Implementing Fear Learning and Classical Conditioning Using a Novel Brain-Inspired Artificial Neural Network

Authors: Wei Dongtao, Wang Guoqing, Wei Dongtao

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Abstract

Neuroscience has served as a significant source of inspiration for artificial intelligence. By drawing upon research findings from this field, we have designed a novel artificial neural network to simulate the amygdala in the human brain. This artificial neural network comprises two components: a long-term memory network and an activation network. The memory network records the neurons that transmit and receive signals, as well as the synaptic weights between them. The activation network, in contrast, records the neurons that transmit and receive signals along with the temporal points at which signals are sent. The activation network retains only a brief memory of events when they occur and modifies the weights in the long-term memory network according to predefined rules. By employing such methods, we have successfully endowed agents with the capabilities of fear emotional learning and classical conditioning learning, which closely resembles the function of the amygdala in biological organisms.

Full Text

Preamble

Realizing Fear Learning and Classical Conditioning with a Novel Brain-Inspired Artificial Neural Network

Dongtao Wei[1]*, Guoqing Wang[2]
(1. Key Laboratory of Cognition and Personality, Ministry of Education, Faculty of Psychology, Southwest University, Beibei, Chongqing, 400715;
2. Kunming, Yunnan, 650000)

Abstract

Neuroscience provides significant inspiration for artificial intelligence. Drawing upon research from these disciplines, we designed a novel artificial neural network to simulate the amygdala in the human brain. The neural network consists of two components: a long-term memory network and an activation network. The memory network records the neurons that send and receive signals along with their connection weights, while the activation network records the sending and receiving neurons and the precise timing of signal transmission. The activation network retains only a brief memory of events and modifies the weights in the long-term memory network according to established rules. Using this approach, we have successfully endowed an agent with the capacity for fear emotional learning and classical conditioning learning, which closely resembles the functions of the amygdala in biological organisms.

Keywords: brain-inspired artificial neural network; amygdala; fear; fear conditioning; SDTP

Author Biographies:

Corresponding Author: Dongtao Wei, Ph.D., Associate Professor. Research interests include the neural basis of anxiety and fear emotions and their computational simulation (E-mail: dongtao@swu.edu.cn).

Co-first Author: Guoqing Wang, M.S., graduated from the Faculty of Psychology at Southwest University. Current research focuses on the integration of neuroscience and artificial intelligence (E-mail: wgg_{wgq}@163.com).

The human brain contains approximately 86 billion neurons [1], each performing simple operations: receiving information, making "decisions" according to simple rules, and transmitting information outward. Yet it is precisely these simple neurons, organized into neural networks, that endow humans with diverse capabilities such as emotion, creativity, language, and thought.

In artificial intelligence research, constructing artificial neuron models analogous to biological neurons and organizing them into artificial neural networks (ANNs) to achieve intelligence represents an important direction. The earliest attempt to simulate biological neurons on computers was made by neuroscientist McCulloch and mathematician Pitts, who in 1943 proposed the original artificial neuron model [2] (the MP model). The MP model and its subsequent variants [3,4] simulated many important characteristics of biological neurons, such as the ability to receive and send signals, the influence of connection strength between neurons, and the modifiability of these connections. However, regarding how connections between neurons change, artificial neural networks differ substantially from biological neural networks. In ANNs (specifically supervised learning types), weight modifications aim to reduce the error between predicted and actual outcomes [5], an algorithm independent of temporal factors. In contrast, weight regulation in biological neural networks is closely related to temporal factors [6-9]. For instance, when a neuron fires at a particular time, it affects

synapses connected to it that are in an active state. If synaptic activation precedes neuronal firing, that synapse is strengthened; conversely, it is weakened. This phenomenon is known as Spike-Timing-Dependent Plasticity (SDTP) [9]. Spiking neural networks (SNNs), proposed in 1997 [10], incorporate temporal elements. In SNNs, neurons transmit discrete signals containing temporal information, thereby possessing the capacity to simulate SDTP [11-16].

Both ANNs and SNNs emphasize the similarity between artificial and biological neurons while overlooking similarities at the neural network level. However, intelligence differences across species likely stem from variations in neural network architecture [17,18] rather than neuronal differences. Here we propose a novel neural network representation that emphasizes similarity to biological brain networks. Using this approach, we simulated a specific neural structure in the brain—the amygdala.

