Computing Dual-Route Fear-Conditioning Circuits using Audio Signals

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Abstract—This paper looks into the dual route model of fear conditioning using gammatone filter-bank for auditory pre-processing, in order to extract contiguous frequency bands from auditory stimuli, as seen in auditory nerves. The model focuses on two routes i.e., the cortical pathway using the auditory cortex as a messenger between auditory thalamus and the amygdala & the sub-cortical pathway where, the amygdala has direct input from the thalamus. Several lesion experiments were simulated on the model to explain the differences between the two routes. But in the same time it has been identified that a requirement of a more biophysical model exists to capture more of the functional differences that exists between these two routes.

I. INTRODUCTION

The classical view of fear conditioning has come a long way to explain the neural correlates associated with the storage and attainment of information about affective significance of events and stimuli. The neural correlates of emotional processing have been studied extensively in experimental animals and quite recently in humans using the classical Pavlovian fear conditioning phenomenon [1]. In this a meaningless stimuli (here a pure tone) elicits fear responses (e.g., suppression of ongoing activity, increase in heart rate, hormonal activity, elevation of blood pressure, etc) after it occurs in connection with an aversive shock (Fig. 1). Our work here explains such a model constrained by the anatomical and physiological findings [2].

A lot of evidence support the fact that amygdala, a small structure in the temporal lobe, is the governing body in the acquisition and expression of conditioned fear responses. Lateral nucleus in the amygdala receives the input from all the main sensory systems and higher association areas in cortex and hippocampus. Thalamus on the other hand sends direct sensory projection into the amygdala from its extra-lemniscal areas. The direct projection occurs from the medial division of the medial geniculate body (MGB) and the posterior intralaminar nucleus (MGm/PIN) to the amygdala. It also sends information to the amygdala via the indirect cortical pathway using the primary and secondary auditory cortices. It has been validated through our simulations that the direct route between the thalamus and the amygdala seems to be more important in...
learning relatively crude conditioned stimulus (CS) - unconditioned stimulus (US) associations, whereas the indirect route from thalamus via the cortex to the amygdala seems necessary for learning more detailed and specific associations. The signals transmitted using the direct pathway are quick but limited in the information content because the thalamic cells are not very precise stimulus discriminators whereas, the cortical pathway is slower but a means for providing robust information about the stimuli. It has been suggested by LeDoux [1] that the subcortical pathway is used to provide a ‘quick and dirty’ representation of the stimuli which prepares and primes the amygdala to receive the information from the cortex.

II. Pre-processing of auditory information using gammatone filterbank

The easiest way for transducing sound is through microphone, though less sophisticated than the techniques used by mammals. We compensated this problem by processing the electrical signal after the process of transduction. We start by using logarithmically spaced pure tones as inputs to the bandpass filters with near constant Q (Eqn. 1, MinBW is the minimum bandwidth for low frequency channels, having a value of 24.7 for humans) to model the effect of cochlea and the organ of Corti. We start by using Patterson’s Auditory Image Model [3], which arises from the interactions associated with the adaptation and suppression observed in the auditory nerve. This also facilitates computation of phase alignment & temporal integration which take place before the formation of the auditory image. The modeling protocol used is identified in Fig. 2.

Here, we emphasize on the cochlear simulation stage which consists of two phases: first, spectral analysis is performed on the acoustic wave which converts it to a multichannel representation of basilar membrane motion (BMM) and second, a two dimensional adaptation is used which converts this BMM into a multi-channel representation of the neural activity pattern. A gammatone filter can be defined in time domain by its impulse response.

\[
ERB(f_c) = \left(\frac{f_c}{Q}\right)^p + (\text{MinBW})^p \right)^{\frac{1}{2}} \tag{1}
\]

\[
g_t = a t^{n-1} \cos(2 \pi f_c t + \phi) e^{-2 \pi b t} \tag{2}
\]

\[
ERB = 24.7(4.37f_c/1000 + 1) \tag{3}
\]

The frequency selectivity of the hearing mechanism is modeled using a 6th order IIR gammatone filter (defined by Eqn. 2 & Eqn. 3) with both poles & zeros. It can be derived from Slaney’s Auditory Toolbox [4] using the parameters defined by Patterson & Holdsworth [3]. The solution for the 6th order variables is shown in Eqn. 4 (refer Fig. 3). The coefficients are computed by Function 5 & Function 6.
Fig. 3. This represents the frequency response of the 6th order gammatone filter bank consisting of 16 channels with a 44.1 KHz sampling rate. The center frequencies are placed equidistant with a gap of 300 Hz in the range of 300 Hz - 4.8 KHz. The figure below is just the output when the 3 KHz tone is fed through a 16 channel gammatone filter bank followed by some non-linearities, and a sliding temporal interaction. The last figure is simply the response of the tone after the gammatone filter bank stage.

