

The Role of Amygdala in Devaluation: A Model Tested with a Simulated Rat

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Abstract

This paper presents an embodied biologically-plausible model investigating the relationships existing between Pavlovian and instrumental conditioning. The model is validated by successfully reproducing the primary outcomes of instrumental-conditioning devaluation tests conducted with normal and amygdala-lesioned rats. These experiments are particularly important as they show how the sensitivity to motivational states exhibited by the Pavlovian system can transfer to instrumentally acquired behaviors. The results presented are relevant not only for neuroscience but also for robotics as they start to investigate how internal motivational systems, as those found in real organisms, might modulate the learning and performance of goal-directed actions in artificial machines, so to **improve** their behavioral flexibility.

1. Introduction

Current robots tend to have many serious limitations as they are programmed or evolved to accomplish just one single task and they typically are not able to cope with significant changes in their environment. Living organisms are not so limited as they are able to accomplish many different tasks and have strategies for coping with novel challenges posed by changes involving both their external environment and their internal states. In order to be able to design robots endowed with a similar autonomy and a flexibility one is led to try to understand and mimic the mechanisms underlying organisms' behavioral flexibility. In order to design robots endowed with a autonomy and a flexibility increasingly comparable to those of living organisms, there's the need to understand and mimic the mechanisms underlying organisms' behavioral flexibility. Recently, machine learning and robotics communities have devoted increasing efforts to the study of autonomous development

and learning in robots. (Zlatev and Balkenius, 2001; Weng et al., 2001; Barto et al., 2004; Schembri et al., 2005). Most of this literature builds upon the machine learning framework of reinforcement learning (Sutton and Barto, 1998), which is intended to provide machines with the capacity to learn new behaviors on the basis of rewarding stimuli. Interestingly, reinforcement learning algorithms have gained increasing interest within the empirical literature on animal behavior as they represent theoretical models that can furnish coherent explanations of several key empirical findings (Schultz, 2002).

Notwithstanding their importance, the standard reinforcement learning models suffer of many limitations. From the machine learning point of view, they require a careful specification of task-specific extrinsic reward functions, thus resulting in very limited degrees of autonomy (Barto et al., 2004). From the scientific point of view, they have been criticized for at least two reasons. (1) They do not take into account the role of internal motivations in modulating the effects of external rewards: if an agent, be it a real organism or a robot, has to engage in several different activities, it needs to be endowed with a complex motivational system which is able not only to guide its learning processes, but also to modulate its behavior *on the fly*; one of the most important empirical phenomena challenging the standard reinforcement learning framework, 'devaluation', demonstrates just this kind of effects. (2) They conflate the notions of classical/Pavlovian conditioning and instrumental/operant conditioning: accumulating empirical evidence is indicating that these are different processes that rely on distinct neural systems and that interplay in complex ways overlooked by standard reinforcement learning models (as demonstrated, for example, by the empirical phenomena of 'Pavlovian-Instrumental Transfer' and 'incentive learning', see Dayan and Balleine (2002) and O'Reilly and Watz (2007) for details).

This paper presents a novel computational model which is strongly rooted in the anatomy and physi-

ology of the mammal brain and starts to address some of these issues. In particular, the model presented here reproduces the results of an empirical experiment (Balleine et al., 2003) which demonstrates the phenomenon of devaluation in an instrumental conditioning task and proposes a coherent picture about the possible neural mechanisms underlying it. The model is based on the following hypotheses: (a) the amygdala constitutes a stimulus-stimulus associator at the core of Pavlovian conditioning (Baxter and Murray, 2002; Cardinal et al., 2002); (b) the cortex-basal ganglia (putamen) pathway, forming stimulus-response associations, constitutes the main actor involved in instrumental conditioning (Yin and Knowlton, 2006); (c) the amygdala-nucleus accumbens pathway constitutes another stimulus-response selector that ‘bridges’ Pavlovian processes happening in the amygdala and instrumental processes taking place in the basal ganglia (Baxter and Murray, 2002). By reproducing the basic results of both normal and lesioned rats the model provides significant evidence for these three fundamental hypotheses and, more importantly, it contributes to clarify the relationships existing between the neural structures and processes underlying them.

The rest of the paper is structured as follows. Sec. 2. reports the original experiments addressed by the model. Sec. 3. describes the robotic setup and the simulated experiment. Sec. 4. contains a detailed description of the model. Sec. 5. reports the main results. Finally, Sec. 6. concludes the paper.