Multiple lines of evidence indicate that the amygdala serves as the center for fear emotional memory and fear classical conditioning learning. For example, presenting threatening stimuli activates the amygdala, whereas amygdala damage eliminates fear responses [19,20]. Normal monkeys typically show timidity toward snakes, but monkeys with amygdala damage do not [21]. In humans, patients with amygdala damage not only have difficulty recognizing fearful expressions [22] but also show impaired fear classical conditioning learning [23]. Neuroimaging studies reveal that fear classical conditioning typically requires amygdala activation [24].

We found that agents equipped with this neural network architecture can exhibit fear emotional learning and classical conditioning capabilities, closely resembling amygdala function.

2.1 Simulation Scenario and Agent Design

To simulate an agent in a realistic scenario, we first designed a simulation environment using Python' s tkinter package. The scenario consists of a 12$×$12 grid, with each cell representing 50 pixels.

The agent's body is a 30$×$30 pixel rectangle equipped with four directional tentacles that serve as sensory organs, enabling perception of object colors within a one-cell radius.

Two objects are implemented: a red threat and a gray neutral object. The threat damages the agent only when occupying the same cell as the agent' s body, whereas the neutral object never causes damage. Similar to real-world conditions, the agent must learn through experience that the threat can cause harm.

When the agent experiences damage or perceives a threat, it flees, jumping three cells per escape movement.

[Figure 1: see original paper] Agent, threat, and neutral object configuration

2.2 Agent Neuron and Neural Network Design

(1) Amygdala Characteristics and Neural Connections

In this experiment, we simulated a specific brain structure—the amygdala—which plays a crucial role in fear emotional learning and classical conditioning [25-27].

The amygdala comprises approximately 12 nuclei, with three most relevant to fear [28,29]: the lateral amygdala, basal nuclei, and central nucleus. The lateral amygdala connects with all cortical areas, receiving sensory information [29-32], and projects to the basal nuclei, which in turn project to the central nucleus [25,33]. The central nucleus executes emotional responses, projecting to lower brain regions including the hypothalamus, ventral tegmental area, and brainstem locus coeruleus. These regions are associated with autonomic components (e.g., sympathetic nervous system), behavioral components, and hormonal components. The behavioral component mediates fear responses (freezing, escape), while autonomic (elevated blood pressure, increased heart rate) and hormonal (adrenaline secretion) components support these behaviors [26], as shown in [Figure 2: see original paper].

[Figure 2: see original paper] Simplified diagram of amygdala neural connections

(2) Learning Processes in the Amygdala

Synapses in biological brains are modified by their activity, changes widely considered fundamental to learning and memory. A notable characteristic of activity-induced synaptic modifications is temporal asymmetry: when a neuron is active, it simultaneously strengthens synapses that were activated earlier and weakens those activated later [6-9], constituting SDTP.

Although direct evidence for SDTP-like learning in the amygdala remains inconclusive, experiments show that fear learning and fear classical conditioning typically strengthen synaptic connections between sensory neurons and lateral amygdala neurons [34-38], indicating a close relationship.

(3) Amygdala Neural Network Model

As noted above, considerable evidence links strengthened synapses between sensory and amygdala neurons to fear and classical conditioning learning [34-38]. However, no existing model explains why this relationship emerges.

Numerous models of classical conditioning exist [39-41], attempting to unify experimental phenomena through mathematical frameworks. Yet without placement within a specific neural network, these models struggle to explain how synaptic changes produce specific behavioral modifications. Here we propose a novel model to explain the relationship between amygdala synaptic changes and behavioral outcomes.

Principles of Fear Learning

Fear emotion has played a crucial role in biological evolution. Through fear

learning, organisms develop escape responses to previously harmful objects, reducing the likelihood of repeated attacks and increasing survival probability.

To illustrate our amygdala model, consider a typical fear learning scenario: A newborn monkey with no prior snake experience sees a snake without fear or escape response, is subsequently attacked, experiences pain, and produces an avoidance reaction (escape).

Through this event, learning occurs. The result is that upon subsequent snake encounters, the monkey experiences fear and immediately escapes, avoiding further attacks.

The following model explains this scenario, as shown in [Figure 3: see original paper]. [Figure 3: see original paper] Fear learning neural network model

The left panel shows the amygdala network before learning; the right panel shows the network after learning. t1 represents when the monkey' s visual system perceives the snake, activating the sensory pathway to the amygdala (pathway α). Since this pathway is unstrengthened, sensory neurons cannot activate the amygdala, preventing activation of pathway γ and escape responses. t2 represents when pain is perceived, activating the pain-to-amygdala pathway (pathway β). Pain typically triggers strong amygdala firing [42], so we assume this pathway can initiate escape responses without prior strengthening. t3 represents when pathway γ activates, causing the monkey' s escape.

