The Effect of Modification and Inhibitory Scale Factors on Learning Performance in Relation to Trace Conditioning

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Abstract

Trace conditioning is a widely studied form of associative learning in which a neutral stimulus (conditioned stimulus or CS) is paired with an aversive or rewarding stimulus (unconditioned stimulus or US) after a brief interval. Computational neuroscience approaches have been used to investigate the mechanisms underlying trace conditioning, and the role of inhibitory processes in shaping neural activity and plasticity has been of particular interest.

This study used a neural network model to investigate the impact of inhibition on trace conditioning across different time scales. The results showed that the relationship between inhibition and successful learning was complex and dependent on several factors, including the duration of the trace interval and the specific modification rules used in the simulation. Specifically, the study found that excessive inhibition could impair learning, and that the optimal level of inhibition varied depending on the duration of the trace interval and the type of modification rules used in the simulation. This study also identified the importance of preliminary modification and the interaction between different modification rules in shaping the relationship between inhibition and successful learning.

These findings have significant implications for our understanding of the role of inhibition in shaping neural activity and plasticity during associative learning. They highlight the importance of considering multiple factors, including the duration of the trace interval and the specific modification rules used, when investigating the relationship between inhibition and learning. Additionally, this study provides insights into the complex mechanisms underlying neural plasticity and the importance of carefully optimizing each parameter for the specific task at hand. Overall, these findings contribute to our understanding of the neural mechanisms underlying learning and memory in the brain and have the potential to inform future research in this field.

Introduction

Trace conditioning is a well-established phenomenon in the field of learning and memory research, which involves the pairing of a conditioned stimulus (CS) with an unconditioned stimulus (US) separated by a silent period known as the trace interval. The trace interval is a crucial component of the conditioning process as it requires the brain to maintain and update representations of the CS during the delay period. Therefore, the ability to accurately predict the onset of the US during the trace interval is essential for successful trace conditioning.

Computational models have been used extensively in the study of trace conditioning to provide insights into the underlying neural mechanisms of the phenomenon. Specifically, these models aim to explain how neural circuits encode and retrieve information about the CS during the trace interval and how this information is used to predict the timing of the US.

Recent research in the field has highlighted the role of inhibitory mechanisms in shaping the neural activity underlying trace conditioning. Inhibitory neurons are known to play a crucial role in regulating the activity of excitatory neurons and shaping the dynamics of neural circuits. In the context of trace conditioning, inhibitory neurons are thought to be involved in suppressing irrelevant neural activity during the trace interval, enhancing the representation of the CS in memory and improving the accuracy of predictions about the timing of the US.

One study that explored the role of inhibition in trace conditioning used a biologically realistic computational model to investigate the effects of inhibitory interneurons on the accuracy of the model's predictions about the onset of the US during the trace interval (1). The model was based on a neural network architecture and included both excitatory and inhibitory neurons, which interacted through synaptic connections with plasticity. The results of the study showed that increasing the strength of inhibition in the model enhanced the accuracy of the model's predictions about the timing of the US, thereby suggesting that inhibitory interneurons play a critical role in shaping the neural activity underlying trace conditioning.

Another study used a similar approach to investigate the role of a specific type of inhibitory neuron, known as the parvalbumin-positive interneuron, in trace conditioning (2). The study found that the loss of these neurons impaired the ability of mice to perform trace conditioning tasks, highlighting the crucial role of inhibitory mechanisms in learning and memory processes.

In addition to the role of inhibitory mechanisms in trace conditioning, recent research has also focused on the contribution of other neural mechanisms, including neuromodulators such as dopamine and acetylcholine. For example, one study found that dopamine signaling is necessary for the formation of trace memories and that the disruption of dopamine signaling impairs the accuracy of predictions about the onset of the US during the trace interval (3).

The goal of this study is to expand upon the research performed by other academics and provide valuable insights into the nature of neural inhibition and the role that it plays within classical learning. In these experiments, the level of inhibition was varied across experimental groups, rules for training and preliminary modification rates for inhibition were changed, and the effect of these parameters on the ability of the neural network to anticipate the trace interval were recorded.

Methods

Dave's Rule

Within this simulation, the manner through which synaptic modification occurs, and inhibitory factors by extension, is determined through the use of Dave's rule. Dave's rule is a powerful tool in computational neuroscience and is widely used to model the learning and plasticity of neural circuits. The rule is based on the idea that changes in synaptic strength can be induced by the precise timing of pre- and post-synaptic action potentials. The rule is simple to implement, making it a popular choice for modeling synaptic plasticity.

In a typical implementation of Dave's rule, the synaptic strength between two neurons is increased if the pre-synaptic neuron fires just before the post-synaptic neuron, and it is decreased if the pre-synaptic neuron fires just after the post-synaptic neuron. The magnitude of the change in synaptic strength is proportional to the product of the pre-synaptic firing rate and the time difference between pre- and post-synaptic spikes.

One of the advantages of Dave's rule is that it can account for a wide range of experimental data, including spike-timing-dependent plasticity (STDP) experiments. It is also computationally efficient and can be implemented in a variety of neural network models. Dave's rule has been used to model the plasticity of neural circuits in a variety of contexts, including visual processing, motor learning, and memory formation.

Dave's Rule Equation:

$$\Delta w = \eta (A_{pre} - A_{post} w) + \beta w$$

Representation of Dave's rule within the CA3 boxcar:

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dw_fbinhib = e_fb* z_prev .* (mean_z - desired_mean_z);
weights_feedback_inhib = w_fbinhib + dw_fbinhib;
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The Model

In the CA3 Boxcar 1 model, the neurons are divided into two groups, with neurons 1-40 representing the conditioned stimulus (CS) and neurons 41-80 representing the unconditioned stimulus (US). These neurons are modeled as simple McCulloch-Pitts devices with binary outputs, meaning that their output on each computational cycle is either one or zero.