2. Target experiment

The target data addressed with the model are reported in Balleine et al. (2003) which illustrates various experiments directed to investigate the relations existing between the manipulation of the value of primary rewards (devaluation) and instrumental conditioning, and the role that Amygdala (Amg) plays in them. The present work focusses on ‘Experiment 1’ reported in the article, a standard ‘devaluation test’.

In two preliminary phases of the experiment, 8 sham plus 8 rats whose Basolateral Amygdala complex (‘BLA’) was lesioned were trained in separate trials to press a lever and pull a chain to obtain respectively Noyes pellets and maltodextrin. The training phase was followed by an extinction test lasting 20 minutes (divided in groups of 2 minutes) where: (1) both manipulanda were present in the experimental chamber; (2) half of the rats had been previously satiated with Noyes pellets while the other half with maltodextrin. The main result is that during the first two minutes of the test non-lesioned rats performed the action corresponding to the manipulandum of the non-satiated food with a much higher rate with respect to the other manipulandum, even if they had never experienced this condition before.

On the other hand, BLA-lesioned rats did not show any devaluation effect: they performed the two actions at the same rate. These experiments clearly demonstrate that BLA plays a fundamental role in the transfer of the diminished hedonic value of food to instrumentally acquired behavior. This key finding, central for clarifying the relationship existing between Pavlovian and instrumental conditioning, is the target of the model presented in the following sections.

3. The simulated environment, the robot and the experiments

The model presented here was tested within an embodied system because, as mentioned in the introduction, the long-term goal of this research is to build robot controllers that on one side are based on sound anatomical and physiological neuroscientific evidence (this should give the robots the flexibility that characterizes real organisms), and on the other side is capable of scaling to function in realistic robotic setups. Although we are aware that the role of the ‘degree of embodiment and situatedness’ of the model and simulations presented here is rather limited, because sensors and actuators are rather simplified and the model’s low-level behaviors are hardwired, nevertheless testing the model in a robotic simulation forced us to design the model in such a way that in the future it might be able to cope with the difficulties posed by more realistic setups. For example, the use of the robotic simulator introduced random durations in the various phases of training, testing, and action execution that posed interesting challenges to the time properties and robustness of the learning algorithms of the model.

The model was tested with a simulation of a rat (‘ICEAsim’) developed within the EU project ICEA with the use of the physics 3D simulator WebotsTM. Webots furnishes a high-level interface for building robot simulations, and is based on the open source library ODE for the simulation of dynamics and interactions between rigid bodies. The model was written in MatlabTM and was interfaced with ‘ICEAsim’ through a TCP/IP connection. The robotic setup used to test the model is shown in Fig. 3.a and it is now briefly described skipping irrelevant details. The training and test environment is composed by a grey-walled chamber containing a yellow lever, a red chain, and a food-dispenser that turns green or blue when food A or food B is delivered in it. When ‘pressed’ or ‘pulled’, the lever and chain make respectively food A or B (the ‘rewarding’ stimuli) available at the dispenser.

The simulated rat is a wheel-chair robot (‘ICEAsim’) equipped with various sensors. Among these, the experiments reported here use the camera

