcomes.

Uniquely in the SLG model, X_{CS} is proportional to the short-term memory trace of the CS, τ_{CS}, modulated by the magnitude of attention z_{CS}. Therefore, X_{CS} is an attention-modulated, sustained activity that is closely related to a working memory process. This aspect of the model converges with previous findings that the dlPFC is involved in holding the representation of a stimulus in working memory (D'Esposito, Postle, & Rypma, 2000; Fuster, 1973).

The simulation shown in Figure 4 appears to capture the physiological SCR data reported by Dunsmoor et al. (2007). Notice that although the internal representation of the CS, X_{CS}, is reduced for the 100% reinforced CS (Figure 3, bottom panel), both the B_{US} (Figure 2, bottom panel) and the CR (Figure 4, bottom panel) are strongest to the most predictive CS. According to the SLG model, as X_{CS} keeps decreasing with an increased number of 100% reinforced trials, the CR will decrease with extended training. Such a decreased responding has been reported during classical conditioning in animals (Pavlov, 1927; Sherman & Maier, 1978).

Finally, projections from the ventral tegmental area to the amygdala and the prefrontal cortex would provide Novelty′ information to these areas to control, respectively, the formation of X_{CS}–US associations and the activation of working memory, X_{CS}. Gray, Buhusi, and Schmajuk (1997) suggested that the activity of dopaminergic cells in the ventral tegmental area represent Novelty′ as defined in the SLG model, an assumption in line with reports that dopamine codes for novel stimuli (Fiorillo, Tobler, & Schultz, 2003; Horvitz, 2000; Legault & Wise, 2001; Williams, Rolls, Leonard, & Stern, 1993). This brain circuitry would provide a substrate for the functional characterization of the amygdala and anterior cingulate in terms of B_{US} and of the dlPFC and insula in terms of X_{CS}.

Although the activity in the amygdala and anterior cingulate cortex can be described by most associative learning models (e.g., Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981), that is not the case with the activity in the dlPFC and insula. The Rescorla and Wagner (1972) rule does not include a variable that can be correlated with the uncertainty of receiving the US, and the attentional variable in Mackintosh's (1975) theory is proportional to the quality of the CS as a predictor of the US and, therefore, predicts that the activity in the dlPFC and insula will show a maximum with 100% reinforcement (CS100). Under the assumption that activity in the dlPFC and insula is proportional to activity generated by the CS in the A2 state, Wagner's (1981) standard operating procedures model expects similar responses to CS−, CS50, and CS100 because in all cases priming of the CS is based on identical CX–CS associations. As mentioned, another model able to describe activity in the dlPFC and insula is the Pearce–Hall (1980) model. However, the SLG model offers advantages over this competitor when addressing the Dunsmoor et al. (2007) data because it relates the temporal course of X_{CS} to previous findings showing that the dlPFC holds the representation of a stimulus in working memory.

More important, the SLG model offers numerous advantages over the Pearce–Hall (1980) model when applied to other data, such as the disruption of latent inhibition by the presentation of an unexpected CS or the omission of an expected CS, the restoration of the orienting response by the omission of an expected CS, motivational effects on latent inhibition, recovery from latent inhibition by extinction of the context, recovery from blocking by

extinction of the blocker CS, backward blocking, spontaneous recovery from backward blocking, recovery from overshadowing by extinction of the overshadowing CS, and spontaneous recovery following extinction (see Schmajuk, 2009, for a review). Therefore, the SLG model, but not the Pearce–Hall model, predicts that blood oxygen level–dependent activity in the dlPFC and insula, proportional to X_{CS}, will increase when Novelty (or uncertainty) increases. That would be the case, for example, when extinction of the blocker follows blocking or extinction of the overshadowing CS follows overshadowing. These predictions are consistent with the fact that the amplitude of the simulated CR, proportional to $X_{CS} \times V_{CS,\,US}$, matches the amplitude of the data CR (SCR) well.

In addition, neurophysiological evidence seems to support the existence of error-correcting mechanisms that together with the attentional mechanisms mentioned earlier, are incorporated into the SLG model but not the Pearce–Hall model. Schultz and Dickinson (2000) reviewed studies showing how neurons within several brain structures appear to code prediction errors in relation to positive and negative reinforcement, CSs, and responses. Schultz and Dickinson indicated that sometimes dopamine, norepinephrine, and nucleus basalis neurons broadcast these error signals to different brain structures, but other times these error signals are coded and broadcast within certain structures (e.g., cerebellum).

Conclusion

In this article, we show that variables representing neural activity in an attentional–associative model of classical conditioning presented by Schmajuk et al. (1996) correspond to the blood oxygen level–dependent activity reported by Dunsmoor et al. (2007) in different areas of the brain during human fear conditioning. Dunsmoor et al. showed that although the amygdala and anterior cingulate appear to mediate the fear response to CSs that reliably predict the US, lateral frontal regions as well as the insula appear to have a greater role in responding to CSs with uncertain outcomes. This report adds to these findings. The use of a real-time neural network model allows for an interpretation of the activity in those brain regions, in terms of attention X_{CS} and associations $V_{CS,\,US}$, and an understanding of how those variables multiplicatively combine to control behavior, $CR = f(X_{CS} \times V_{CS,\,US})$. On the basis of this equation, the model provides a number of predictions for blood oxygen level–dependent activity in other associative tasks. Moreover, the model provides a clear framework in which these variables play important roles in the description of numerous paradigms.

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