The CA3 Boxcar 1 model is designed to simulate both training and testing trials. During training, the model is presented with a sequence of inputs that includes both CS and US neurons. In contrast, during testing, only the CS neurons are presented, and there is no external activation of US neurons. Moreover, during testing, the synaptic

modification rules are deactivated. The model output is detailed in the Results sections of research papers that use the CA3 Boxcar 1 model.

The goal of the CA3 Boxcar 1 model is to closely mimic the mechanisms of neuron activation and excitation in order to accurately recreate the process of learning in the hippocampus. The model has been used in numerous studies to investigate the neural mechanisms underlying trace conditioning and other hippocampal-dependent tasks. The CA3 Boxcar 1 model has also been influential in shaping our understanding of how the hippocampus processes information and learns to associate stimuli.

Within this CA3 model, the ability of the network to successfully predict the trace interval is given by five consecutive predictions with at least 12 neurons firing within the correct time interval (3-10 timesteps before the US onset). Additionally, each combination of parameters was tested through 10 trials to evaluate the degree of successful learning.

Inhibition

In these simulations, numerous parameters representing various types of inhibition were changed. Notably, the measure of overall inhibition at a given trial is determined through the following equation based on Dave's rule:

inhib = k_0 + k_fb*(inhib_fb) + k_ff*sum(x)

1. 'K_0_start' is an important parameter that determines the initial value of k_0, which is the primary measure of inhibition. The value of k_0 ensures that the mean_z activity of the network equals the desired mean_z specified. This parameter is modified at the end of each trial by increasing k_0, and therefore inhibition, whenever there is too much activity, and decreasing it when there is too little. By adjusting k_0, we can regulate the overall level of inhibition in the network and make it more or less sensitive to input stimuli.
2. 'K_fb_start' represents the measure of feedback inhibition at the start of the simulation, which corresponds to the inhibition based on the activity of individual neurons. This parameter plays a crucial role in regulating the overall activity of the network.

3. 'K_ff_start' controls the initial value of k_ff, which is similar to k_0 in that it controls overall activity, but is multiplied by a factor of external inputs. By modifying the value of k_ff, we can adjust the network's sensitivity to external stimuli.

Modification Rules

To research how changes in the modification rate of inhibition could impact the performance of our model, the following parameters were changed through a range of combinations and values.

1. 'Epsilon_k_0' is the modification rate used to adapt k_0 before and/or during training. This parameter determines the rate at which k_0 is adjusted to ensure that the network's activity remains within a desired range.
2. 'Toggle_k0_preliminary_mod' adjusts the value of k_0 before training begins to attune to desired activity.
3. 'Toggle_k0_training_mod' modifies the value of k_0 at the end of each trial to maintain desired activity.

Recorded Variables

To best determine the relationship between inhibition, trace duration, k0 epsilon modification rules, and their effect on the ability of our model to successfully predict the trace interval, numerous parameters were recorded.

1. Elapsed time: The amount of time taken to run the simulation.
2. Whether the network learned successfully the stimulus: This is a binary variable where 0 represents the network's inability to learn successfully while 1 demonstrates successful learning, representing the dependent variable.
3. The last trial number of the simulation run: This represents the number of trials that were completed before the simulation halted. The algorithm was set to end the simulation after 140 trails or after five consecutive successful trials, whichever comes first.
4. The total number of successful trials where the network fired at least 12 neurons 3-10 timesteps before the unconditioned stimulus onset.
5. The number of trials where the network fired prematurely in relation to the US.

6. The number of trials where the network fired too late in relation to the US.

Parameters and values used are recorded in the tables below:

Table 1 Constant Parameter Values

Name	Value
Number of Neurons	1000
Conditioned Stimulus Neurons	40
Unconditioned Stimulus Neurons	40
Connectivity	0.1
On Noise	0.005
Off Noise	0.5
External Activation	30
Stutter	5
Shift	15

Table 2 Experimental Group 1

Parameter	Low Inhibition	Low-Moderate Inhibition	High-Moderate Inhibition	High Inhibition
k_0_start	0.2	0.4	0.6	0.8
k_fb_start	0.01	0.03	0.05	0.065
k_ff_start	0.005	0.01	0.015	0.02

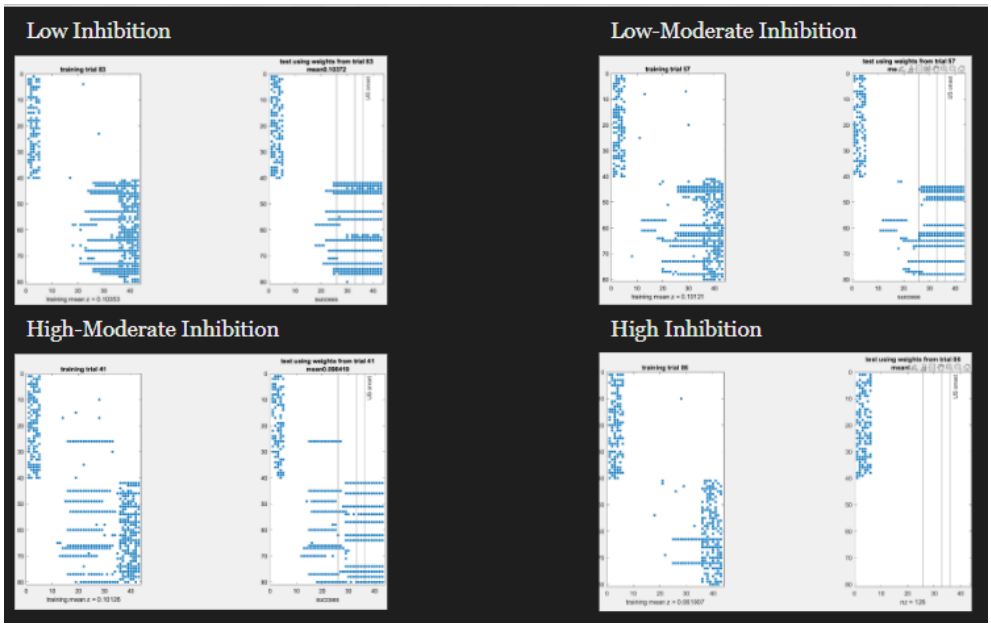
Trace Duration* - Set to 20 & 30 timesteps for each category of experimental group 1, where each timestep represents 20ms of time.