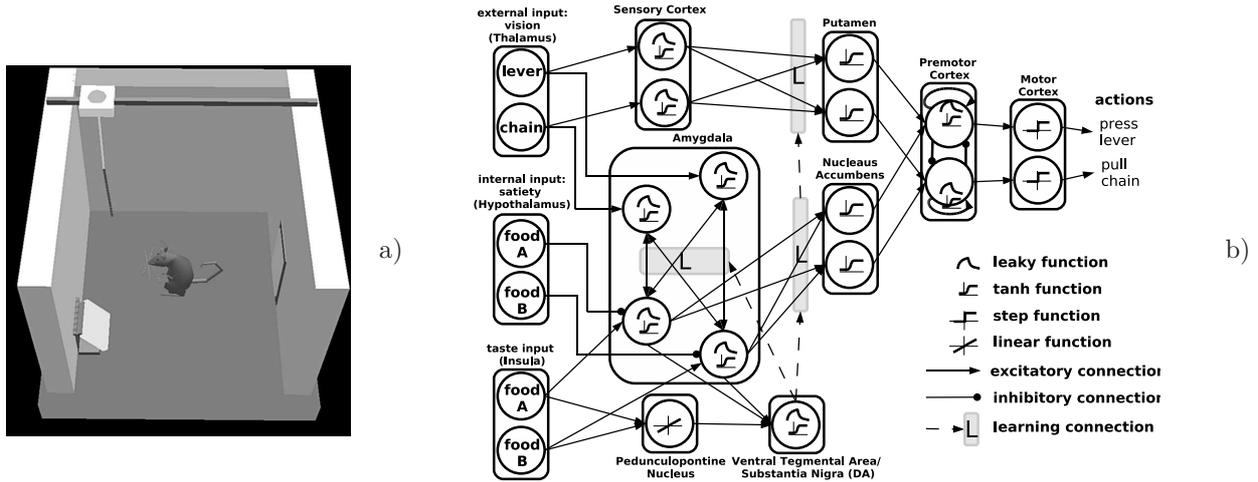


Figure 1: a) A snapshot of the simulator, showing the simulated rat at the center of the experimental chamber, the white/yellow food dispenser (patch on the wall behind the rat), the lever (at the rat’s left hand side) and the chain (at the rat’s right hand side). b) The architecture of the model.

and the whisker sensors. The rat uses the camera, having a panoramic 360 degrees view, to detect the lever, the chain and the food dispenser, in particular their presence/absence (via their color) and their (egocentric) direction. The whiskers, activated with one if bent beyond a certain threshold and zero otherwise, allow the rat to detect contacts with obstacles. The rat is also endowed with *internal* sensors related to satiety for either food A or B (these sensors assume the value of one when the rat is satiated, and zero otherwise). The rat’s actuators are two motors independently controlling the speed of the two wheels.

The information fed to the model is only related to the presence/absence of the lever and chain in chamber and food A and food B in mouth, whereas the other information is used to control a number low-level hardwired behavioral routines. The routines, triggered either by the model or directly by stimuli, are as follows: (1) ‘obstacle avoidance routine’: triggered by the whiskers, this routine ‘overwrites’ all other actions to avoid obstacles; (2 and 3) ‘lever press routine’ and ‘chain pull routine’: whenever activated by the model, these routines causes the rat to approach the lever/chain on the basis of its visually detected direction **the lever/chain are considered to be pressed/pulled when the rat’s distance from them is below a threshold**; (4) ‘consummatory routine’: when the dispenser turns **green or blue**, the rat approaches it **and ‘consumes’ its content so** causing the perception of either food A or food B in mouth.

The devaluation experiment is divided in a ‘training phase’ and a ‘test phase’. The training phase lasts **480 s** whereas the test phase lasts **240 s**. Each phase is divided in trials. In each trial of the training phase, either the lever and food A (when after the

lever is pressed) or the chain and food B (when after the chain is pulled) are present in an alternate fashion. The rat is set in the middle of the chamber with an **orientation set between lever and chain**. The trial ends either when the rat executes the correct action (i.e., it executes the lever press or pull chain action in presence of respectively the lever or chain) or when a timeout of **15 s** elapses. The consummatory routine related to either food A or B (rewards) lasts **until the rat touches the food panel more than 10 times**. During training rats are ‘hungry’, meaning that their satiation sensors are both set at 0.

The test phase is divided in two sub-parts, one with trials where the rat has been satiated with food A (i.e., its satiation sensors for food A and B are respectively set to one and zero) and one with food B. In all trials *both* the chain and the lever are present and the rat is evaluated in extinction, that is without delivery of food after the execution of actions. Trials end when either one of the actions is executed or a timeout of 10 s elapses. The experiment was run 20 times with ‘unlesioned’ artificial rats and 20 times with ‘lesioned’ rats (the statistics reported in Sec. 5. regard 40 measurements related to lesioned rats and 40 to unlesioned rats as the test is divided in two sub-parts).

4. The model

The model’s input is formed by six neurons activated by the sensors illustrated in Sec. 3.: two neurons encode the presence/absence of the lever and the chain (s_{lev} and s_{cha}), two neurons encode the presence/absence of food A food B in the rat’s mouth (s_{fA} and s_{fB}), and two neurons encode the satiation for food A and food B (s_{sfA} and s_{sfB}).

The model (Fig. 3.b) is formed by three major

components: (1) a S-S associator, corresponding to Amg; (2) a S-R static action selector, corresponding to the sensory cortico-striatal pathway (SC, PUT); (3) a S-S-R dynamic associator, corresponding to the Amg-NAc core pathway.

