A Computational Model of the Amygdala Nuclei's Role in Second Order Conditioning⋆

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Abstract. The mechanisms underlying learning in classical conditioning experiments play a key role in many learning processes of real organisms. This paper presents a novel computational model that incorporates a biologically plausible hypothesis on the functions that the main nuclei of the amygdala might play in first and second order classical conditioning tasks. The model proposes that in these experiments the first and second order conditioned stimuli (CS) are associated both (a) with the unconditioned stimuli (US) within the basolateral amygdala (BLA), and (b) directly with the unconditioned responses (UR) through the connections linking the lateral amygdala (LA) to the central nucleus of amygdala (CeA). The model, embodied in a simulated robotic rat, is validated by reproducing the results of first and second order conditioning experiments of both sham-lesioned and BLA-lesioned real rats.

1 Introduction

Individual learning plays a fundamental role in adaptive behavior of organisms, especially in most sophisticated ones like mammals. Some of the most important mechanisms underlying learning are those studied in classical (Pavlovian) conditioning experiments. In these experiments an animal experiences a systematic association between a neutral stimulus, for example a light (the "conditioned stimulus" or "CS"), and a biologically salient stimulus, for example food (the "unconditioned stimulus" or "US"), to which it tends to react with an innate set of responses appropriate for the US, for example orienting and approaching (the "unconditioned responses" or "UR"). After repeated exposure to couples of CS-US the animal produces the UR even if CS are presented alone.

Since Pavlov's pioneering works [1], a lot of research has addressed classical conditioning phenomena producing a huge amount of behavioral and neural data

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[2]. However, we still lack a comprehensive theory able to explain the full range of these empirical data. Trying to build detailed *biologically plausible* computational models is a necessary step to overcome this knowledge gap. The current most influential models on classical conditioning, those based on "temporal-difference reward prediction error" [3,4] , suffer of several limitations. The main reason is that they have been developed within the machine learning framework with the aim of building artificial machines capable of autonomously learning to perform actions useful for the user. For this reason they are suitable to investigate *instrumental conditioning* phenomena – a type of associative learning based on stimulus-actions associations – but less adequate to explain Pavlovian phenomena mainly based on stimulus-stimulus associations [5,6].

A crucial question on classical conditioning regards the nature of the acquired association between the CS and the UR: is this association direct (CS-UR), as Pavlov himself seemed to claim [1], or does it pass through the unconditioned stimuli (CS-US-UR), as Hull [7] suggested? The long-lasting debate on this topic [2] seems now settled in favor of both hypotheses: in fact, there is now strong empirical evidence supporting the co-existence of both CS-UR and CS-US associations [5,8]. However, a clear understanding of the neural substrates which might be responsible for these two kinds of associations has yet to be gained. In particular, none of the computational models of classical conditioning based on the temporal-difference mechanisms, nor the models which have been proposed as alternatives to them [5,6,9,10], make any significant claim on this point.

Within the empirical literature, Cardinal et al. [8] formulated an interesting hypothesis on the neural basis of stimulus-stimulus and stimulus-response Pavlovian associations. According to this hypothesis, the basolateral amygdala (BLA) stores the CS-US associations, whereas the central nucleus of amygdala (CeA) receives or stores the CS-UR associations (CS-UR associations encoded in the cerebellum [11] are not considered here).

This paper presents an original computational model implementing that general hypothesis. In particular, it represents the first working model specifying the different functions played by the main sub-nuclei of amygdala in classical conditioning. The model, embodied in a simulated robotic rat, is validated by reproducing the results obtained with some first and second order conditioning experiments conducted with sham and BLA-lesioned real rats [12].

Sect. 2 presents the target experiment and the simulated experimental setup. Sect. 3 describes the model's general functioning and the biological constraints taken into account. The mathematical details of the model are presented in the Appendix. Sect. 4 shows the results of the tests of the model and compares them with those obtained with real rats. Finally, Sect. 5 concludes the paper.