Table 3 Experimental Group 2

	Modification Rule	Values	Ko Epsilon Values
Control	Preliminary Modification	False	N/A
	Training Modification	False	
Category 1	Preliminary Modification	True	{0.1, 0.2, 0.3}
	Training Modification	False	
Category 2	Preliminary Modification	False	{0.1, 0.2, 0.3}
	Training Modification	True	
Category 3	Preliminary Modification	True	{0.1, 0.2, 0.3}
	Training Modification	True	

Results

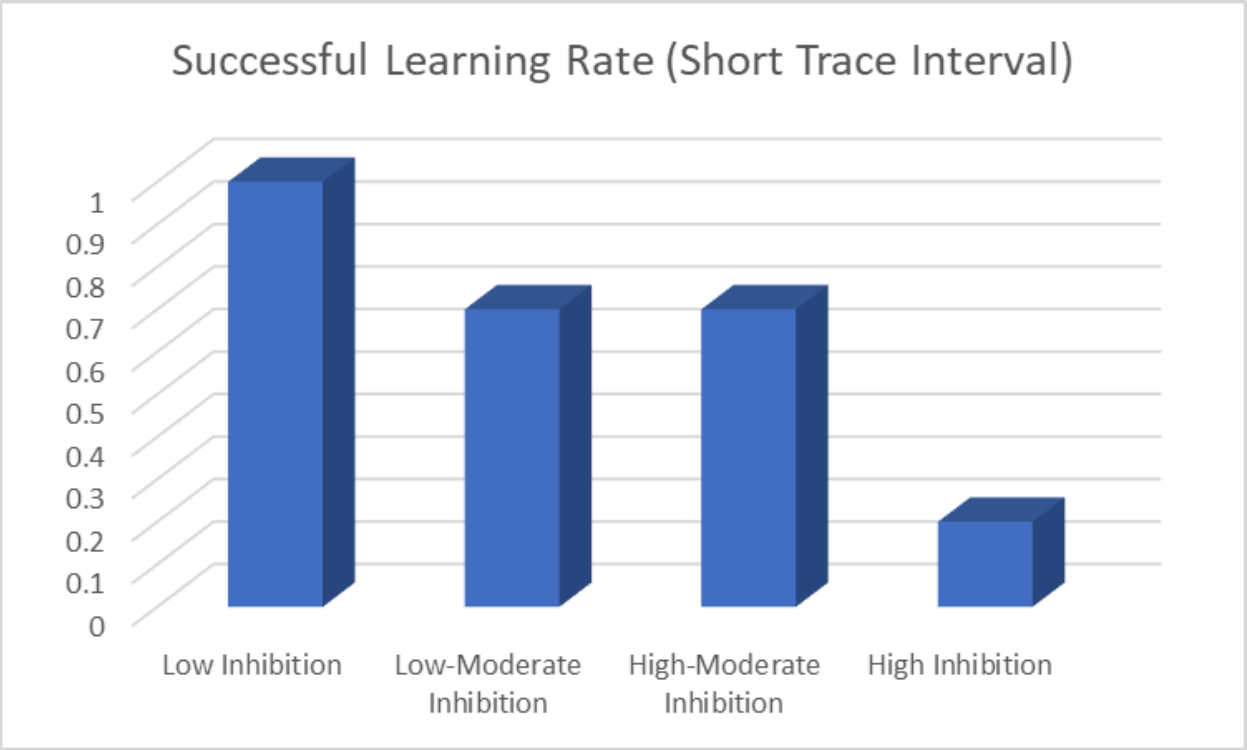
Figure 1. Neural Activity Across Level of Inhibition



The figure above outlines the results of the set of simulations using a longer trace interval across varying levels of inhibition. The purpose of this graphic is to provide a general overview of the trends in how inhibition impacts neural activity across US and CS neurons over the course of the simulation's time. This graphic makes it clear that there is a strong negative correlation between the strength of the inhibition and overall neural activation.

In relation to real world biological mechanisms, one possible explanation for this negative correlation is that inhibitory neurons suppress irrelevant neural activity during the trace interval, allowing for the enhanced representation of the CS in memory and improving the accuracy of predictions about the timing of the US. In this way, inhibition may function as a gating mechanism, filtering out unwanted noise and enhancing the signal-to-noise ratio of the neural activity underlying trace conditioning.