4.1 The amygdala: a stimulus-stimulus associator

The associator implements Pavlovian conditioning through the association between CSs and USs ('stimulus substitution'). In real brains this role seems to be played by the Amg (Baxter and Murray, 2002; Cardinal et al., 2002). There are massive reciprocal connections between the Amg and several brain areas, including: inferotemporal cortex (IT), insular cortex (IC), prefrontal cortex (PFC), and hippocampus (Hip) (Price, 2003; Rolls, 2005; Baxter and Murray, 2002; Cardinal et al., 2002). Furthermore, Amg receives inputs from posterior intralaminar nuclei of thalamus (PIL) (Shi and Davis, 1999). These connections underlie an interplay between processes related to perceived or represented external context (IT, PFC, Hip) and processes related to internal states (IC, PIL). In general Amg can be seen as playing the function of assigning a subjective valence to external events on the basis of the animal's internal context (needs, motivations, etc.), and to use this to both regulate learning processes and directly influence behavior.

Our associator, which is considered as an abstraction of the processes taking place in the Amg, performs 'asynchronous learning/synchronous functioning' associations: first, stimuli perceived in different times are associated (CSs are associated to USs): **For this associative learning to take place there's the need for dopamine (DA) to be released with the activation of USs (see below).** When the association is established, CSs are able to synchronously recall the USs. The associator is composed by a vector $\mathbf{amg} = (amg_{lev}, amg_{cha}, amg_{fA}, amg_{fB})$ of four leaky neurons, that process the input signals as follows:

$$\begin{aligned} \tau_{\mathbf{amg}} \cdot \dot{\mathbf{amg}}_p &= -\mathbf{amg}_p + \\ & (s_{lev}, s_{cha}, (s_{fA} - s_{sfA}), (s_{fB} - s_{sfB}))' + \\ & \mathbf{W}_{amg} \cdot \mathbf{amg} \end{aligned} \quad (1)$$

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

where \mathbf{amg}_p are the activation potentials of Amg's activations, $\varphi[x] = 0$ if $x \leq 0$ and $\varphi[x] = x$ otherwise and \mathbf{W}_{amg} is the matrix of all-to-all lateral connection weights within Amg. Note that while external stimuli have a binary representation (0/1 for absence/presence), internal stimuli *modulate* the representation of external stimuli. In particular s_{sfB} and

s_{sfB} assume a value in $\{0, 5\}$ when the corresponding satiation has respectively a low or high value, and this simulates the fact that satiation for a food inhibits the hedonic representation of such food within Amg. This assumption is supported by evidence indicating that a similar computation is performed in the secondary taste areas of the prefrontal/insular cortex (Rolls, 2005) connected with Amg. This part of the model is particularly important because, as we shall see, it mediates the influence of the shifts of primary motivations on both learning and behavior.

The associator's learning is based on the *onset* of input signals, detected as follows. First, 'leaky traces' \mathbf{tr} of \mathbf{amg} derivatives, trunked to positive values, are computed as follows:

$$\tau_{\mathbf{tr}} \cdot \dot{\mathbf{tr}} = -\mathbf{tr} + C_{Amg} \cdot \varphi[\mathbf{amg}] \quad (2)$$

where C_{Amg} is a coefficient used to amplify the increments of \mathbf{amg} . Second, the derivatives of \mathbf{tr} are computed: when positive, these derivatives detect the onset of the original signals, whereas when negative they detect the fact that some time has elapsed since such onset took place.

The weights between Amg's neurons are updated on the basis of the DA signal (see below) and the signs of $\dot{\mathbf{tr}}$. In particular, when the derivative of the presynaptic neuron's trace is negative and the derivative of the postsynaptic neuron's trace is positive (i.e. when the presynaptic neuron fires before the postsynaptic neuron) the related connection is strengthened (this condition related to all couples of neurons is denoted with the Boolean matrix \mathbf{L}):

$$\Delta \mathbf{W}_{amg} = \eta_{amg} \cdot \varphi[da - th_{da}] \cdot \mathbf{L} \quad (3)$$

where η_{amg} is a learning rate coefficient, da is the dopamine signal and th_{da} is a threshold over which dopamine elicits learning. DA release (corresponding to activation in the ventral tegmental area, VTA, and in the substantia nigra pars compacta, SNpc) is triggered by Amg through the units representing the hedonic impact of food and by the primary reward signals received from the pedunculo pontine tegmental nucleus (PPT) (Kobayashi and Okada, 2007):

$$\begin{aligned} \tau_{da_p} \cdot \dot{da}_p &= -da_p + da_{baseline} + \\ & w_{amg-da} \cdot (amg_{fA} + amg_{fB}) + \\ & w_{ppt-da} \cdot ppt \end{aligned} \quad (4)$$

$$da = \varphi[\tanh[da_p]]$$

where $ppt = s_{fA} + s_{fB}$ is the PPT's primary reward signal. DA drives learning in both the associator and the action selectors (see Sec. 4.2 and 4.3).

