

# Neural Affective Decision Theory

## Reimplementing the ANDREA model

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**Abstract**—This work presents a reimplementation of a biologically realistic neural model of human emotional reward processing and decision making. It aims to provide a better understanding of how low and high level processes in the brain contribute to human preferences and behaviour. Five brain areas and their interactions are modelled: amygdala, orbitofrontal cortex, dopaminergic neurons, serotonin in dorsal raphe neurons, and ventral striatum. Design decisions of the reimplementation are based on the original model, or determined by testing reasonable estimates. The activity of the current model matches that of the original model well, though it has considerably more noise, and the emotional responses match the curve of loss aversion predicted by prospect theory. This reimplementation will make it easier for researchers to access and build on the model, thus improving our understanding of the neural processes underlying human decision making.

**Keywords**—decision making; neural; emotion; loss aversion

### I. INTRODUCTION

Human reward processing happens on at least two levels, with a low level ‘automatic’ process and a high level ‘cognitive’ process. There have been many names for these two levels of human thought, including Kahneman’s System 1 and System 2 [1] and Damasio’s primary and secondary emotions [2]. The current model attempts to provide a possible neural explanation of how these low and high level systems work together to form a person’s preferences and behaviour.

To understand better how emotion plays a part in human preferences and decision making, [3-5] created a model of neural affective decision theory. Neural affective decision theory has four principles: *affect*, the importance of emotions to evaluating possible actions; *brain*, the importance of neural interactions; *valuation*, using the opponency of serotonin and dopamine to determine preferences; and *framing*, the importance of context [5]. The model was called ANDREA, which stands for “Affective Neuroscience of Decision through Reward-based Evaluation of Alternatives” [3].

In ANDREA, valuation is based on the opponent processes of serotonin and dopamine. Serotonin (5-hydroxytryptamine, 5HT<sup>1</sup>) and dopamine have well-studied interactions, and drugs that treat many neuropsychiatric disorders often influence the

transmission of these neurotransmitters [6]. Their opponency was discussed in [7], where it was argued that dopamine and serotonin are opponent processes that together explain aversive and appetitive conditioning. The dopamine system is well known to be related to reward detection [8] and decision making [9], and is often characterized with the temporal difference (TD) model of reinforcement learning [5, 7, 10]. However, dopamine alone is not sufficient to explain some theoretical issues, so an opposite signal is needed, which [7] suggested is provided by serotonin. The original ANDREA model made use of interacting dopamine and serotonin systems, with dopamine representing positive prediction error, and serotonin representing negative prediction error [5].

The amygdala (AMYG) is a low-level brain structure with connections to many other parts of the brain, both low and high level [11]. It is commonly associated with attention, fear, and fight-or-flight responses [11]. In the original ANDREA model, the amygdala represented the overall emotional arousal level, incorporating positive and negative reward, the cost of dealing with conflict, and arousal unrelated to reward [5].

The orbitofrontal cortex (OFC) has not been the focus of as much study as other brain regions [12], but it has been found to be involved in emotion-related learning, by keeping track of stimulus-reinforcement associations and updating them when the associated reward value of a stimulus changes [13]. In the original model, OFC used the overall arousal level output of the amygdala to modulate an initial valuation of the stimulus that came from sensory or other frontal cortex areas [5]. Thus, it represented the subjective, or modulated, valuation of the stimulus; a high-level cognitive valuation.

The ventral striatum (VS) has been found to be involved in classical conditioning [11], and processing reward prediction errors [14]. In the original model, the ventral striatum was used to represent an error (or learning) signal, that was made up of the addition of the dopamine and serotonin systems’ outputs [5].

These five brain areas and their representations have been combined to create the basic structure of the ANDREA model. The original model was demonstrated to be consistent with the behavioural predictions of prospect theory [15] and decision affect theory [16], while also proposing specific neural

<sup>1</sup> This is usually abbreviated as 5-HT; however, in this paper the hyphen is omitted to improve the readability of equations.

explanations of these behaviours [5]. Decision affect theory looks at decision making when options are risky, and predicts that people do not make decisions based on the utility of the options, but rather on the emotion that each option produces [16].