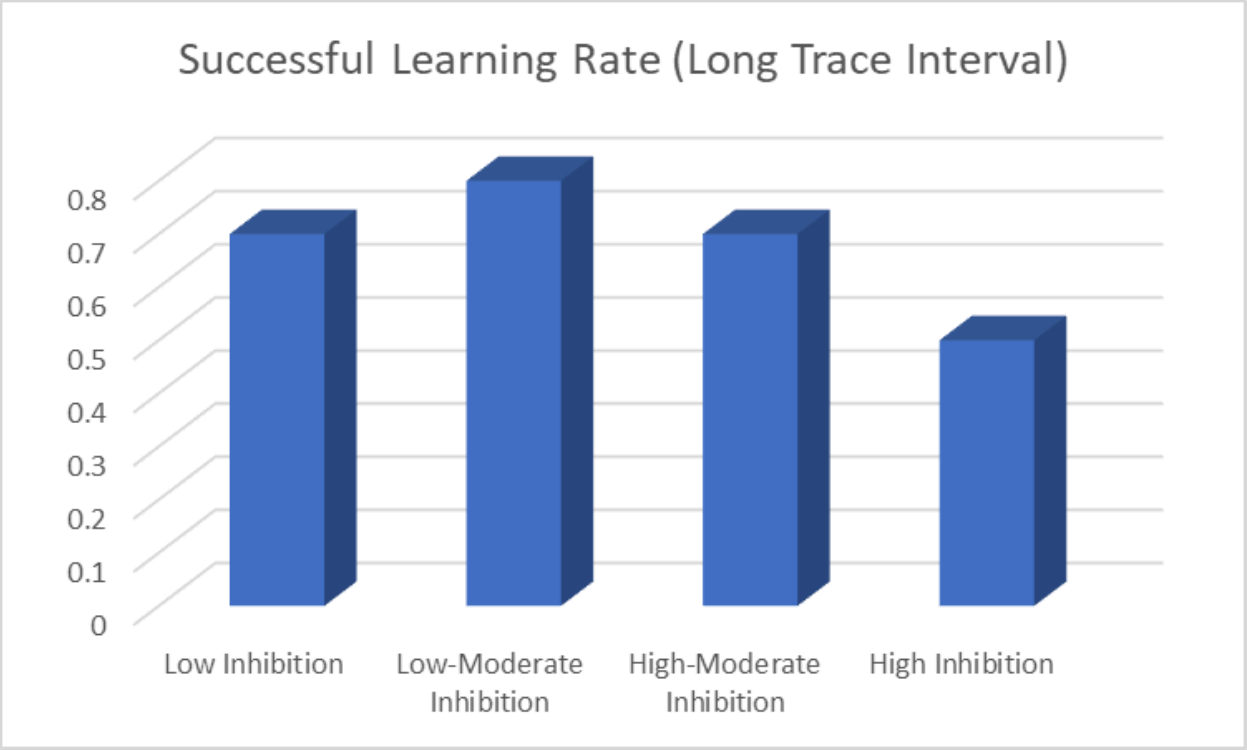
Figure 2: The impact of varying levels of inhibition on learning with a short trace interval



Based on the simulations with parameters representing Experimental Group 1, when the trace duration was set to 20 timesteps (400 ms), the simulations showed a negative correlation between the degree of inhibitory scale factor and the rate of successful learning. This means that higher levels of inhibition resulted in a slower rate of successful learning, while lower levels of inhibition led to a faster rate of successful learning.

The negative correlation between the degree of inhibition and the rate of successful learning suggests that too much inhibition can be detrimental to learning in this trace conditioning simulation. This finding aligns with previous studies that have shown that inhibition plays a crucial role in shaping neuronal activity and plasticity in the brain. However, an excessive amount of inhibition may hinder the ability of neurons to fire in response to specific stimuli, thus impairing learning.

Figure 3: The impact of varying levels of inhibition on learning with a long trace interval



The discovery that a longer trace duration resulted in a higher rate of successful learning with low-moderate levels of inhibition carries significant implications. It implies that the ideal level of inhibition for a particular task may depend on its specific requirements. Moreover, it underscores the significance of assessing the interplay between various parameters while creating models for neural systems. The fact that the optimal level of inhibition differed between the two trace durations highlights the importance of optimizing each parameter for the task at hand. Furthermore, this finding suggests that the relationship between inhibition and learning may be more intricate and non-linear than previously thought, and additional research may be necessary to fully comprehend the underlying mechanisms.

Figure 3: Relationship between the number of premature predictions and the degree of inhibition with a shorter trace interval.