Typically, the monkey sees the snake before being attacked, establishing a temporal sequence: t1 precedes t2, which precedes t3. The interval between t1 and t2 represents the time from snake detection to attack. Since both visual and pain neurons project to amygdala neurons, amygdala activation according to SDTP rules strengthens the earlier-activated pathway α, producing learning.

After learning, pathway α is strengthened. When the monkey' s visual system is again activated by a snake, it triggers amygdala activation and subsequent escape, preventing further attacks. Therefore, t2 in the right panel of [Figure 3: see original paper] is a hypothetical time point (marked with a red box). The interval between t3 and t1 represents the time from snake perception to escape initiation, while t2-t1 represents the objective time from seeing the snake to being attacked. Typically, t3-t1 is shorter than t2-t1, demonstrating that fear learning enhances survival probability.

Principles of Classical Conditioning

Fear classical conditioning experiments typically employ a conditioned stimulus (CS) and unconditioned stimulus (US). Before training, the CS does not elicit escape responses, whereas the US does. During training, the CS and US are paired repeatedly, after which the organism learns to escape upon CS presentation.

[Figure 4: see original paper] Fear classical conditioning

Similar to neuronal STDP, behavioral studies reveal that timing is critical in fear classical conditioning. The CS must precede or coincide with the US; if the CS follows the US, organisms never learn the conditioning [43]. Some researchers suggest the CS serves a warning function [44]. Thus, classical conditioning closely resembles fear learning, with the difference that both US and CS are sensory information, so model signals originate from sensory neurons. As shown in [Figure 5: see original paper], for classical conditioning, different activation times involve sensory information, where S1 is the CS and S2 is the US.

[Figure 5: see original paper] Classical conditioning neural network model

Habituation and Classical Conditioning Extinction

In the real world, both fear learning and classical conditioning are dynamic and reversible.

Habituation, the opposite of fear learning, describes the process by which organisms gradually adapt to harmless stimuli and reduce escape responses. In our fear learning context, habituation refers to the gradual disappearance of escape responses to an object that previously caused harm but now appears repeatedly without causing damage.

Extinction, the opposite of fear classical conditioning, occurs when a CS is presented alone multiple times without the subsequent harmful US, causing the organism to gradually lose escape responses to the CS.

Both effects likely result from decreased neural pathway weights, opposite to previous strengthening.

Amygdala Neural Network Model

Integrating the principles of fear learning and classical conditioning with amygdala connectivity, we designed an amygdala-like neural network model, as shown in [Figure 6: see original paper].

[Figure 6: see original paper] Fear memory network design

All sensory neurons project to the amygdala, including pain neurons. Unlike other sensory neurons, pain neuron connections to amygdala neurons are stronger (represented as solid lines). Additionally, amygdala neuron activation directly triggers escape responses.

(4) Neuron and Neural Network Implementation

To simulate agent behavior in realistic environments, we first modeled the agent' s perception.

Agent Perceptual Simulation

The agent possesses two sensory capabilities. First, it uses tentacles to perceive object colors, meaning that when either the threat or neutral object contacts a tentacle, it activates the agent' s sensory neurons.

Second, it perceives pain. When the threat occupies the same position as the agent' s body, the agent experiences damage, activating pain sensory neurons.

In the human brain, memory is categorized by duration into sensory memory, short-term memory, and long-term memory [45]. Sensory memory (also called sensory register) has an extremely brief retention time (approximately 50ms for visual sensory memory [46,47] and 1-2s for auditory sensory memory [48,49]) but can hold substantial information. Most sensory memories are discarded after exceeding their retention period; only attended information undergoes further processing to enter short-term memory and potentially long-term memory. Inspired by this, we implemented two networks: a long-term memory-like network (called the fear memory network) and a sensory memory-like network (called the activation network). The fear memory network maintains stable long-term memories, while the activation network stores immediate sensory memories during events.

Fear Memory Network Design

The fear memory network simulates the stable neural network in an organism' s brain before experiencing events. As shown in [Figure 6: see original paper], an amygdala-like network includes amygdala neurons, sensory neurons (including pain neurons), and escape responses triggered by amygdala activation.