Prospect theory examines loss aversion in risky choice situations [15]. Loss aversion refers to the tendency for people to be more averse to a loss than they would be drawn to an ‘equivalent’ (according to utility theory) gain. This can be plotted as a value function curve, as shown in Fig. 1. The original ANDREA model was able to replicate the shape of this curve, which indicated that it may explain some of the neural processes that underlie human loss aversion [5].

The original ANDREA model was built using the principles of the Neural Engineering Framework (NEF) [18] and implemented in Matlab [5]. The original code is no longer available, and since ANDREA is an interesting model, it is important to update it so that more people can study it. There is now a new Python software package for building neural models that makes use of NEF principles, and is publicly available, called the Nengo Neural Simulator<sup>2</sup>. The current model was implemented using Nengo.

The first part of this paper details the design decisions made in the reimplemention of the ANDREA model, including the structure of the model, the equations calculated over the connections between neural ensembles (i.e., populations or brain areas), and the selection of other model parameters. The second part compares the neural activities of the current and original models, and examines whether the value function of prospect theory can be matched. Finally, possible avenues of future work are discussed.

## II. MODEL ADAPTATION

Since the original code was not available, the design of the current model was based on only the information available in [3-5] and reasonable guesses when details were not included. The most useful paper was not published [3], but it contained the most information about the basic workings of the model, especially the output activity of the various neural ensembles.

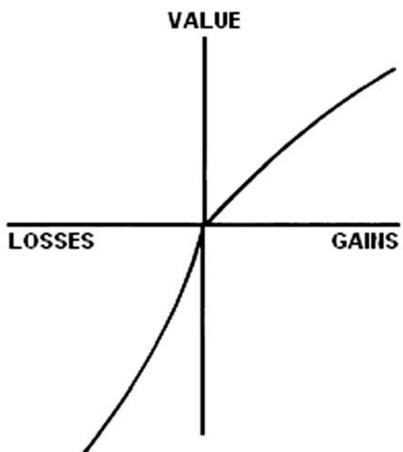


Fig. 1. Prospect theory value curve. From [15].

<sup>2</sup> <http://www.nengo.ca/>

Fig. 2 shows the connectivity and the activity of the main neuron ensembles in the original model [3]. This was of great help in determining the structure of the current model, and also in testing whether the current implementation was producing reasonable values and activities in each of its ensembles.

Five main neural ensembles are used in the current model, and they are based on five brain areas: amygdala (AMYG), orbitofrontal cortex (OFC), dopaminergic neurons (DA), dorsal raphe neurons (5HT), and ventral striatum (VS).

### A. Neural ensembles

#### 1) Amygdala

The amygdala represents the overall activation, or state of arousal, of the system, denoted by the function  $A(t)$ . This activity is the modulated sum of activity in the dopamine system, the serotonin system, as well other emotional arousal unrelated to reward:  $A_0(t)$  [4].

$$A(t) = A_0(t) + \beta \cdot DA(t) + \gamma \cdot 5HT(t) \quad (1)$$

The constants  $\beta$  and  $\gamma$  in (1) refer to the strength of the connections from DA to AMYG and from 5HT to AMYG [5]. In effect, they increase the weight the amygdala places on a particular input signal when it amalgamates the signals from DA and 5HT. Since these are effectively opposite signals, it was assumed that both constants should have a value greater than 1, so that one signal does not overpower the other. In the current model, the specific values used are  $\beta=3$  and  $\gamma=4$ . These values were not provided in any of the papers on the original model, and so were determined by starting from the statement that the amplitude of the signal during a loss is approximately 200% higher than during a gain [3], which indicates that  $\gamma$  should be approximately twice the value of  $\beta$ . The output of the model was then tested with many values of  $\beta$  and  $\gamma$ , with the values above providing the most similar output to the original model.

#### 2) Orbitofrontal cortex

OFC represents the two parts of the subjective modulated valuation calculation in its two dimensions. The multiplication of these two takes place over the output connections to the ensembles which have  $S(t)$  as an input.  $S(t)$  is determined by

$$S(t) = A(t) \cdot V(t) \quad (2)$$

where  $V(t)$  is a baseline stimulus valuation, assumed to be determined by sensory and cognitive processing that is not included in the model [5]. Instead, it is provided as an input signal.

