

Figure 2-Figure supplement 1

Model predictions of LO lesions as a partial loss of outcome expectancy. (A) The model predictions for simple Pavlovian acquisition using a standard Rescorla-Wagner learning rule, and following modelled lesion deficits as a partial loss of outcome expectancy information specific to learning. Using the following update rule: , the change in associative strength (V) was calculated on a trial by trial basis for the CS and the context (preCS) periods. Parameters used were = .05 for the context and .2 for the CS, = 1, and γ = 0.8 for lesions and γ = 1.0 for the control condition. **(B)** Modelled performance following pre-training sham or LO lesions. **(C)** Modelled performance following temporary LO inactivation via muscimol after initial training.

One potential account of the heightened responding observed during simple acquisition following LO lesions is enhanced cue-outcome learning. However, given the hypothesised role of the OFC in the representation of outcome expectancy value (Baxter et al., 2000; Pears, Parkinson, Hopewell, Everitt, & Roberts, 2003; Schoenbaum et al., 1999; Schoenbaum & Roesch, 2005; Takahashi, Roesch, Stalnaker, Haney, Calu, et al., 2009), it is not immediately clear how this could be the case. The Rescorla-Wagner model of the acquisition of associative value suggests that in the absence of a representation of the expected value of an outcome, no conditioned responses should be expressed. In fact, in most associative learning theories the rules that govern performance are some function of the expected value of the outcome (LePelley, 2004; Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Bouton, 2002; Rescorla & Wagner, 1972). However, OFC damage may not abolish all outcome expectancy information but instead degrades some aspect (e.g. sensory specific properties) of the outcome expectancy information made available at the time of calculating the prediction error (e.g. mid brain Dopamine neurons; Takahashi et al., 2011). This would assume that the expected value used in prediction error learning may not necessarily be the same as the full expected value used to govern performance. To model this mathematically, a constant (γ) is used to represent the proportion of the outcome expectancy information that was available for learning (Figure 2-Figure supplement 1A). The strength/value (V) of the learned association changes in the following manner:

Such that is the change in associative strength on a given trial; are learning rate parameters based on the properties of the cue and outcome respectively such that and ; is the experienced value of the outcome on a given trial; is the sum of the associative strength i.e. the expected value. The constant γ represents the proportion of available for learning such that . In a healthy control animal 100% (γ = 1) of the available outcome expectancy information is available to guide learning. Consequently, further learning will stop once as the current expected value of the outcome fully accounts for the actual value of the outcome. Following OFC lesions some of this information is lost and, for example, 80% (γ = 0.8) of the available outcome expectancy information is available to guide learning. Therefore, learning will continue beyond the point at which and will only stop once . Therefore, the asymptote of conditioned behaviour (expressed as some function of ) will be determined by , therefore asymptotic conditioned behaviour will be higher following a lesion in this model ( than an intact OFC (.

The following simulation of this model (with the lesion parameter set at γ = 0.8) reveals a pattern of acquisition that is similar at the start of acquisition but diverges considerably towards the end of acquisition. This pattern strongly resembles the findings in Figure 1B with a range of values of γ > 0.6. This provides prima facie evidence of how well the model accounts for the present data, however the most valid test of the model would be an experimental test of a hypothesis generated from the model. This model predicts that if acquisition initially occurs with a functional LO until asymptote (i.e. ) and no more learning is evident, inactivation of OFC should allow learning to temporarily increase above asymptote because . Informally, the inactivation of LO will cause the outcome to no longer be fully predicted by the CS because only part of this information is being used. Similarly, if the function of LO is returned, then any new learning will extinguish until again. This prediction is modelled in (**C**). It is noteworthy that modelling the effect of LO damage in this way produces identical predictions in a number of other associative learning models (Esber & Haselgrove, 2011; Mackintosh, 1975; Pearce & Hall, 1980; Rescorla & Wagner, 1972) and therefore may also affect other aspects of learning such as attention. Furthermore, even though the hypothesis was generated using the current mathematical model, the prediction and underlying mechanism would be the same for any interpretation of the increased responding in Figure 1B as increased cue-outcome learning. If OFC damage increases learning, then transient inactivation of OFC should temporarily allow learning to increase. Therefore, the following experiment explicitly tested this model.