## 2   The Target Experiment and the Simulated Environment

The model is validated by reproducing second-order conditioning experiments on real rats (reported as experiment 1a in [12]). The real experiment was conducted

with 19 BLA-lesioned rats and 27 sham-lesioned rats, measuring the behaviours of walking, orienting and "food-cup" (insertion of head in the food dispenser). Namely, in the first phase both groups were trained for 8 sessions lasting 64 min each to acquire a first order conditioned behaviour. Each session was formed by a sequence of trials. In each trial a 10 sec light stimulus was presented, followed by the delivery of Noyes pellets (food) in the food dispenser. Recordings showed that both sham and lesioned rats were able to acquire first order conditioned behaviours. In the second phase the same rats were trained for 3 sessions of 64 min each to acquire a second order conditioned behaviour. A tone stimulus was presented for 10 sec followed by the light stimulus; every 3 trials a "reminder" of the light-food association was presented. The key result was that only sham rats acquired the second order CS-UR association. In accordance with other empirical evidences (see [8] for a review), these experiments suggest that BLA plays a fundamental role in the formation of the association between the CS and the incentive value of the US, and that this association plays a key role in the acquisition of the CS-UR association in second order conditioning.

The real experiment was simulated through a robotic rat ("ICEAsim") developed within the EU project ICEA on the basis of the physics 3D simulator Webots^{TM}. The model was written in Matlab^{TM} and was interfaced with ICEAsim through a TCP/IP connection. The robotic setup is shown in Fig. 1. The environment is formed by a gray-walled chamber, and the stimuli are expressed by 3 panels (vision is used, as no sound is supported by Webots): food delivering in the dispenser occurs when the green panel turns on, light when the yellow one is on, and tone when the red one is on. When one of those stimuli elicits an orienting response within the controller (see Sect. 3), the rat turns toward the panel and then approaches it (these behaviors are hardwired). This behavioural sequence terminates when the rat touches the food-dispenser (that is assumed to correspond to a food-cup behaviour).Although the "degree of embodiment and situatedness" of the setup is rather limited, nevertheless a robotic test was used because in the future we plan to scale the model to more realistic scenarios (for example, the random-lasting time intervals elapsing between rats' orienting and food deliver already started to challenge the robustness of the associative learning algorithms used).

3   The Model

This section presents a general description of the functioning of the model and the biological constraints that it satisfies, while a detailed mathematical description of it (included all the equations) is reported in the Appendix. A key feature of the model (Fig. 1) is the explicit representation of the three major anatomical components of the amygdala [13]: the lateral amygdala (LA), the basolateral amygdala (BLA), and the central nucleus of amygdala (CeA). The model assumes that these components form two functional sub-systems: (1) the LA-CeA sub-system, which forms S-R associations, and (2) the BLA sub-system, which forms S-S associations. Note that in the following "neurons" have to be
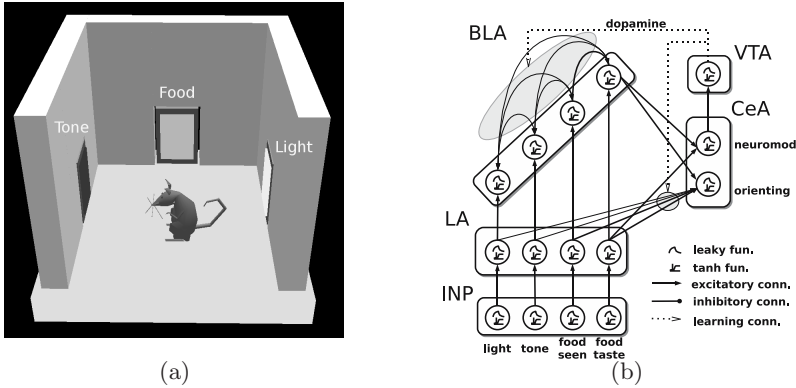
Fig. 1. (a) A snapshot of the simulator, showing the simulated rat at the centre of the experimental chamber, the food dispenser (at the rat's right hand side), the light panel (behind the rat) and the tone panel (in front of the rat). (b) The architecture of the model: bold and plain arrows indicate innate and trained connections, respectively.

intended as units whose functioning abstracts the collective functioning of whole assemblies of real neurons.