#### 3) Dopaminergic neurons

These three independent neural ensembles make up the dopamine system and collectively represent positive valuation in the model. See Table 1 for the transformation equations.

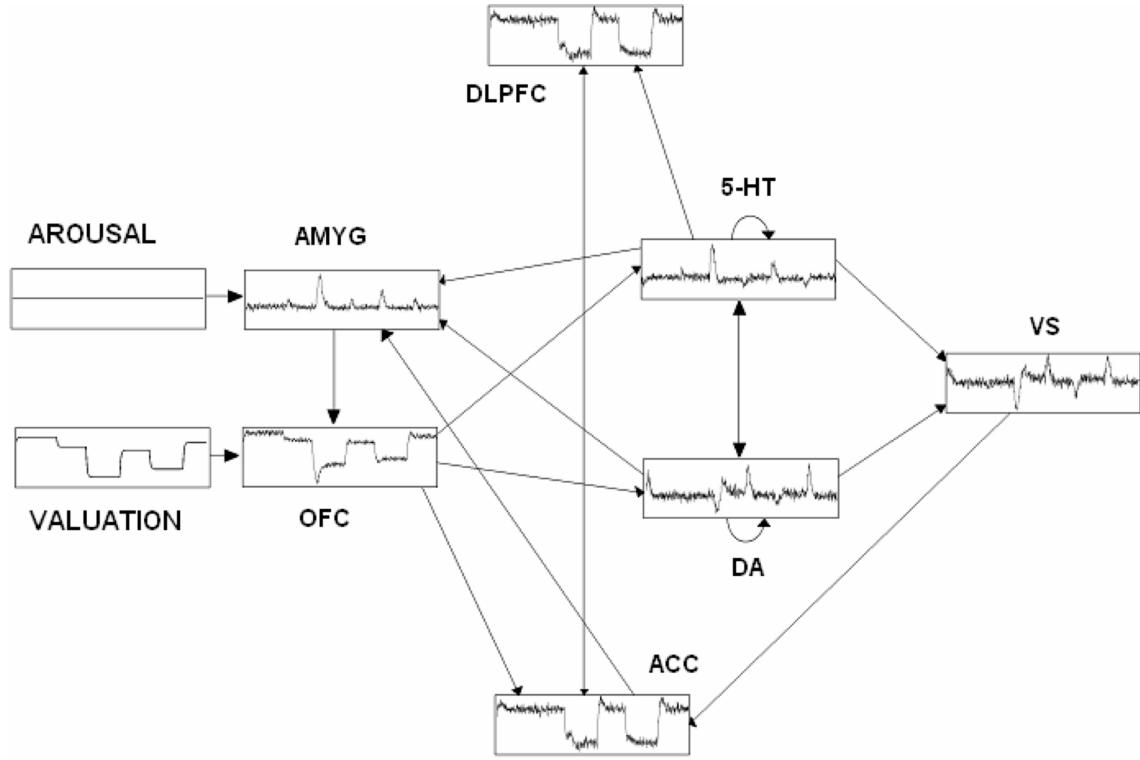


Fig. 2. Connectivity and main neural ensemble activities from original ANDREA model. Note that the y-axis of the arousal plot have a midpoint of 1, while the midpoint is 0 for the other plots. From [3].

- DA\_P: A recurrent ensemble to calculate the weighted sum of previous rewards  $P(t)$ , with  $\alpha$  (learning rate between 0 and 1) denoting the weight given to previous error.
- DA\_E<sup>+</sup>: This represents the difference between the subjective modulated valuation signal  $S(t)$  and the weighted sum  $P(t)$ , i.e., the positive prediction error.
- DA: This activity is shown in Fig. 2, and combines the positive and negative prediction errors, with more emphasis on its own positive error ( $\sigma = 0.75$ ).

#### 4) Dorsal raphe neurons

These three independent neural ensembles make up the serotonin system and collectively represent negative valuation in the model. See Table I for the transformation equations.

- 5HT\_P: A recurrent ensemble to calculate the weighted sum of previous rewards  $P(t)$ . It is not clear why this is calculated both here and the the dopaminergic neurons.
- 5HT\_E<sup>-</sup>: This represents the difference between the weighted sum  $P(t)$  and the valuation signal  $S(t)$ , i.e., the negative prediction error.
- 5HT: This activity is shown in Fig. 2, and combines the negative and positive predictions errors, with more emphasis on its own negative error ( $\sigma = 0.75$ ).