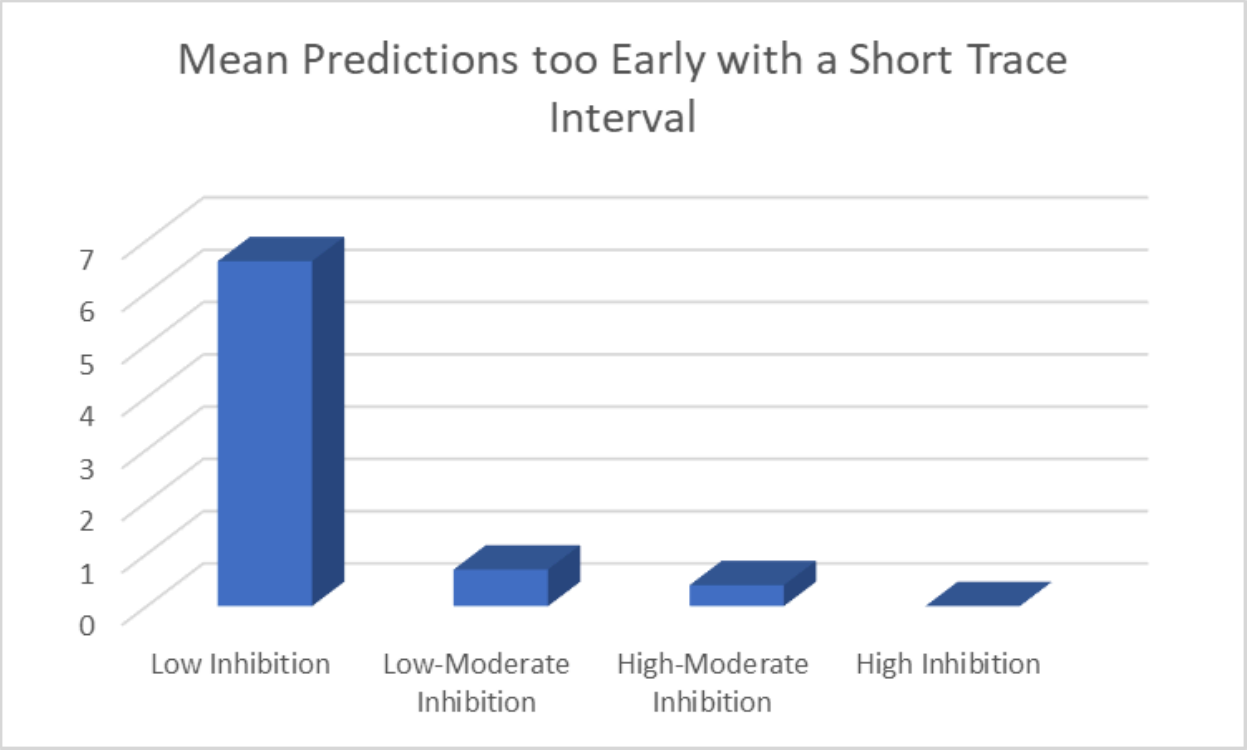
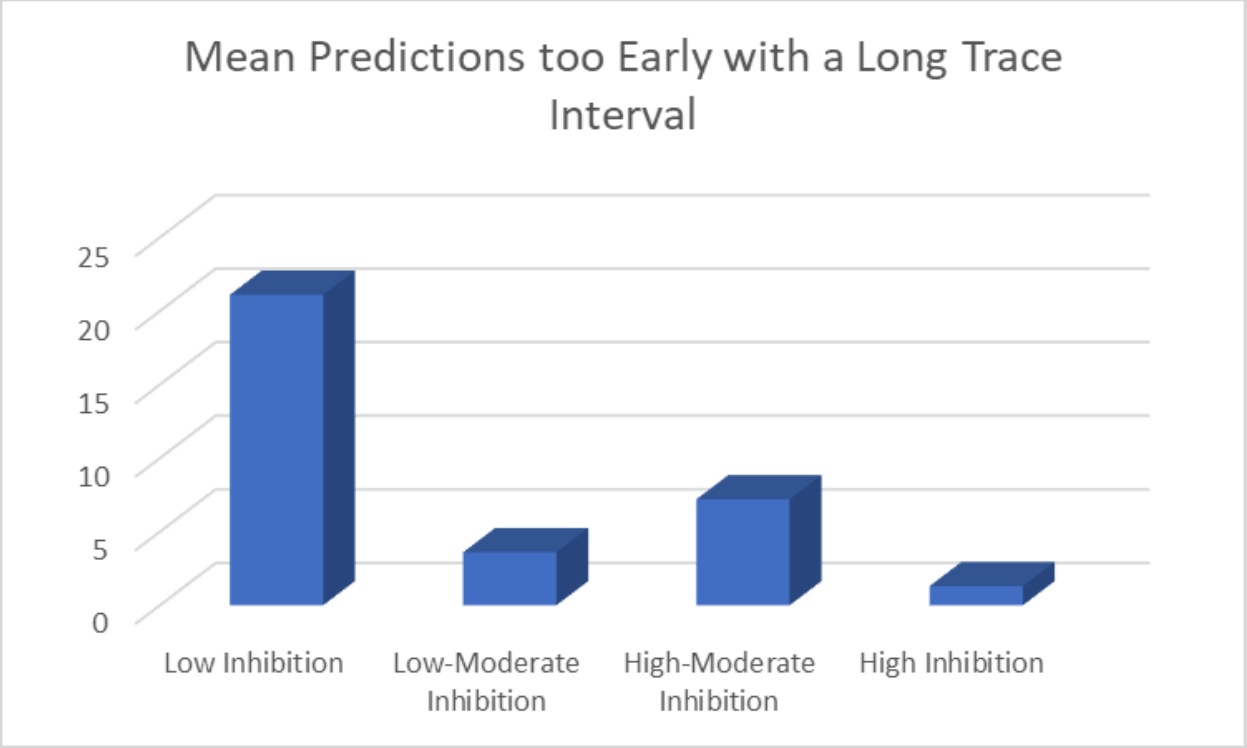


Figure 4: Relationship between the number of predictions too early and the degree of inhibition with a longer trace interval.