The Stimulus-Response Associator (LA-CeA). The LA is the main input of the amygdala system. It receives afferent connections from various sensory and associative areas of cortex, from thalamus, and from deeper regions within the brain-stem, and it sends efferent connections both to BLA and to CeA. The model has an input layer (INP) of four leaky neurons (**inp**) activated by four binary sensors (**s**) which encode the presence/absence of four stimuli: light (s_{li}), tone (s_{to}), food sight (s_{fs}) and food taste (s_{ft}) (Eq. (1)). LA (**la**) is formed by four leaky neurons receiving one-to-one afferent connections from INP (Eq. (2)).

The CeA is one of the main output gates of amygdala. Its efferent connections innervate regions of the brainstem controlling mainly: (1) body and behavioral reactions through the hypothalamus and periaqueductal gray [14]; (2) the release of basic neuromodulators through the ventral tegmental area (dopamine), the locus coeruleus (norepinephrine), and the raphe nuclei (serotonin) [13,15,16]. These neuromodulators play a fundamental role in learning processes but for simplicity this model considers only dopamine [17] (in particular it ignores the role that norepinephrine plays in AMG learning [18]). In the model CeA (**cea**) is formed by two leaky neurons, one (cea_{or}) encoding the rat's orienting behavior, and one (cea_{da}) connected to the ventral tegmental area (VTA) to produce the dopamine signal (da) (Eqs. (4) and (5)).

In the model, all LA neurons are connected to the orienting neuron of CeA (cea_{or}), whereas only the food taste neuron (la_{ft}) is connected to the neuromodulator neuron of CeA (cea_{da}). These connectivity allows stimuli representations of LA to be associated with the orienting behaviour in CeA but not with the dopamine neuromodulation. This is a key assumption to explain why LA-CeA

associations can learn first order CS-US associations but not second order ones: conditioned stimuli cannot access the incentive value of rewarding stimuli.

The connections from LA to CeA are trained on the basis of a Hebb rule. In particular, the strengthening of connections takes place in the presence of three conditions (Eq. (6)): (1) a high value of the trace of the LA activation onset (**la_tr**): the use of the *onset* makes learning happen only when LA neurons' activation precedes CeA neurons' activation, while the use of the *trace* allows overcoming the time gap between CS and UR; (2) a high activation of CeA neurons (cea_{or} and da); (3) a dopamine level (da) over its threshold (th_{da}).

The Stimulus-Stimulus Associator (BLA). The BLA has afferent connections from LA and efferent connections to CeA [19,20]. BLA is also interconnected with the orbitofrontal cortex and hippocampus, and sends efferent connections to the nucleus accumbens: all these connections are ignored here (see [21] for a model where BLA-nucleus accumbens connections play a key role).

In the model, BLA (**bla**) is formed by four leaky units which receive one-to-one connections from LA (**la**) and have all-to-all lateral connections (Eq. (7)). Only the neuron encoding food taste (bla_{ft}) is connected to CeA neurons. This implies that all neurons of BLA representing stimuli different from the US (bla_{ft}) can exert effects on the CeA output neurons only via lateral stimulus-stimulus connections with the BLA's US neuron.

Learning of BLA lateral connections is based on a time-dependent Hebb algorithm. The key aspect of the algorithm is that it allows both the onset and the offset of BLA neurons preceding the onset of other BLA neurons to increase the connection from the former to the latter, provided that dopamine overcomes its threshold (Eqs. (8), (9), (10)). The sensitivity to the offset of stimuli was necessary due to the long duration of the CS stimuli, see Sect. 2 (cf. [21] for a simpler version of the algorithm using only the onset of presynaptic neurons).