#### 5) Ventral striatum

This ensemble represents the difference between the positive valuation  $DA(t)$  and the negative valuation  $5HT(t)$ , providing an error signal.

#### 6) Anterior cingulate cortex (ACC)

In the original model, this ensemble was used to represent the behaviour selection based on the representations of emotional activity in the rest of the system, however, since the current model does not use this behaviour ‘signal’, the ACC is used simply to represent the result of the calculation of (2).

TABLE I. TRANSFORMATION SUMMARY

Brain area	Inputs	Outputs
AMYG	$A_0(t)$ (ext.) $DA(t)$ $5HT(t)$	$A(t) = A_0(t) + \beta \cdot DA(t) + \gamma \cdot 5HT(t)$
OFC	$V(t)$ (ext.) $A(t)$	$S(t) = A(t) \cdot V(t)$
5HT_E <sup>-</sup>	$S(t)$ $P_{5HT}(t-1)$	$E^-(t) = P_{5HT}(t-1) - S(t)$
5HT	$E^+(t)$ $E^-(t)$	$5HT(t) = \sigma E^+(t) - (1-\sigma)E^-(t)$
5HT_P	$P_{5HT}(t-1)$ $E(t)$	$P_{5HT}(t) = P_{5HT}(t-1) + \alpha E(t)$
DA_E <sup>+</sup>	$S(t)$ $P_{DA}(t-1)$	$E^+(t) = S(t) - P_{DA}(t-1)$
DA	$E^+(t)$ $E^-(t)$	$DA(t) = \sigma E^+(t) - (1-\sigma)E^-(t)$
DA_P	$P_{DA}(t-1)$ $E(t)$	$P_{DA}(t) = P_{DA}(t-1) + \alpha E(t)$
VS	$DA(t)$ $5HT(t)$	$E(t) = DA(t) - 5HT(t)$
ACC	$S(t)$	$S(t)$

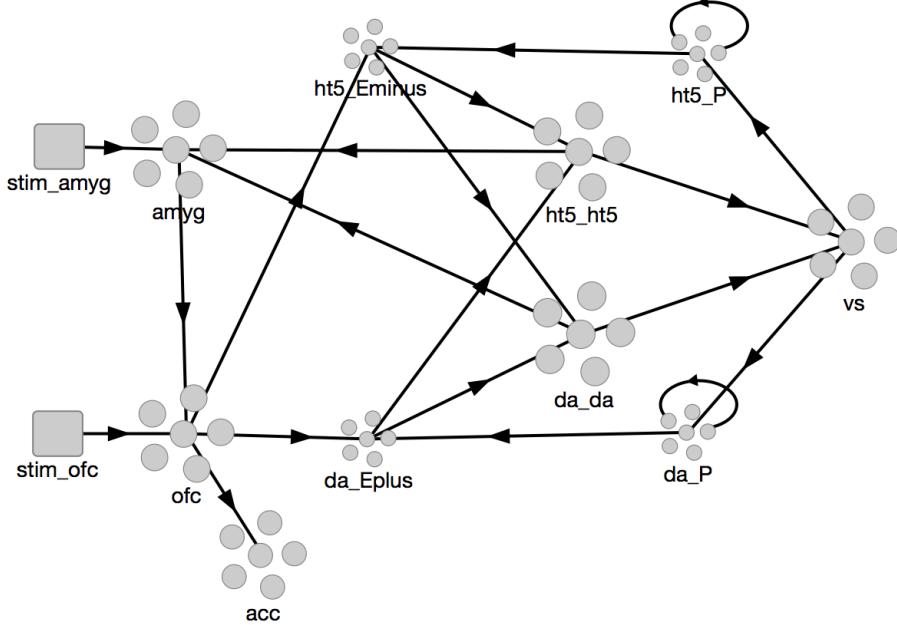


Fig. 3. Connectivity of the current model. Abbreviations: stim\_amyg, A0(t); stim\_ofc, V(t); amyg, amygdala; ofc, orbitofrontal cortex; ht5\_ht5, dorsal raphe neurons (5HT); ht5\_Eminus, 5HT\_E-; ht5\_P, 5HT\_P; da\_da, dopaminergic neurons (DA); da\_Eplus, DA\_E+; da\_P, DA\_P; vs, ventral striatum (VS). Main ensembles of the original model are shown larger. Note that the original DA and 5HT ensembles have each been divided into three separate subensembles.