4.2 The cortex-putamen pathway: a static S-R action selector

The static action selector learns ‘habits’, rigid S-R associations, through reinforcement learning processes. In real brains this function might be implemented in the cortex-mediolateral striatum pathway involving in particular the Putamen (PUT) (Yin and Knowlton, 2006). In the model this component receives s_{lev} and s_{cha} as input and, on the basis of this, selects one of the two lever-press/chain-pull actions (together with NAc, see Sec. 4.3).

The component is formed by four layers of neurons corresponding to four vectors: (1) a visual sensory cortex (SC) leaky-neuron layer: \mathbf{sc} ; (2) a layer corresponding to PUT’s encoding of the ‘votes’ for the two actions: \mathbf{put} ; (3) a layer corresponding to pre-motor cortex (PM), formed by reciprocally inhibiting neurons that implement a competition for selecting one of the two actions (this function might be implemented by the reciprocal thalamo-cortical connections (Dayan and Balleine, 2002): \mathbf{pm} ; (4) a layer corresponding to motor cortex (M), representing the selected action with a binary code: \mathbf{m} .

The visual leaky-neuron layer processes the input signal in a straightforward fashion:

$$\tau_{sc} \cdot \dot{\mathbf{sc}}_p = -\mathbf{sc}_p + (s_{lev}, s_{cha})' \quad (5)$$

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

SC is fully connected with PUT. PUT’s non-leaky neurons collect the signals from SC that tend to represent the evidence (‘votes’) in favor of the selection of either one of the two actions:

$$\mathbf{put}_p = \mathbf{W}_{(sc-put)} \cdot \mathbf{sc} \quad (6)$$

$$\mathbf{put} = \varphi[\tanh[\mathbf{put}_p + \mathbf{put}_{baseline}]]$$

The selection of actions is performed on the basis of these votes (and NAc’s votes, see Sec. 4.3) through a competition taking place between the leaky neurons of PUT:

$$\tau_{pm} \cdot \dot{\mathbf{pm}}_p = -\mathbf{pm}_p + w_{put-nac-pm} \cdot (\mathbf{put} + \mathbf{nac}) + \mathbf{W}_{pm} \cdot \mathbf{pm} + \mathbf{n} \quad (7)$$

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

where C_v is a coefficient scaling the votes, \mathbf{W}_{pm} are the PM’s lateral connection weights, and \mathbf{n} is a noise component.

When one of the \mathbf{pm} neurons reaches an activation threshold th_A , the execution of the corresponding action is triggered via the MC :

$$\mathbf{m} = \psi[\mathbf{pm} - th_A] \quad (8)$$

where $\psi[x] = 0$ if $x \leq 0$ and $\psi[x] = 1$ otherwise. Once the execution of the routine corresponding to the selected action terminates, the connection weights between VS and PUT, \mathbf{W}_{sc-put} , are modified according to the dopamine signal (this might be null in the case the wrong action has been selected):

$$\Delta \mathbf{W}_{sc-put} = \eta_{sc-put} \cdot \varphi[da - th_{da}] \cdot (s_{lev}, s_{cha})' \cdot \mathbf{m}' \quad (9)$$

where η_{sc-put} is a learning coefficient.