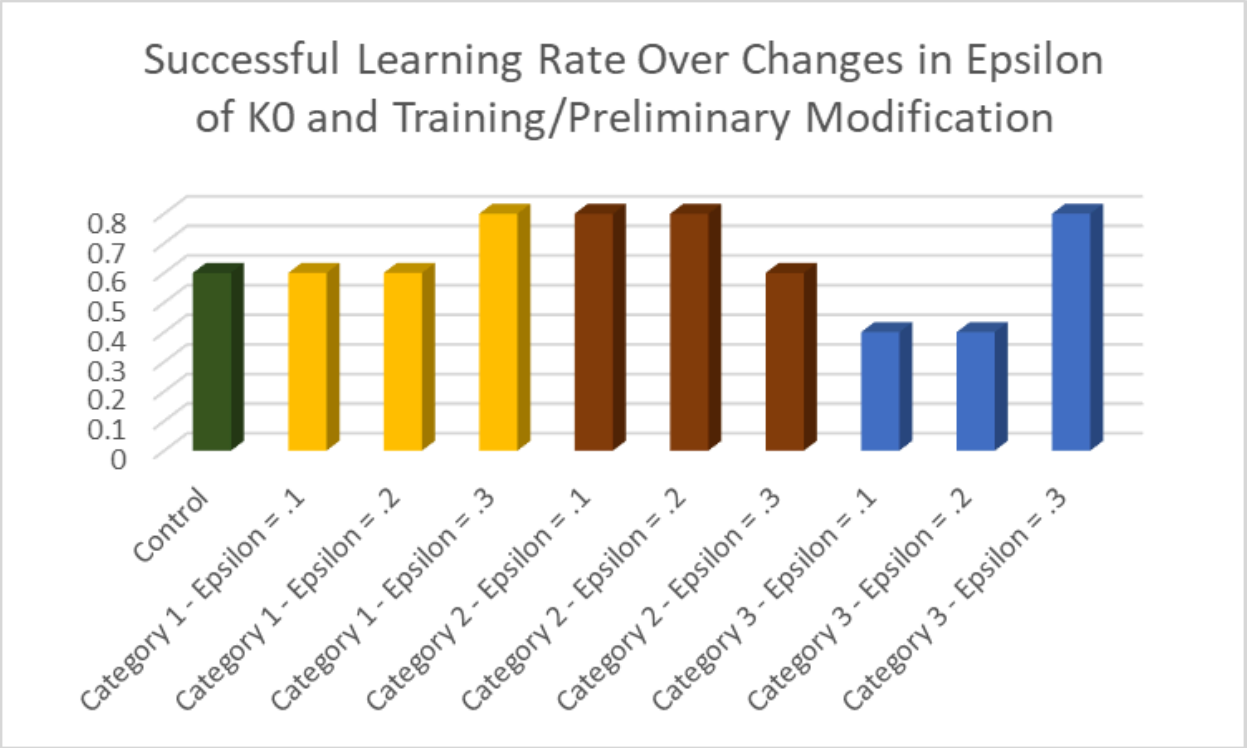
The results of this experiment suggest that inhibition plays a vital role in regulating and optimizing neural activity. Specifically, insufficient inhibition can lead to excessive neural activity, which can contribute to premature firing in the context of trace conditioning. This finding highlights the importance of maintaining an appropriate balance of inhibition in neural circuitry to prevent maladaptive firing patterns.

Interestingly, I also found that the rate of premature firing was higher with a longer trace interval. This observation suggests that the demands placed on the neural circuitry are greater under such conditions. This finding is particularly relevant, as it underscores the importance of considering the specific task demands when modeling and studying neural systems. The results suggest that the length of the trace interval is a critical parameter to consider when designing and interpreting experiments, as it can

significantly impact the rate of premature firing and, ultimately, the effectiveness of learning.

Furthermore, the finding that insufficient inhibition can lead to excessive neural activity has implications beyond the specific context of trace conditioning. It suggests that the relationship between inhibition and neural activity may be more complex than previously thought, with insufficient inhibition potentially leading to maladaptive firing patterns in a range of neural systems. For this reason, I'd like to highlight the need for further research to better understand the precise mechanisms underlying this relationship and to develop strategies to optimize inhibition in neural circuitry.

Figure 5: Impact of Modification Rules on Learning Rate



The effect of modifying k0 on the performance of a trace conditioning simulation was studied, and it was found that the type of modification rule used had a significant impact on the ability of the model to learn. Specifically, the results showed that changes in preliminary modification had a greater impact on the model's performance compared to changes in training modification. When preliminary modification was the only

modification rule in effect, there was a positive correlation between inhibition and learning. On the other hand, when only training modification was applied, there was a negative correlation between inhibition and learning. Interestingly, when both preliminary and training modification were turned on, there was a stronger positive correlation between inhibition and learning compared to the other groups.

These findings suggest that the rules governing how k0 is modified play a crucial role in learning in the trace conditioning simulation. Preliminary modification, in particular, appears to be more important than training modification, and the interaction between the two may be crucial for optimal learning. Furthermore, the different correlations observed between inhibition and learning depending on the modification rules indicate that the mechanisms underlying learning in the simulation are complex and multifaceted.

This provides valuable insights into the underlying mechanisms of neural plasticity and highlights the importance of considering multiple time factors when investigating learning and memory in the brain. Overall, these findings contribute to our understanding of the complex interplay between different factors in neural systems, and may have important implications for the development of more effective therapies for learning and memory disorders.

Discussion

Overall, the results of our study highlight the importance of inhibition in the process of neural learning and the detrimental effects of excessive neural activity. Our experiments demonstrate that the degree of inhibition has a significant impact on the rate at which successful learning occurs, with a "sweet spot" of low-moderate inhibition providing the optimal conditions for learning. The results also suggest that the duration of the trace interval plays a critical role in learning and that a longer trace interval leads to a higher rate of premature firing.

Furthermore, the experiments show that changes in the epsilon of k0 have different effects on learning depending on the modification rules that are applied.

Preliminary modification has a greater impact on learning than training modification, and there is a stronger positive correlation between inhibition and learning when both preliminary and training modification are turned on. These findings provide insights into the mechanisms of neural learning and may inform the development of more effective neural network models.

The inefficiency caused by insufficient inhibition observed in our experiments sheds light on the purpose of inhibition in nature. It appears that inhibition serves to prevent excessive neural activity that can interfere with learning and memory processes. Our study suggests that a fine balance between excitation and inhibition is necessary for optimal learning and that excessive neural activity can actually hinder the learning process. These findings have implications for understanding the role of inhibition in other neural processes and may ultimately inform the development of new therapies for neurological disorders.

While these experiments may have provided some valuable insights into the relationship between modification rates, rules, and the nature of inhibition in relation to learning across various trace durations, there are several areas that could be improved. One limitation of the study is that it focuses solely on simulations and does not involve real-world experimental data that use living animals or record results based on neural scanning. For instance, if we were to record the levels of extracellular sodium, GABA, or glycine concentration in the CA3 region of a primate, and expose them to more traditional Pavlovian conditioning techniques, we would have a more realistic and biologically rooted model that proves or disproves that legitimacy of these simulations in actual brains.