### B. Other model details

Table 1 details the equations that define the transformations of the neural activity over the connections between ensembles. This was adapted from Table 1 in [3], but it was not clear how exactly this table worked. For instance, were the equations in the output column meant to specify what was being represented by the neural ensembles, or the transformations that took place over the connections between two ensembles? It is not certain which was the strategy chosen, though it is likely that it varied by ensemble. In most cases in the current model, the ensembles represent the result of transformations that take place over their inputs. The one exception to this is due to the multiplication taking place in the OFC. Due to the principles of the NEF, multiplication is best done through the connections coming out of a two-dimensional population, so for OFC to represent the value  $S(t)$  there would need to be an earlier population with  $A(t)$  and  $V(t)$  in its two dimensions. Since an earlier population is not present in the original model, it is more likely that the transformations occurred in the output connections.

There are direct connections between the amygdala and the ventral striatum in human brains [11], and the authors of the original model do not explain why these direct connections are not included in their model. In fact, the error signal of VS is never explicitly sent to any ensembles in the original model, it simply appears in the previous reward equations of DA and 5HT, with no connection or input shown (see Fig. 2). The current implementation assumes that there are connections from VS to both DA and 5HT carrying this signal.

The dorsolateral prefrontal cortex (DLPFC) was included in the original model to represent behaviour planning, but similarly to the activity of ACC, this activity is not necessary to the calculation of subjective valuation. Its output is included in the

original model as a final addition to (1) called  $C(t)$ ; however,  $C(t)$  appears to be another multiplication of the output of 5HT, so in the current implementation it was incorporated into the  $\gamma \cdot 5HT(t)$  term. It is possible that this term diminished the influence of 5HT, since it represented a cost of dealing with conflict [3], in which case it is more likely that  $\gamma$  should be less than twice the value of  $\beta$ , as it is in the current implementation.

The populations representing  $E^+$  and  $E^-$  in DA and 5HT, respectively, are unique in that the neurons only fire when their input is positive, due to having tuning curves with minimum firing thresholds above zero and only positive slopes [3]. The firing thresholds of the other ensembles are drawn from a uniform distribution over the representation range, and the tuning curves can have positive and negative slopes. The maximum firing rates of all the ensembles are chosen from a uniform distribution between 100 and 200 Hz.

Each of the AMYG, OFC, VS, and ACC ensembles have 1000 neurons, chosen as an average of the 800 to 1200 range for these ensembles specified in [5]. The subensembles of 5HT and DA have 400 neurons each, since [5] stated that both of these ensembles have 1200 neurons in total.

The structure of the current model is shown in Fig. 3.

### III. RESULTS AND DISCUSSION

The current model was first evaluated by providing an input signal similar to that of the original model (see Fig. 4), and verifying whether the current model's activity matched. As can be seen from Figs. 5-9, the current model replicates the neural activities of the original five main ensembles fairly well, though there appears to be a great deal more noise in the current implementation.

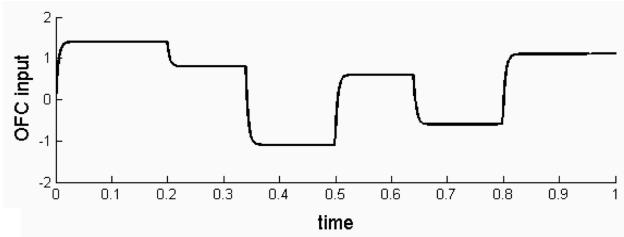


Fig. 4. Input signal  $V(t)$ . From [3].