We set initial connection weights: pain neuron-to-amygdala projections at 3, and other sensory connections at 1.

We used Python' s pandas package to describe the memory network, including three components: sending neurons, receiving neurons, and connection weights. Initially, we only established connections between pain neurons and amygdala neurons.

We employed a special method to represent other sensory projections to amygdala neurons: when the agent perceives a stimulus, it first checks the memory network for an existing stimulus-to-amygdala pathway. If none exists, indicating novel stimulation, the pathway is added to the memory network with a default weight of 1, as shown in [Figure 7: see original paper].

[Figure 7: see original paper] Fear memory network representation

We set the amygdala neuron activation threshold at 1.5. When received signals exceed this threshold, escape responses are triggered. Otherwise, no response occurs.

In biological brains, neuronal signals vary in intensity, with stronger stimuli eliciting higher action potential frequencies. However, evidence suggests intensity differences do not differentiate objects; instead, the brain uses distinct neurons to label different objects. For example, some experiments found that for the same object, attention enhances firing in higher-level visual cortical areas (V4) [50]. Additionally, many studies show different object categories are stored in

distinct temporal lobe regions [51-57], indicating that firing frequency is less related to object identity.

For these reasons, we ignored signal intensity in this experiment. For receiving neurons, activation depends solely on connection weights. Therefore, without learning, only pain neuron activation can trigger amygdala activation and escape responses.

Activation Network Design

We implemented an activation network to simulate what occurs when an organism encounters an event.

The activation network records immediate signals entering the agent' s "brain," documenting three aspects: sending neurons, receiving neurons, and signal initiation times. The activation network is empty without stimulation; upon signal entry, it records the event ([Figure 8: see original paper]).

[Figure 8: see original paper] Activation network representation

Neural Network Modification Rules

Weight modification rules in this network draw upon SDTP principles. In the activation network, if the interval between two signals is small and the later signal activates the amygdala, it strengthens connections between the earlier signal and the amygdala.

We assume the strengthening rule is: (Formula 1)

where t is the interval between preceding and subsequent signals in the activation network, and weight(pre) is the pre-learning weight in the memory network.

The time interval defines the reinforcement window, which we set at 200ms—much longer than biological windows [9]. Note that this formula and window are not fixed and can be modified according to specific scenarios. In our simulation, agent movement requires refreshing every 100ms; for display convenience, we used a longer window.

For habituation, we assume the rule: (Formula 2)

For classical conditioning extinction, we assume the rule: (Formula 3)

Parameter Design and Significance

We fixed two types of neural connections: pain neuron-to-amygdala connections and amygdala-to-escape response connections. These represent innate neural connections in the agent' s "brain," indicating unlearned, reflexive responses.

The amygdala activation threshold represents pain tolerance. Lower values indicate lower pain tolerance, producing faster learning and more escape responses, which increases survival probability but reduces working time due to excessive avoidance.

The interval time (t) in Formula 1 defines the reinforcement window, presenting a trade-off: longer windows enable neural strengthening across broader temporal ranges but consume more memory, while shorter windows save resources but may prevent learning real-world CS-US contingencies.

Both formulas and parameters are not entirely fixed; agents can modify them based on operational contexts to optimize the trade-off between survival and functional efficiency.

2.3 Signal Processing in the Neural Network

When the agent' s perception changes, the pathway between sensory and amygdala neurons is recorded in the activation network through a process called "sensory registration."

Following sensory registration, the agent consults the memory network based on recorded pathways. If a matching pathway exists, its weight is retrieved. If the weight exceeds the threshold, the appropriate response is executed. If not, the pathway is added to the memory network with a default weight of 1.

Each perceptual change triggers this process once, constituting "one cycle."When the entire process concludes and perception remains unchanged for an extended period, the period from initial change to process completion is called "one event." After each event, the agent modifies memory network weights according to adjustment rules and clears the activation network, as shown in [Figure 9: see original paper]. Blue arrows represent processing within one cycle; orange arrows represent post-event processing.

[Figure 9: see original paper] Signal processing in the neural network

[Figure 10: see original paper] illustrates changes in activation and memory networks throughout the process.

[Figure 10: see original paper] Signal changes in memory and activation networks at different stages (fear learning example)

3 Experimental Results

This section presents results for fear learning, classical conditioning, and extinction effects. Video files and code are available in supplementary materials.