4.3 The amygdala-nucleus accumbens core pathway: a dynamic (S-)S-R action selector

The dynamic action selector learns (S-)S-R associations through a reinforcement learning process that exploits the information encoded as the Amg’s S-S associations (e.g., the ‘lever-hedonic value of food A’ association). In real brains this function might be implemented by the neural pathway connecting the BLA nuclei of Amg to NAc (Baxter and Murray, 2002). In the model this component is implemented as an all-to-all connection matrix $\mathbf{W}_{amg-nac}$ linking the Amg’s hedonic representation of food, \mathbf{amg}_{fA} and \mathbf{amg}_{fB} neurons, to the NAc’s non-leaky neurons:

$$\mathbf{nac}_p = \mathbf{W}_{amg-nac} \cdot (mg_{fA} \text{ } mg_{fB})' \quad (10)$$

$$\mathbf{nac} = \varphi[\tanh[\mathbf{nac}_p + \mathbf{nac}_{baseline}]]$$

NAc’s neurons play the same function as PUT’s neurons, i.e. they represents ‘votes’ that bias the action competition taking place in PM. Similarly to VS-PUT connections, Amg-NAc connections $\mathbf{W}_{amg-nac}$ are modified, after action execution, on the basis of the dopamine signal:

$$\Delta \mathbf{W}_{amg-nac} = \eta_{amg-nac} \cdot \varphi[da - th_{da}] \cdot (amg_{fA}, amg_{fB})' \cdot \mathbf{m}' \quad (11)$$

where $\eta_{(amg-nac)}$ is the learning rate coefficient. Note that in the experiments reported in Sec. 5. the lesions of rats’ BLA have been simulated by setting the Amg-NAc connections to zero.

The importance of the Amg-NAc dynamic action selector resides in the fact that its ‘votes’ for the various actions can be *modulated on the fly* by the system’s motivational states, e.g. by satiety for either one of the two foods. In general, these mechanisms opens’ up the possibility for the motivational-sensitive Pavlovian system (mainly the Amg in the model) to exert a direct effect on actions without the need to pass through re-learning processes, as it will be exemplified by the devaluation experiments illustrated in the next section.¹

¹The model’s parameters were set as follows: $\eta_{amg} = .015$,

5. Results

This section describes the basic functioning of the model on the basis of Fig. 2 which shows the activations of various neurons related to the lever during both the training and testing phases of an experiment run with a non-lesioned simulated rat (the chain-related data, omitted to save space, are qualitatively similar).

At the beginning of the training phase, the baseline activations of PUT and NAc (put_{lev} , nac_{lev}), together with noise, are sufficient to occasionally trigger the execution of an action at the level of the competition taking place in PM (pm_{lev}). When the behavioral routine corresponding to the selected action is appropriate for the environment configuration ('lever press' in the presence of lever), the dispenser becomes yellow, the rat approaches it and consumes the corresponding food (s_{fA}). The food consumption activates both the internal hedonic representation of food in Amg (amg_{fA}) and neurons in VTA-SNpc ($vta-snpc$) with a consequent release of DA in PUT which drives the learning of the cortex-putamen instrumental pathway.

Figure: Activations of the system

The effect of these events is that after a few learning trials the model learns to reliably and fastly perform the action which is appropriate to the current context. The progress of learning can be seen in terms of the increase of PUT's votes for the press lever action (put_{lev}) in the trials in which the lever is present and in terms of the increase of the regularity of the peaks of the food A amygdala neurons (amg_{fA}), of the DA release in VTA-SNpc ($vta - SNpc$), and of the trials' duration.

When the instrumental S-R association begins to be formed due to the instrumental learning process, the vision of the neutral stimuli of the lever starts to be reliably followed, within a relatively small time interval, by the food perception and the consequent DA release. This contingency and the DA signal allow the Pavlovian learning taking place within Amg to 'take off' and form S-S associations between the lever and Amg's food A representation. This is evident from the fact that after a few successful trials the amg_{fA} neuron's activation does not show only a peak when the food A is delivered, but is pre-activated by the simple presence of the lever: this demonstrates the acquired link between the unconditioned (food) and the conditioned (lever) stimuli. The pre-activation of the amg_{fA} neuron due to the perception of the conditioned stimulus is responsible

$\eta_{amg-nac} = .02$, $\eta_{sc-put} = .02$, $th_{DA} = 0.6$, $th_A = .6$, $da_{baseline} = 0.3$, $nac_{baseline} = .3$, $put_{baseline} = .3$, $\tau_{sc} = 500ms$, $\tau_{amg} = 500ms$, $\tau_{tr} = 1000ms$, $\tau_{da} = 50ms$, $\tau_{pm} = 500ms$, $C_{amg} = 50ms$, $w_{put-nac-pm} = .5$, $w_{amg-da} = .3$, $w_{ppn-da} = .6$, $w_{pm} = \begin{pmatrix} 1 & -0.5 \\ -0.5 & 1 \end{pmatrix}$. The model's equations were integrated with a 50 ms step.

of the early DA release in the $VTA-SNpc$, which anticipates the future delivery of the reward and which constitutes a very important and well-known phenomenon in real animals (Schultz, 2002).