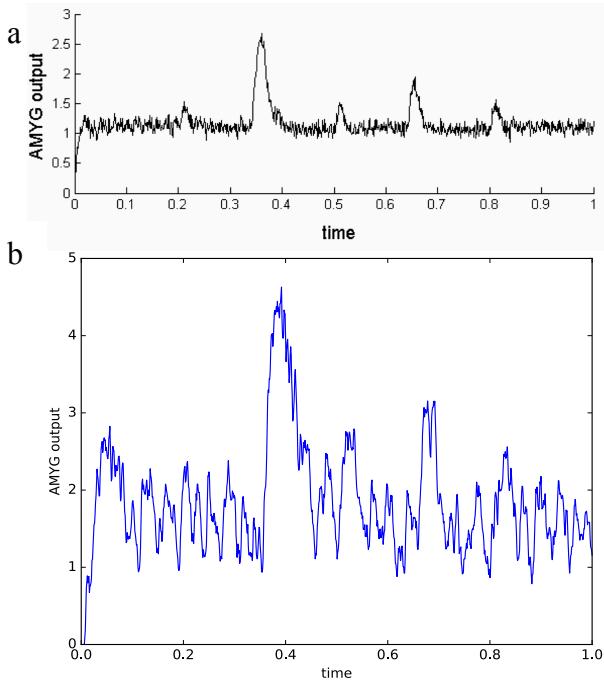


Fig. 5. Amygdala output activity. (a) Original model, from [3]. (b) Current model.

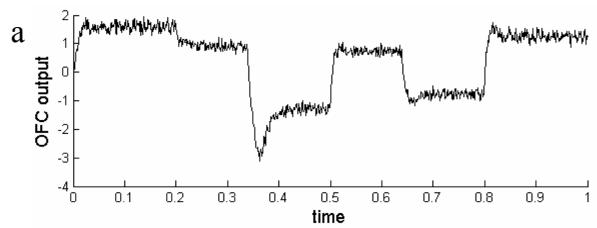


Fig. 6. OFC output activity. (a) Original model, from [3]. (b) Current model.

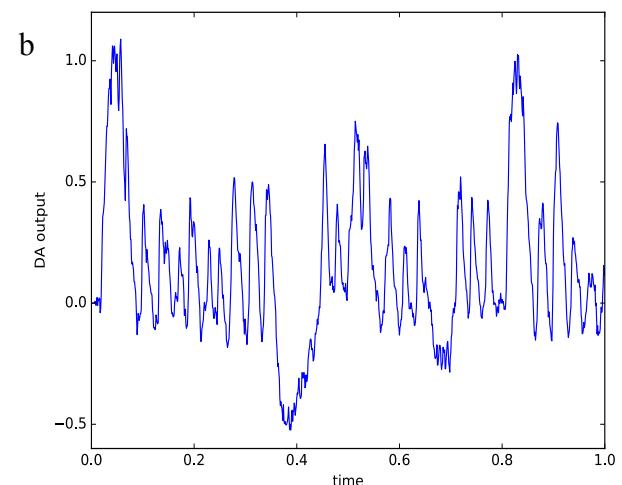
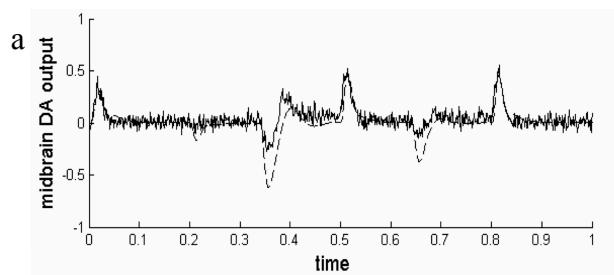


Fig. 7. DA output activity. (a) Original model, from [3]. (b) Current model.