Finally, this study did not explore the potential neural mechanisms underlying the observed correlations between inhibition, modification rules, and learning. Future research could use electrophysiological or imaging techniques to investigate the neural activity and plasticity underlying these findings within biologically based models. In particular, there is no precise biologically based equivalent of our preliminary

modification rule outside of computation. For these reasons, experimental data should be collected on animals to determine the legitimacy of this trace conditioning model.

Acknowledgements

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References

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Appendix A

Lowest K Vals		Based on 400ms Trace interval							
k_0_start'	0.2		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
k_fb_start'	0.01	Trial 1	1	11.890254	59	5	8.474576271	8	1
k_ff_start'	0.005	Trial 2	1	8.829441	65	6	9.230769231	9	1
		Trial 3	1	12.20385	61	7	11.47540984	6	0
		Trial 4	1	11.581343	59	5	8.474576271	7	0
		Trial 5	1	11.581343	61	7	11.47540984	5	0
		Trial 6	1	12.712187	75	7	9.333333333	5	0
		Trial 7	1	11.881695	68	6	8.823529412	4	0
		Trial 8	1	10.46954	58	10	17.24137931	6	4
		Trial 9	1	11.247976	64	8	12.5	9	0
		Trial 10	1	15.725311	93	12	12.90322581	7	0

Low-Mid K Vals			Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
k_0_start'	0.4	Trial 1	1	15.725311	93	12	12.90322581	7	0
k_fb_start'	0.03	Trial 2	1	9.911782	56	8	14.28571429	0	2
k_ff_start'	0.01	Trial 3	1	8.825852	49	6	12.24489796	0	2
		Trial 4	0	23.283655	140	2	1.428571429	0	0
		Trial 5	0	37.716096	140	8	5.714285714	0	1
		Trial 6	1	7.531138	39	7	17.94871795	0	0
		Trial 7	0	24.628063	140	9	6.428571429	0	0
		Trial 8	1	8.63496	50	6	12	0	0
		Trial 9	1	7.877367	42	6	14.28571429	0	0
		Trial 10	1	7.612898	42	7	16.66666667	0	1
Mid-High K Vals		Based on 400ms Trace interval							
k_0_start'	0.6		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late

k_fb_start'	0.05	Trial 1	1	9.424908	47	11	23.40425532	0	1
k_ff_start'	0.015	Trial 2	0	28.030864	140	22	15.71428571	0	3
		Trial 3	0	29.428279	140	2	1.428571429	0	0
		Trial 4	1	9.132654	44	13	29.54545455	0	0
		Trial 5	1	6.530395	36	15	41.66666667	1	0
		Trial 6	1	15.214195	42	13	30.95238095	1	0
		Trial 7	1	6.921983	39	9	23.07692308	0	1
		Trial 8	1	10.032597	46	9	19.56521739	0	3
		Trial 9	0	24.015577	140	9	6.428571429	1	0
		Trial 10	1	14.447281	44	13	29.54545455	1	3
Highest K Vals		Based on 400ms Trace interval							
k_0_start'	0.8		Success/Fail (1,0)	Elapsed Time	Total Trials / Trials before successful	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late

					prediction				
k_fb_start'	0.065	Trial 1	0	28.321639	140	1	0.7142857143	0	0
k_ff_start'	0.02	Trial 2	1	7.83678	39	5	12.82051282	0	1
		Trial 3	0	23.856052	140	7	5	0	1
		Trial 4	0	36.737123	140	3	2.142857143	0	0
		Trial 5	1	7.508293	40	7	17.5	0	1
		Trial 6	0	32.415053	140	3	2.142857143	0	0
		Trial 7	0	24.425681	140	3	2.142857143	0	1
		Trial 8	0	35.636929	140	3	2.142857143	0	1
		Trial 9	0	54.656151	140	3	2.142857143	0	1
		Trial 10	0	24.235202	140	3	2.142857143	0	1
Lowest K Vals		600ms Trace Interval	Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late

k_0_start'	0.2	Trial 1	0	27.435834	140	1	0.7142857143	31	0
k_fb_start'	0.01	Trial 2	1	17.945881	91	7	7.692307692	8	0
k_ff_start'	0.005	Trial 3	0	27.846722	140	5	3.571428571	52	0
		Trial 4	1	12.401541	54	9	16.66666667	9	0
		Trial 5	1	9.63249	58	5	8.620689655	6	0
		Trial 6	1	16.391307	91	5	5.494505495	7	0
		Trial 7	0	24.989444	140	2	1.428571429	77	0
		Trial 8	1	8.121045	49	7	14.28571429	7	0
		Trial 9	1	16.597752	90	6	6.666666667	6	0
		Trial 10	1	9.739532	54	9	16.66666667	8	0
Low-Mid K			Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
k_0_start'	0.4	Trial 1	1	9.978504	55	15	27.27272727	1	1
k_fb_start'	0.03	Trial 2	0	28.901315	140	3	2.142857143	0	0
k_ff_start'	0.01	Trial 3	1	13.053036	65	7	10.76923077	1	0

		Trial 4	0	27.001282	140	2	1.428571429	9	0
		Trial 5	1	6.271921	38	10	26.31578947	9	0
		Trial 6	1	6.068993	36	9	25	0	0
		Trial 7	1	12.868692	83	12	14.45783133	3	0
		Trial 8	1	6.690955	43	10	23.25581395	5	0
		Trial 9	1	13.723739	77	11	14.28571429	5	0
		Trial 10	1	13.424677	75	6	8	3	0
Mid-High K Vals			Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
k_0_start'	0.6	Trial 1	1	12.217128	77	8	10.38961039	6	0
k_fb_start'	0.05	Trial 2	1	7.508382	50	13	26	9	0
k_ff_start'	0.015	Trial 3	1	6.626352	46	10	21.73913043	7	0
		Trial 4	0	19.486683	140	3	2.142857143	5	0
		Trial 5	1	11.283226	82	7	8.536585366	9	0
		Trial 6	1	5.614184	37	10	27.02702703	9	0
		Trial 7	1	5.578335	38	11	28.94736842	6	0
		Trial 8	1	8.868698	66	5	7.575757576	10	0