The last important learning phenomenon takes place in the amg-NAc pathway. The rat's consumption of food A activates both Amg's hedonic representation of it (amg_{fA}) and, via the , the VTA-SNpc, which results in a strong DA signal. This creates a strong association between the hedonic representation of food and the last executed action (here for simplicity M activations were used as the output of both PUT and NAc: in the future we plan to use eligibility traces instead). The key point here is that once the S-S associations are formed in the Amg, conditioned stimuli such as the lever can trigger the activation of the Amg's hedonic representation of the related food and, via these, influence action selection via NAc. This is shown by the fact that, after some training, NAc starts to be activated and to vote for the correct actions (nac_{lev}). The importance of the formation of this Stimuli-Amg-NAc-PM pathway resides in the fact that it constitutes the fundamental bridge between the the pavlovian processes happening in the amygdala and the instrumental processes happening in the pathway involving basal ganglia (cortex-dorsal striatum-putamen-thalamus-frontal cortex). We argue that this pathway plays a central role in the flexibility demonstrated by real organisms. In particular, it is through this pathway that instant motivational manipulations that characterize Pavlovian conditioning are able to affect instrumentally learned behaviors, as in the devaluation experiments now illustrated.

During the first and second halves of the test phase, the satiety of respectively food A or B are kept at one, i.e. at their maximum level (the other satiety level is kept at zero as in the training phase). This satiety for a food causes a strong inhibition to the Amg's hedonic representation of corresponding food: both the direct consumption of that food and the perception of the conditioned stimulus previously associated with it, fail to elicit the related Amg hedonic reaction. This is visible in the lack of amg_{fA} activation during the second test phase when the rat is satiated with food A.

The perception of both the lever and the chain leads PUT to 'vote' for both the lever press and chain pull actions at the same time. This rules out the influences of the S-R instrumental pathway on action selection: this experimental condition was precisely designed to stop the effects of habits that would otherwise 'mask' the motivation-sensitive Pavlovian influence on action selection. On the other hand, satiation stops only one of the two influences of the Amg-NAc pathway on action selection in that it inhibits only the amygdala representation of the con-