3.1 Fear Emotional Learning

Pre- and post-learning effects are shown in [Figure 11: see original paper]. Comparing agent responses across stages reveals that pre-learning agents do not avoid the red threat. After attack-induced learning, agents develop escape responses, preventing further harm.

[Figure 11: see original paper] Agent performance before and after fear emotional learning

3.2 Classical Conditioning Learning

Once agents learn to escape the red threat, it becomes the unconditioned stimulus (US), while the gray neutral object serves as the conditioned stimulus (CS).

[Figure 12: see original paper] shows behavioral changes before and after classical conditioning. Pre-learning agents do not escape the gray neutral object. However, when the neutral object consistently precedes the threat, satisfying classical conditioning requirements, post-learning agents develop escape responses to the neutral object.

[Figure 12: see original paper] Agent performance before and after classical conditioning learning

3.3 Habituation and Classical Conditioning Extinction

[Figure 13: see original paper] shows habituation results, demonstrating that habituation eliminates escape responses to the threat.

[Figure 13: see original paper] Agent performance before and after habituation

[Figure 14: see original paper] shows classical conditioning extinction results, revealing that post-extinction agents lose escape responses to the CS (neutral object) while retaining responses to the US (threat).

[Figure 14: see original paper] Agent performance before and after classical conditioning extinction

4 Discussion

In this experiment, we first proposed a model explaining the relationship between amygdala neuronal changes and behavioral modifications. Based on this model, we designed a novel neural network that simulates amygdala structure and function, enabling fear emotional learning and classical conditioning. Unlike conventional ANN and SNN frameworks, we emphasize similarity to the human brain—a factor we believe should not be overlooked in neural network design.

Compared to other primates, the human brain' s neuron-to-body ratio is not exceptional [1]. What accounts for human intelligence? Evidence suggests differences arise from neural network architecture [17,18], with more intelligent species possessing more complex networks. For instance, primates have significantly higher proportions of supragranular layer (II and III) neurons in the neocortex compared to carnivores and rodents [18]. Supragranular layers primarily project signals to other cortical areas, indicating that primate brains contain more neurons for inter-regional connectivity, forming complex networks. Some researchers propose this as the source of advanced cognitive abilities in primates, including humans [60,61]. Another widely accepted principle is that neural networks determine the meaning expressed by individual neurons [62]. While sig-

nals from different brain regions all manifest as axonal action potentials, the same signals in different networks express different meanings—for example, V1 neuron firing may represent line orientation [63], MT cortex firing may represent motion direction [64], and temporal lobe neuron firing may represent face detection [65]. These facts underscore the importance of neural networks in implementing intelligence.

In biological neural networks, time is another crucial factor [6-9]. Neurobiological experiments demonstrate that timing critically influences synaptic connections: if a neuron fires, it strengthens synapses activated earlier and weakens those activated later [9]. We therefore recorded neuronal firing times and simulated synaptic changes by comparing activation times across different neural pathways. This resembles SNN capabilities [10], but unlike membrane-level neuronal simulations, we ignored signal transmission forms between neurons, representing them with numerical values—similar to the MP model [2].

Our network design incorporates sensory registration. Psychological research reveals extremely brief memory retention for different sensory systems [46-49], termed sensory memory or sensory registration. Only attended sensory information enters short-term memory and may be further processed into long-term memory, while unattended information is discarded [45]. Sensory registration allows the brain to retain external stimuli temporarily and adjust synapses based on subsequent feedback, producing memory and learning. This approach is particularly suitable for organisms processing unknown environments. In contrast, computer science conditional statements also enable different actions based on conditions, but require designers to anticipate scenarios. In open-ended environments with nearly infinite possibilities (e.g., exploration on an unfamiliar planet), our method proves highly practical.

Agent learning is also cross-modal. In our network, learning is not restricted to specific sensory pathways. Regardless of sensory modality, as long as sensory neurons project to the amygdala and satisfy network modification rules, fear emotional learning and classical conditioning can occur.

Our designed neural network involves minimal computation. Agent learning primarily involves memory and weight modification, requiring low computational power—similar to the brain. The human brain processes information with remarkably low energy consumption, leading some researchers to hypothesize that the brain is not a calculator but a memory and prediction system [66]. Our experiment supports this hypothesis.

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Author Contributions:
Dongtao Wei: Conceptualization, methodology, final manuscript revision;
Guoqing Wang: Conceptualization, methodology, software implementation, original draft preparation.

Note: Figure translations are in progress. See original paper for figures.