		Trial 9	0	20.320702	140	6	4.285714286	9	0
		Trial 10	0	20.675993	140	1	0.7142857143	2	0
Highest K Vals			Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
k_0_start'	0.8	Trial 1	0	31.15334	140	10	7.142857143	0	4
k_fb_start'	0.065	Trial 2	1	5.100563	29	6	20.68965517	3	0
k_ff_start'	0.02	Trial 3	1	10.049733	62	6	9.677419355	0	0
		Trial 4	0	29.809246	140	1	0.7142857143	0	0
		Trial 5	0	18.426227	140	1	0.7142857143	3	0
		Trial 6	0	20.596367	140	3	2.142857143	1	1
		Trial 7	1	4.460407	29	6	20.68965517	2	0
		Trial 8	1	7.912569	48	8	16.66666667	1	0
		Trial 9	0	23.555679	140	1	0.7142857143	1	0
		Trial 10	1	5.16429	32	9	28.125	2	0

Appendix B

Control			Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	FALSE	Trial 1	1	16.639591	76	8	10.52631579	1	0
toggle_k0_training_mod'	FALSE	Trial 2	1	15.671474	71	6	8.450704225	1	0
		Trial 3	0	29.504798	140	1	0.7142857143	27	0
		Trial 4	1	9.049084	40	6	15	3	0
		Trial 5	0	39.60606	140	1	0.7142857143	3	0
Category 1	epsilon_k_0 = .1		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	TRUE	Trial 1	1	18.25004	52	6	11.53846154	1	0
toggle_k0_training_mod'	FALSE	Trial 2	0	91.715732	140	1	0.7142857143	5	0
		Trial 3	1	15.818656	40	9	22.5	1	1
		Trial 4	0	81.252997	140	2	1.428571429	3	0

		Trial 5	1	26.335197	53	5	9.433962264	1	0
Category 1	epsilon_k_0 = .2		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	TRUE	Trial 1	0	35.636571	140	5	3.571428571	15	0
toggle_k0_training_mod'	FALSE	Trial 2	1	13.89467	35	6	17.14285714	6	0
		Trial 3	0	58.262189	140	0	0	2	0
		Trial 4	1	18.861155	67	8	11.94029851	2	0
		Trial 5	1	14.077789	40	5	12.5	5	0
Category 1	epsilon_k_0 = .3		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	TRUE	Trial 1	1	23.795078	50	10	20	2	0
toggle_k0_training_mod'	FALSE	Trial 2	0	64.79192	140	1	0.7142857143	15	0
		Trial 3	1	64.79192	78	9	11.53846154	1	0
		Trial 4	1	50.980129	120	6	5	10	0
		Trial 5	1	29.251564	57	7	12.28070175	2	0

Category 2	epsilon_k_0 = .1		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	FALSE	Trial 1	1	12.299813	62	9	14.51612903	1	2
toggle_k0_training_mod'	TRUE	Trial 2	1	7.049105	35	7	20	2	0
		Trial 3	1	7.472232	36	8	22.22222222	5	0
		Trial 4	1	25.473245	78	7	8.974358974	2	0
		Trial 5	0	61.624418	140	1	0.7142857143	1	0
Category 2	epsilon_k_0 = .2		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	FALSE	Trial 1	1	8.394592	43	11	25.58139535	3	0
toggle_k0_training_mod'	TRUE	Trial 2	1	10.63392	54	9	16.66666667	3	0
		Trial 3	0	47.146474	140	1	0.7142857143	5	0
		Trial 4	1	16.045761	76	6	7.894736842	2	0

		Trial 5	1	10.09059	49	6	12.24489796	2	0
Category 2	epsilon_k_0 = .3		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	FALSE	Trial 1	0	49.709173	140	1	0.7142857143	3	0
toggle_k0_training_mod'	TRUE	Trial 2	1	6.68235	32	5	15.625	3	0
		Trial 3	1	21.827502	55	12	21.81818182	4	0
		Trial 4	0	61.453357	140	4	2.857142857	3	0
		Trial 5	1	12.528684	54	7	12.96296296	3	0
Category 3	epsilon_k_0 = .1		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	TRUE	Trial 1	1	12.528684	55	5	9.090909091	3	0
toggle_k0_training_mod'	TRUE	Trial 2	1	12.232285	33	6	18.18181818	1	0
		Trial 3	0	77.58125	140	0	0	29	0
		Trial 4	0	66.911301	140	2	1.428571429	3	0

		Trial 5	0	79.373756	140	4	2.857142857	2	0
Category 3	epsilon_k_0 = .2		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	TRUE	Trial 1	0	63.907378	140	4	2.857142857	33	0
toggle_k0_training_mod'	TRUE	Trial 2	0	81.792625	140	1	0.7142857143	2	0
		Trial 3	1	49.559822	77	12	15.58441558	0	2
		Trial 4	0	56.768521	140	5	3.571428571	4	0
		Trial 5	1	50.012085	88	11	12.5	3	0
Category 3	epsilon_k_0 = .3		Success/Fail (1,0)	Elapsed Time	Total Trials	# Successful Prediction	% Successful Prediction	# Pred Too Soon	# Pred Too Late
toggle_k0_preliminary_mod'	TRUE	Trial 1	1	35.454055	63	7	11.11111111	5	0
toggle_k0_training_mod'	TRUE	Trial 2	0	34.459567	140	4	2.857142857	6	0
		Trial 3	1	14.110039	35	7	20	2	0
		Trial 4	1	20.83082	68	7	10.29411765	4	0
		Trial 5	1	16.904968	56	8	14.28571429	2	0