4   Results

Figure 2a compares the percentage of times the tone elicits an orienting behaviour in real [12] and simulated rats after the second order conditioning phase. The main result of the experiment has been qualitatively reproduced by the model: in both real and simulated rats a BLA lesion prevents second order conditioning to take place. The analysis of the detailed functioning of the model provides an explanation for this result. Figure 2b shows the activations of some key neurons of: (1) a simulated sham rat during the first order conditioning phase with the light-food contingency; (2) the same sham rat during the second order conditioning phase with the noise-light contingency; (3) a simulated BLA-lesioned rat during the second order conditioning phase.

Figure 2b, first block, shows the mechanisms underlying first order conditioning in a sham simulated rat. At the beginning of the first trial, the appearance of light activates the light-related BLA neuron (bla_{li}). After a while, the appearance of food activates the food-sight BLA neuron (bla_{fs}). The bla_{fs} pre-activates the BLA food-taste neuron (bla_{ft}) before the rat actually reaches the food thanks
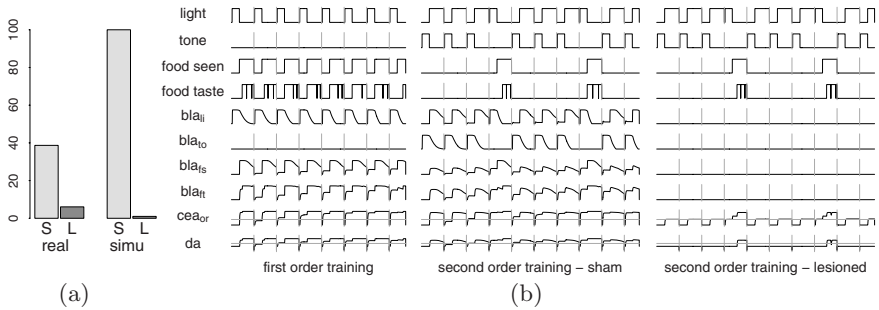
Fig. 2. (a) Percentage of orienting behaviours of sham (S) and lesioned (L) rats in response to the tone after second order conditioning: data from real (first two bars) and simulated rats (last two bars). (b) Stimuli, activations of key neurons, and dopamine release in 3 conditions: first-order and second-order conditioning phases of a sham rat (first and second block, respectively), and second-order conditioning phase of a BLA-lesioned rat. Trials are separated by short vertical dotted lines; thresholds (for orienting behavior and dopamine learning) are represented as gray horizontal dotted lines.

to a bla_{fs}-bla_{ft} excitatory connection which is assumed to be learned before the conditioning training (see the Appendix). In turn, the bla_{ft} triggers both the orienting behavior via the orienting CeA neuron (cea_{or}) and the release of dopamine (da) by the VTA via the CeA neuromodulation neuron. The release of above-threshold dopamine triggers the learning of both the connection between the light neuron in LA and the orienting neuron in CeA (implementing the CS-UR association) and the connections linking the light neuron with the food sight and food taste neurons in BLA (implementing the CS-US association). The result is that after a very few trials the bla_{fs} and bla_{ft} neurons start to be pre-activated as soon as the light is perceived. This results in an early activation of both CeA neurons and, consequently, in an early dopamine release and an early orienting response to the light.

As in the target experiment, during the second order conditioning phase the rats are exposed to sequences of four trials composed by three tone-light presentations and one light-food "reminder". Thanks to the CS-US BLA association acquired during the first phase, in sham rats (Fig. 2b, second block) the presentation of light immediately triggers both orienting behavior and dopamine release. This ability of light to trigger dopamine release permits the acquisition of the second-order association between the tone and the URs (orienting response and dopamine release) in a manner which is completely analogous to what happens in the first-order conditioning with respect to light.