\[ T_{sim} = \frac{az e^{\phi - 2\pi b - 2i\pi f_c}}{(z - e^{-2\pi (b+i\phi)})^6} \times \\
( z^4 + 26 z^3 e^{-2\pi (b+i\phi)} + 26 e^{-6\pi (b+i\phi)} z \\
+ e^{-8\pi (b+i\phi)} + 66 z^2 e^{-4\pi (b+i\phi)}) \]

\[ A = (b, f_c, \phi, a) \rightarrow [0, ae^{\phi - 2\pi b - 2i\pi f_c}, 26 ae^{\phi - 2\pi b - 2i\pi f_c} e^{-2\pi (b+i\phi)}, 66 ae^{\phi - 2\pi b - 2i\pi f_c} e^{-4\pi (b+i\phi)}, 26 ae^{\phi - 2\pi b - 2i\pi f_c} e^{-6\pi (b+i\phi)}, ae^{\phi - 2\pi b - 2i\pi f_c} e^{-8\pi (b+i\phi)}, 0, a7, a8] \]

\[ B = (b, f_c, \phi, a) \rightarrow [1, -6 e^{-2\pi (b+i\phi)}, 15 (e^{-2\pi (b+i\phi)})^2, -20 (e^{-2\pi (b+i\phi)})^3, 15 (e^{-2\pi (b+i\phi)})^4, -6 (e^{-2\pi (b+i\phi)})^5, (e^{-2\pi (b+i\phi)})^6, b7, b8] \]

III. CONNECTIONIST MODEL

Our goal here was to bridge the gap between neural activity and the emergent behavior. Connectionist computational model help us to understand this consistency of the theoretical idea driving electrophysiological and biophysical models. Our model is described in Fig. 4. The architecture is very modular pertaining to the anatomical connectivity in the brain. The basic computing elements are threshold-type non-linear units. The output of these are assumed to behave similar to the time-averaged firing rate of the neuron. These units are organized into MGv, MGm, auditory cortex & the amygdala, which is the output layer containing the behavioral response.

The input layer consists of contiguous frequency pure tones pre-processed using a gammatone filterbank. One of the frequency of the dataset serves as the CS during the conditioning run. The US is simply a positive quantity added to the units of amygdala & MGm/PIN during conditioning phase. The US was externally set & was not subject to the learning algorithms. The units arranged in different modules attain receptive fields (RF) after the training process. Each unit develops its own RF and responds to only a set of contiguous input stimuli, centered around a best frequency (BF). The relative width of the RF is set for each module by adapting the lateral inhibition connections [5]. This was used to model the broad RF of the MGm
& amygdala and the narrow RF of the MGv & the auditory cortex.

The connections between the modules are feed-forward and positive (excitatory) whereas, units within the same module are mutually inhibitory. The output response of the activation units, were assumed to be comparable to the time-averaged firing rate of a real neuron, calculated using a soft competitive learning algorithm [6]. Simply stated, the unit in a module receiving the strongest input is the ‘winner’ and inhibits the other units by an amount proportional to its activation. The values for lateral inhibition are 0.1 (MGm/PIN), 0.3 (MGv), 0.6 (cortex) & 0.4 for amygdala. The weights of the excitatory connections between the modules were modified using the Hebb-Stent rule [7]. The weights of the excitatory connections between the modules were modified using the Hebb-Stent rule [7]. The learning rate of the network which is set to 0.2. The learning in the network continues to a stipulated amount of training cycles.

The increase in response to the CS-US pairing was proportional to the number of training (conditioning) trials but saturated after extended training cycles. First the weights of the network were assigned a random value between 0 & 1. In the development stage, the input frequencies were presented randomly to the network and one of the frequency was chosen as the CS. After that it was paired with the activation of the US. Each unit’s receptive field (RF) was calculated by measuring the activation of that particular unit to each of the input frequencies. All the modules have less units than the number of input patterns.