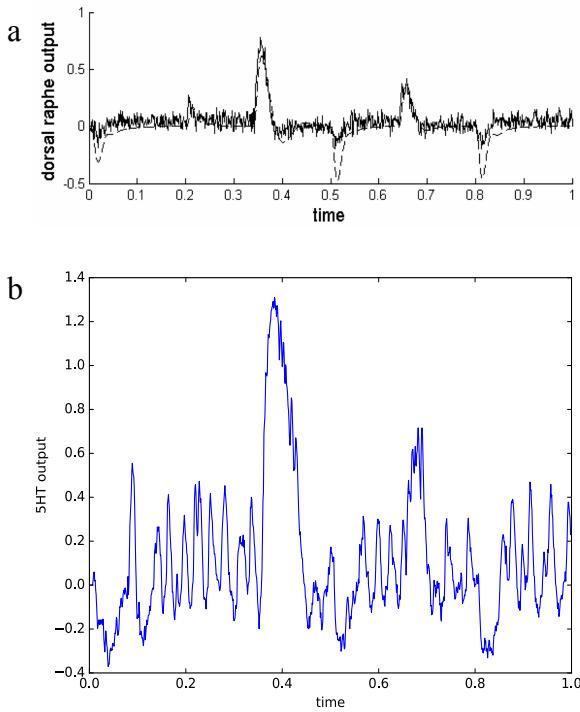


Fig. 8. SHT output activity. (a) Original model, from [3]. (b) Current model.

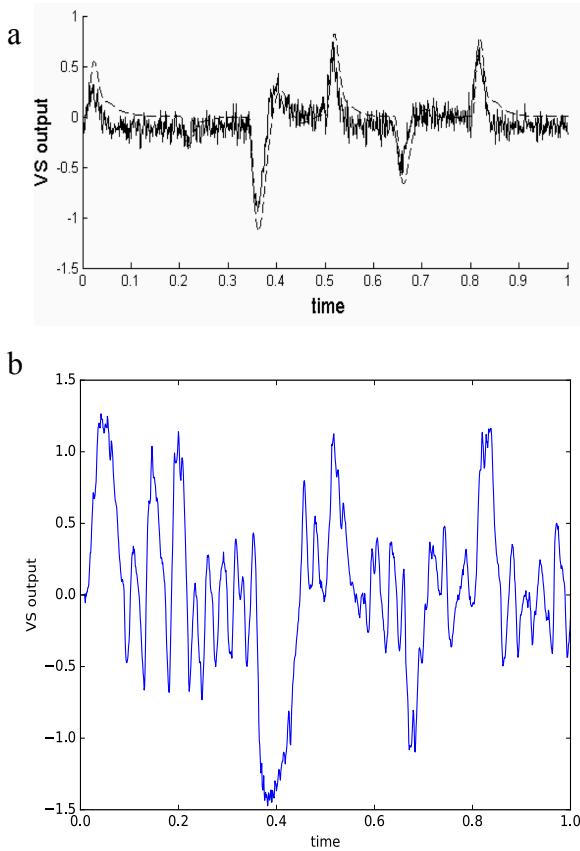


Fig. 9. VS output activity. (a) Original model, from [3]. (b) Current model.

Fig. 5 shows that the peaks in the output of the current implementation are much wider than those of the original, and that the arousal produced by positive changes in reward is very difficult to distinguish from the random noise that is also present. It is not yet clear which model parameter(s) are influencing this result, since changes to the synaptic time constants (i.e., how long it takes for neurons to respond to synaptic input [17]) and changes to the connection weight constants  $\beta$  and  $\gamma$  did not have much effect on peak width. Increasing the learning rate  $\alpha$  from 0.5 to 0.73 produced slightly narrower peaks, but increasing further than that seemed to only add more noise.

The current DA and 5HT output, shown in Fig. 7 and Fig. 8 are also much noisier than the original model, yet the overall shape matches very well. In both cases, there is far more response to positive values than negative. For DA, the amplitude of the responses are approximately twice those of the the original model, which may be related to the increase in noise as well.

The VS output, shown in Fig. 9, is clearly the noisiest of all the current model's ensemble outputs, especially at the beginning of the signal. This is likely because it is a combination of DA and 5HT outputs, which both have a large amount of positive noisy activity.

The original model was tested on its ability to replicate findings from prospect theory [15]. It was not clear how the authors of the original model adapted it from taking  $V(t)$  inputs in the range (-2, 2), as shown in Fig. 4, to the range (-20, 20), shown in Fig. 10a. Due to neural saturation (when neurons reach their maximum firing rate [18]), it does not seem likely that the original OFC population would have been able to represent the smaller range well (i.e., with so little noise) if it also had to be able to represent the larger range. There may have been some unspecified translation of the inputs. The emotional response of the current model over different reward value changes was measured over the range (-2, 2), as shown in Fig. 10b. The negative evaluations do not decrease immediately as quickly as they get farther from zero, but the slope of the loss values is clearly steeper than that of the gain values, which agrees with the general principle of loss aversion.