On the other hand, second order conditioning cannot take place in BLA-lesioned rats (Fig. 2b, third block). The reason is that in this case light can trigger only the orienting response via the connection linking the light representation in LA with the orienting neuron in CeA (the direct CS-UR association), but not the dopamine release, which requires the activation of the food-taste representation in either BLA (which is lesioned) or LA (which is activated only when food is

effectively eaten). As a result, since synaptic modification depends on dopamine, no learning can takes place during second-order conditioning.

5   Conclusions

This paper presented an original computational model of the basic brain mechanisms underlying classical conditioning phenomena. The architecture and functioning of the model was constrained on the basis of neural empirical data on the amygdala. The fundamental assumption underlying the model is that the association between conditioned stimuli (CS) and unconditioned responses (UR) formed in classical conditioning experiments is due to two related but distinct mechanisms: (1) stimulus-stimulus associations (CS-US-UR) involving unconditioned stimuli (US) stored in the BLA; (2) direct stimulus-response associations (CS-UR) stored in the LA-CeA neural pathway.

The model was embedded in a simulated robotic rat and was validated by reproducing the behaviours exhibited by both sham and BLA-lesioned rats in first and second order conditioning experiments. In particular, as in real rats, while after training the simulated sham rats react with UR (orienting) to both first and second order CS, BLA-lesioned simulated rats associate UR only to first order CS, but not to second order CS. The model is able to reproduce and explain these results thanks to the fundamental aforementioned assumption. During first order conditioning sham rats acquire both the direct CS-UR and the indirect CS-US-UR association. It is the first order CS-US association within BLA which permits the acquisition of the second order association as it allows the CS to reactivate the appetitive value of the US even when the US is absent. In contrast, BLA-lesioned rats can acquire direct first order CS-UR associations stored in the LA-CeA neural pathway but they cannot acquire the second order association because the first order CS has not access to the appetitive value of the US. To the best of the authors knowledge, this is the first model to propose such a specific computational hypothesis regarding the double association CS-US and CS-UR in classical conditioning.

Notwithstanding its strengths, the model suffers at least two significant limitations. First, the whole behavioral sequence triggered by the activation of the orienting neuron in CeA (orienting, approaching, and food-cup) is fully hardwired. For this reason, the model cannot reproduce the results on CeA-lesioned rats which are reported in the same article of the experiment targeted here [12]. Second, in contrast to most existing models of classical conditioning [5,6,9], the current model does not implement any mechanism for reproducing the exact timing of dopamine release observed in real animals. For this reason the model cannot reproduce another fundamental aspect of classical conditioning, that is extinction (the ability to re-learn not to respond to the CS if it stops to be followed by the US). We are currently working on improved versions of the present model for tackling both these limits.

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Appendix: Mathematical Details of the Model

Throughout the Appendix, τ_x denotes the decay rate of a leaky quantity x, the sub-index $\cdot_p$ denotes the activation potential of the corresponding neuron, symbols $\mathbf{X}$, $\mathbf{x}$, and x are used respectively to denote matrices, vectors and scalars, the function φ is defined as $\varphi[x] = \max[0, x]$ and the function χ as $\chi[x] = 1\,if\,x \geq 0\,else\,\chi = 0$. The values of parameters are listed at the end of the Appendix.

LA-CeA: Functioning and Learning. INP (**inp**) processes the input signal from sensors $\mathbf{s} = (s_{li}, s_{to}, s_{fs}, s_{ft})'$ with a leak function:

$$\tau_{\mathbf{inp}} \cdot \dot{\mathbf{inp}} = -\mathbf{inp} + \mathbf{s} \; . \tag{1}$$

LA is formed by four leaky neurons (**la**) activated as follows:

$$\tau_{\mathbf{la}} \cdot \dot{\mathbf{la}}_p = -\mathbf{la}_p + w_{\mathbf{inp,la}} \cdot \mathbf{inp} \; , \qquad \mathbf{la} = \varphi[\tanh[\mathbf{la}_p]] \tag{2}$$

where $w_{\mathbf{inp,la}}$ is the fixed weight of the connections from IMP to LA. The "double leak" processing of signals implemented by IMP and LA is used to smooth the derivative of LA (see Eq. (3)).