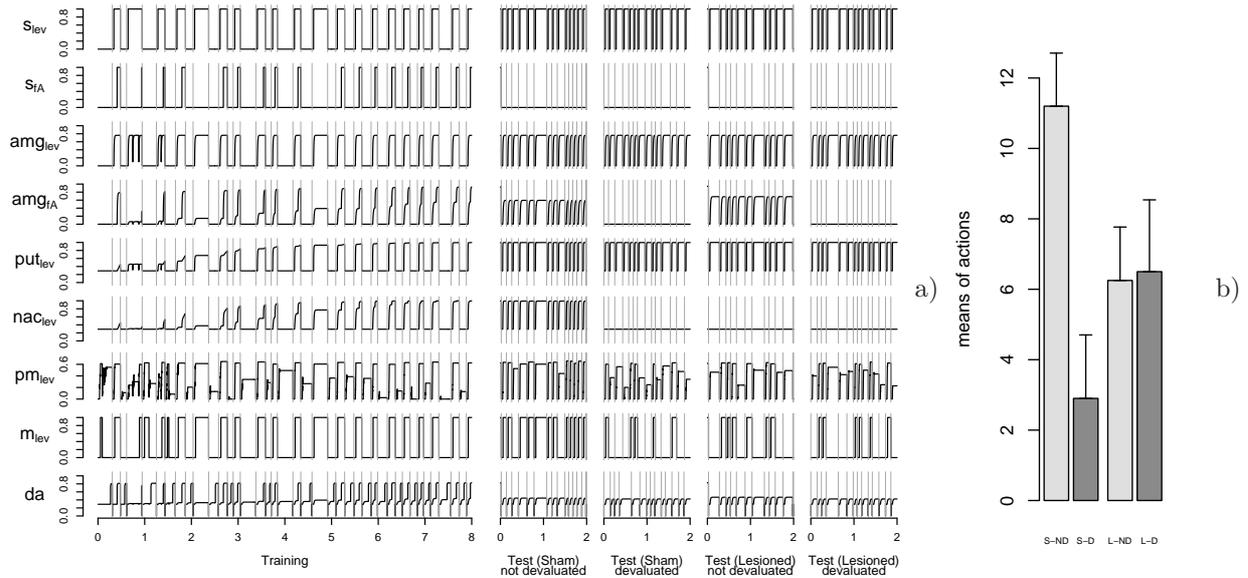


Figure 2: a) Activations of some key neurons during the whole duration of a non-lesioned rat experiment. Trials are separated by short vertical lines; the test phase is divided into two sub-parts where the rat was satiated either with food A or B. on the right the test phase of a lesioned rat is presented. b) means of of action production (S-ND: sham non-devaluated; S-D: sham devaluated; L-ND:lesioned non-devaluated; L-D: lesioned devaluated)

ditioned stimulus which has been satiated (compare the NAc_{lev} activation in the two test phases). The fact that the Amg-NAc pathway ‘votes’ only for the action associated with the non-satiated food breaks the symmetry and makes the related action reliably win the competition in PM (compare the pm_{lev} and m_{lev} activations in the two test phases).

The comparison between the lesioned and non-lesioned conditions reproduces the basic finding of the target experiment by Balleine and colleagues and confirms the aforementioned interpretation of the devaluation tests. During the two minutes of test, non-lesioned (SHAM) rats perform the action associated to the non-devaluated (ND) food with an average of 11.20 times while they perform the action associated to the devaluated (D) 2.9 times in average ($t = 15.7003, df = 19, p - value < 0.001$). On the contrary, BLA-lesioned (LESIONED) rats select actions randomly: the averages of performed action associated with the non-devaluated and the devaluated foods are 6.25 and 6.5 ($t = -0.4346, df = 19, p - value > 0.05$), respectively (see Fig. 2b). In other words, as it happens with real rats, a lesion to the BLA pathway linking the amygdala to the NAc prevents the devaluation of a food from having any effect on the action selection process (see Fig. 2a). The current model provides an existence proof which supports the idea that this Amg-(BLA)-NAc pathway can bridge the Pavlovian processes happening in the amygdala and

the instrumental processes happening in the cortex-basal ganglia pathway, allowing animals’s action selection mechanisms to be modulated *on the fly* by the current state of the motivational system.

6. Conclusions

This paper presented an embodied model of some important relations existing between Pavlovian and instrumental conditioning. The model’s architecture and functioning has been constrained with relevant parts of current neuroscientific knowledge on the brain structures underlying such processes. The model was validated by successfully reproducing the primary outcomes of some instrumental conditioning devaluation tests conducted with normal and amygdala-lesioned rats. These tests are particularly important as they show how the sensitivity to motivational states exhibited by Pavlovian responses can transfer to instrumentally acquired behaviors.

To the best of the authors’ knowledge, the model represents the first attempt to propose a comprehensive interpretation of the aforementioned phenomena, tested in an embodied model. The works most closely related to this one are those of Dayan and Balleine (2002), Morén and Balkenius (2000), and O’Reilly and Watz (2007). The model presented here differs from these works in that it proposes an embodied model (absent in all mentioned researches), presents a fully developed model (Dayan and Balleine (2002) presented only a ‘sketched’

model), and tackles the issue of the relations existing between Pavlovian and instrumental conditioning (Armony et al. (1997) , Morén and Balkenius (2000) and O'Reilly and Watz (2007) focussed only on Pavlovian conditioning).

We are aware that the proposed model is limited under many respects which will be tackled in future work. First of all, it has been tested only with a simple embodied model having simple sensors and relying on hardwired low-level behaviors. Second, it has several limitations with respect to well-known biological phenomena: for example, it does not learn to inhibit the dopamine error signal at the onset of the USs, as it happens in real organisms (Schultz (2002); this prevents it to perform extinction and to stop updating weights, O'Reilly and Watz (2007)), it cannot reproduce Pavlovian modulation of the vigor with which instrumental actions are performed, and it does not model the triggering of innate actions by Pavlovian conditioning (Dayan and Balleine, 2002).

Notwithstanding these limitations, we think that the proposed model represents an important step in the construction of an integrated picture on how animals' motivational systems can both drive instrumental learning and directly regulate behavior. Constructing such a picture is of paramount importance not only from the scientific (i.e. psychological and neuroscience) point of view, but also from the technological one. In fact, it might suggest us fundamental design principles for endowing future robots with the behavioral flexibility that characterizes living organisms.

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