IV. Results

The pairing of US & CS captured many of the salient features of the conditioning paradigm - some of the threshold units showed frequency-specific retuning of their RF, such that the BF shifted towards the CS (Fig. 5). It was seen that only units receiving convergent CS-US showed retuning of their RF. This process has also been identified by Weinberger in the experiments performed on guinea pigs [8]. As a result, our model captured, to some relevance the experimental result hence minimizing the gap between non-linear units and behavior. Also it was noticed that the decrease in response with respect to the frequency was not monotonic leading to a ‘Mexican hat’ suppression of the side-bands. We also agree that the competitive learning paradigm used in these experiments is not critical for obtaining the results, they seem to arise from the neurobiologically constrained network.

A. Latent Inhibition

Latent inhibition is a way to determine if the network has a lower susceptibility to conditioning to a familiar stimulus compared to an unfamiliar one. The model predicts that both novelty and US could account for fear conditioning response. With a novel stimuli the fear response would be more than that with a familiar one. In one trial, we choose to present the CS for 100 times (Fig. 6) & in the other we present a different CS for 200 times (Fig. 7). The results were striking - conditioning was more...
Fig. 7. Frequency no. 5 was conditioned for 200 epochs.

for low familiar stimulus (frequency 4) than for the more familiar stimulus (frequency 5).

B. Extinction

Here, the repeated presentation of the CS without the US, lead to the extinction of the conditioned response. Repeated presentation of a single stimulus under a learning framework would in principle lead to a higher activation & hence to an improved representation. To test this, we presented CS with all other frequency to the network. The other frequencies were presented twice as often as the CS to ensure interference. The network state that resulted from this was exposed to a random batch consisting the CS and two instances of all other frequencies. The CS showed a decrease in fear conditioning over repeated presentation whereas, the control stimulus lacked such a decrease (Fig. 8). After presenting the stimulus for 15 times the activation approximately converged. Though it is generally argued that extinction is caused by interference but we consider inhibition to be a more robust explanation of this phenomenon. Ref. [9] has shown that after extinction, the lesioning of the indirect route can be used to restore the conditioning phenomenon.

C. Lesion Simulations

Here the cortical lesions didn’t interfere with the acquisition of the conditioned response to the stimuli; also in the presence of other stimulus, the model demonstrated a non-monotonic generalization trend. This was done by setting the connections of the direct and indirect pathways to zero & unmodifiable by the learning rule. We observe that the absence of the auditory cortex with only direct inputs to the amygdala from the thalamus (Fig. 10) is a sufficient condition to induce conditioned fear, thus thalamic pathway has a higher information processing capacity although it has been suggested [2] that the auditory cortex (Fig. 9) may be involved in the processing of complex sound stimuli notably temporal patterns, relative pitch and tone duration.

V. Conclusion

The observations that we make here mainly are based on pure tones, it might be that for more complex stimuli the auditory cortex remains as important as the thalamus for integration of multimodal
stimuli and interactions between the amygdala and higher cognitive networks in the cortex. The cortical lesions didn’t produce any overgeneralization, this is due to the fact that though the individual units code for a range of contiguous frequencies and hence poor “similar stimuli” discriminators, the overall response is determined as the sum of all the units’ response. This is in principle like a ‘coarse population coding’ where the network codes as a better stimulus discriminator compared to an individual unit. Due to the presence of an additional layer, the signals from the thalamus are greatly attenuated, which might be a proposition to explain the reduced conditioning [2].

In the simulations, especially the lesion experiments on the model, it can be suggested that the subcortical pathway, possibly by using population coding, is capable of performing crude stimulus discrimination. It has also been suggested by unit recordings in free behaving rats that much of the plasticity that occurs in lateral amygdala involves thalamic as opposed to cortical input [10]. This had initiated a barrage of work, to account for the processing capacities and limitations of cortical and sub-cortical pathways. Our work demonstrates one of such endeavors. We attained success in showing that a neuroanatomically constrained network with a biologically plausible learning algorithm can capture important aspects of fear conditioning.

It is also wise to point out few limitations that this model has. Here the network is purely feed-forward and hence ignores all the recurrent connections that might exist, which mainly governs the physiological consolidation of plasticity - its strength & duration. This model also overlooks important facts like intracortical laminations & cortico-cortical pathways in sensory modalities and sensory-specific association areas & higher processing areas. Also the model overlooks the temporal facet of stimulus presentation, though physiological receptive fields [8] are consistent with this protocol.

REFERENCES