The trace of LA neurons (**la_tr**) is a leak function of the positive value of the derivative of their activation (**la**):

$$\tau_{\mathbf{la\_tr}} \cdot \dot{\mathbf{la\_tr}}_p = -\mathbf{la\_tr}_p + b_{\mathbf{la\_tr}} \cdot \varphi[\dot{\mathbf{la}}] \; , \qquad \mathbf{la\_tr} = \varphi[\tanh[\mathbf{la\_tr}_p]] \tag{3}$$

where $b_{\mathbf{la\_tr}}$ is an amplification coefficient.

CeA is formed by two leaky neurons (**cea**) activated as follows:

$$\tau_{\mathbf{cea}} \cdot \dot{\mathbf{cea}}_p = -\mathbf{cea}_p + \mathbf{W}_{\mathbf{la,cea}} \cdot \mathbf{la} + \mathbf{W}_{\mathbf{bla,cea}} \cdot \mathbf{bla} \tag{4}$$

$$\mathbf{cea} = \varphi[\tanh[\mathbf{cea}_p]]$$

VTA is formed by a dopamine leaky neuron (da) which activates as follows:

$$\tau_{da} \cdot \dot{da}_p = -da_p + bl_{da} + \mathbf{w}_{\mathbf{cea},da} \cdot \mathbf{cea} \; , \qquad da = \varphi[\tanh[da_p]] \tag{5}$$

where bl_{da} is the dopamine baseline.

The weights of the LA-CeA connections ($\mathbf{W}_{\mathbf{la,cea}}$) are updated with a three-element Hebb rule involving CeA, LA's trace and dopamine:

$$\Delta\mathbf{W}_{\mathbf{la,cea}} = \eta_{\mathbf{la,cea}} \cdot (\chi[da - th_{da}] \cdot da) \cdot \mathbf{cea} \cdot \mathbf{la\_tr}' \cdot (1 - |\mathbf{W}_{\mathbf{la,cea}}|) \tag{6}$$

where $\eta_{\mathbf{la,cea}}$ is a learning rate, the term $(\chi[da - th_{da}] \cdot da)$ implies that learning takes place only when $da \geq th_{da}$, and the term $(1 - |\mathbf{W}_{\mathbf{bla}}|)$ keeps the weights in the range $[-1, 1]$.

BLA: Functioning and Learning. BLA is formed by four leaky neurons (**bla**) activated as follows:

$$\tau_{\mathbf{bla}} \cdot \dot{\mathbf{bla}}_p = -\mathbf{bla}_p + \mathbf{W_{bla}} \cdot \mathbf{bla} + (w_{\mathbf{la,bla}} \cdot \mathbf{la} + c_{\mathbf{bla}} \cdot \mathbf{la\_tr}) \tag{7}$$
$$\mathbf{bla} = \varphi[\tanh[\mathbf{bla}_p]]$$

where $c_{\mathbf{la\_tr}}$ is an amplification coefficient. According to this equation, with a transient constant input signal the activation of a BLA neuron presents a high initial peak (due to **la_tr**) followed by a lower constant value (due to **la**) and then by a smooth descent to 0 (due to the leak after the signal end): this activation has a derivative suitable for BLA learning (see below).