A major weakness of the original model is that there is not much evidence that serotonin is implicated in loss. The basic argument of [4] was that there is a great deal of evidence for dopamine's involvement in reward processing, especially of positive prediction errors, and there is a proposal of the opponency of dopamine and serotonin in representing reward and punishment in reinforcement learning [7], so it is likely that the serotonin system is responsible for negative prediction errors. However, more recent evidence has suggested that dopamine may be responsible for both positive and negative reward signals [19]. As well, increasing serotonin may increase positive emotions and mood [20]. This confusion may be due to the difficulty in studying these neurotransmitters *in vivo* [21], so it is clear that further research is needed before a fully plausible model of valuation can be developed.

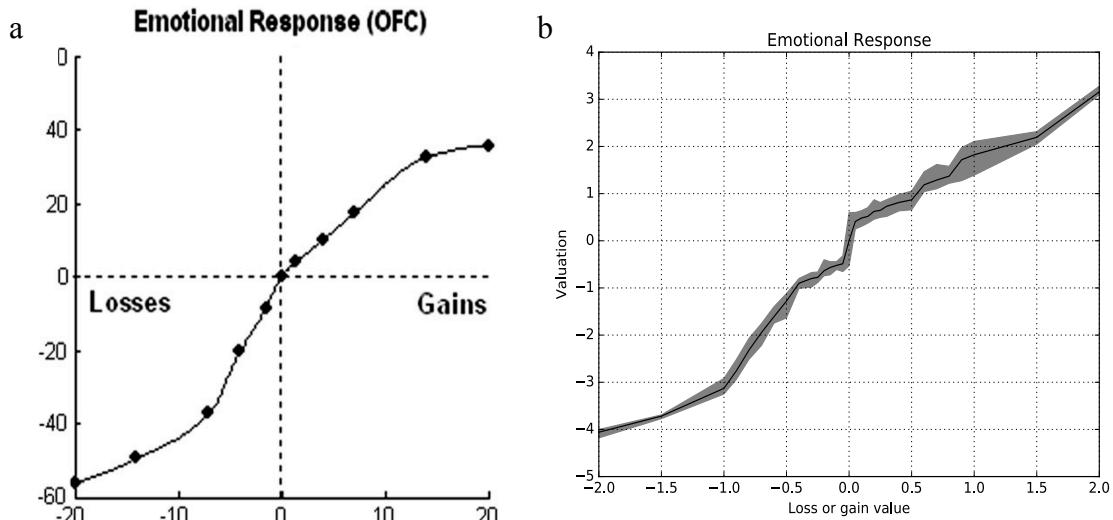


Fig. 10. Comparison of value function simulations. (a) Original model, from [3]. (b) Current model.

#### IV. CONCLUSIONS AND FUTURE WORK

The current implementation of the ANDREA model still requires refinement, but it matches the general behaviour of the original model fairly well. Despite many of the implementational details being left out of the papers that describe the model, a reasonable reimplemention was attained.

The main focus of future work will be to improve the performance of the model by further adjusting the many model parameters that were poorly defined in the original model. The most pressing goal is to decrease the amount of noise in the model, and it is not yet clear how this could easily be done. Another goal is to decrease the width of the peaks to better match the original. This problem may be related to the one that is causing the excess noise, so solving either of these problems may ameliorate the other.

Another avenue to explore in future work is modelling the brain areas where  $V(t)$  and  $A_0(t)$  are calculated. The fact that these are currently provided as external inputs decreases the biological plausibility of the model. At the moment, this model suggests that emotions are “added in” to an “emotionless” valuation performed by other parts of the brain [5], which seems more similar to theories of appraisal structure than appraisal processes [22]. Extending the model to include earlier sensory processes could be very informative for how valuation is determined in the human brain.

Finally, it could be useful to extend the model to account for risk aversion. Risk aversion is also described in prospect theory, and refers to people preferring certain options to uncertain options with the same expected value as the certain option [15]. A recent paper has looked at the neural basis of risk aversion [23], and could provide useful data for refining and testing the model.

In conclusion, the reimplemention of the ANDREA model in Nengo has been successfully started, and will provide a good base upon which to build as research provides a better understanding of human reward processing and decision making.

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