In order to train lateral connections of BLA, a trace of the derivative of the activation of BLA neurons **bla_tr** is computed as follows:

$$\tau_{\mathbf{bla\_tr}} \cdot \dot{\mathbf{bla\_tr}}_p = -\mathbf{bla\_tr}_p + \cdot \dot{\mathbf{bla}} . \tag{8}$$

Small values of this trace are ignored in the learning algorithm by considering the "cut trace" **bla_tr_cut** defined as: **bla_tr_cut** = **bla_tr** *if* $|\mathbf{bla\_tr}| < th_{\mathbf{bla\_tr}}$ *else* **bla_tr_cut** = 0. Given the activation dynamics of BLA (Eq. (7)), the corresponding derivative (and, with some delay, its trace) presents: (1) an initial peak at signal onset; (2) a negative peak at the end of the signal onset; (3) a negative peak at the signal offset. The key point of the learning algorithm of BLA is that a connection between two neurons is potentiated in coincidence of a negative peak of the presynaptic neuron and a positive peak of the postsynaptic neuron. These two events mark a pre-synaptic-onset/post-synaptic-onset sequence (or a pre-synaptic-offset/post-synaptic-onset one). The matrix **S**, reported below, captures these conditions for all couples of neurons:

$$\mathbf{S} = \chi[\mathbf{bla\_tr\_cut}] \cdot \chi[-\mathbf{bla\_tr\_cut}]' - \chi[-\mathbf{bla\_tr\_cut}] \cdot \chi[\mathbf{bla\_tr\_cut}]' . \tag{9}$$

Denoting with *pre* and *post* the presynaptic and postsynaptic neurons, **S** has an entry equal to 1 when $bla\_tr\_co_{pre} < 0$ and $bla\_tr\_co_{post} > 0$, equal to -1 when $bla\_tr\_co_{pre} < 0$ and $bla\_tr\_co_{post} > 0$, and equal to 0 otherwise. The learning rule of lateral connections is then:

$$\Delta\mathbf{W_{bla}} = \eta_{\mathbf{bla}} \cdot \chi[da - th_{da}]da \cdot (ltp_{\mathbf{bla}} \cdot \varphi[\mathbf{S}] + ltd_{\mathbf{bla}} \cdot \varphi[-\mathbf{S}])(1 - |\mathbf{W_{bla}}|) \tag{10}$$

where $\eta_{\mathbf{bla}}$ is a learning rate, $ltp_{\mathbf{bla}}$ is a long time potentiation coefficient, and $ltd_{\mathbf{bla}}$ is a short term depression coefficient.

Model's Parameters. The model's parameters were set as follows: $\tau_{\mathbf{inp}} = \tau_{\mathbf{la}} = \tau_{\mathbf{bla}} = 500$ ms, $\tau_{\mathbf{la\_tr}} = \tau_{\mathbf{bla\_tr}} = 5000$ ms, $\tau_{\mathbf{cea}} = 100$ ms, $\tau_{da} = 50$ ms, $w_{\mathbf{inp,la}} = 10$, $b_{\mathbf{la\_tr}} = 1000$, $w_{\mathbf{la,bla}} = 0.5$, $c_{\mathbf{bla}} = 60$, $bl_{da} = 0.3$, $th_{da} = 0.6$, $th_{\mathbf{la\_tr}} = 0.00001$, $\eta_{\mathbf{bla}} = 0.0005$, $\eta_{\mathbf{la,cea}} = 0.15$, $ltp_{\mathbf{bla}} = 1.0$, $ltd_{\mathbf{bla}} = 0.3$. Some connections, assumed to be innate or pre-learned, are clumped to 1 (*l*=learned): $w_{bla\,fs,ft} = 1$, $\mathbf{w_{cea,da}} = (1,0)$, $\mathbf{W_{la,cea}} = \begin{pmatrix} l\,l\,l\,1 \\ l\,l\,1 \end{pmatrix}$, $\mathbf{W_{bla,cea}} = \begin{pmatrix} l\,l\,l\,1 \\ l\,l\,l\,1 \end{pmatrix}$. The model's equations were integrated with the Euler method with a 50 ms step.