The Role of Temporal Parameters in a Thalamocortical Model of Analogy

Yoonsuck Choe
Department of Computer Science, Texas A&M University
3112 TAMU, College Station, TX 77843-3112
Email: choe@tamu.edu

Abstract

How do the multiple specialized cortical areas in the brain interact with each other to give rise to an integrated behavior is a largely unanswered question. In this paper, I propose that such an integration can be understood under the framework of *analogy*, and that the thalamus and the thalamic reticular nucleus (TRN) may be playing a key role in this respect. The proposed thalamocortical model of analogy heavily depends on a diverse set of temporal parameters including axonal delay and membrane time constant, each of which is critical for the proper functioning of the model. The model requires a specific set of conditions derived from the need of the model to process analogies. Computational results with a network of integrate-and-fire (IF) neurons suggest that these conditions are indeed necessary, and furthermore, data found in the experimental literature also support these conditions. The model suggests that there is a very good reason for each temporal parameter in the thalamocortical network having a particular value, and that to understand the integrated behavior of the brain, we need to study these parameters simultaneously, not separately.

Keywords: Thalamus, Thalamic Reticular Nucleus, Cortiothalamic Feedback, Analogy, Axonal Delay, Membrane Time Constant.

I. Introduction

Understanding how cortical maps in the brain interact with each other to generate an integrated behavior is an important unsolved problem. Although we now know a lot more about the anatomical connectivity and physiology of the cortical maps than decades ago, such as layout of visual areas [1], [2], long range horizontal connectivity [3], [4], we still lack the understanding of how these maps work as an integrated system to give rise to a coherent behavior.

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One key insight, from recent advances in neuroscience, can be gained from the thalamus. The thalamus is a centrally located nucleus in the brain with a high degree of feedforward and feedback connections to and from the cortex (see [5], [6], [7] for a review). The thalamus was previously thought of as a passive relay station for sensory-motor signals, however, this explanation was not satisfactory once the existence of massive feedback from the cortex and a thin inhibitory network covering the thalamus called the thalamic reticular nucleus (TRN; see [8] for a review) became to be known.

Several different functions have been attributed to the thalamus and its associated structures: (1) attentional modulation [9], [10], [11]; (2) predictive preactivation [12]; (3) temporal binding [13]; (4) cross-modal modulation [14]; (5) active blackboard (or global workspace) [15], [16], [17]; (6) modulation of sleep and wake states [18], [19]; (7) action selection [20]; (8) mediation of cortico-cortical communication [21]; and finally, (9) consciousness and awareness [22], [23], [24], [25]. Although these explanations sound very different from each other, at the core they share a common theme that the thalamus integrates and/or selectively directs information received from the sensory periphery and also from the cortex. This insight is due to the massive convergence of both afferent and cortical feedback on to the thalamus (which is supposed to integrate diverse information), and also due to the inhibitory network surrounding the thalamus (i.e., the TRN, which performs selection, filtering, or gating). However, *what* gets integrated or selectively directed, or *what* is the property or nature of that which gets integrated or selectively directed remains a question.

Taking a slightly different perspective from the above, I recently proposed that the interplay between the thalamus, TRN, and the cortex may be implementing the function of analogy (the *analogy hypothesis*; [26], [27]). Analogy, defined broadly, plays an important role in human perception, cognition, and language [28], [29], [30], [31], [32], [33], [34], and in a more general sense, in motor control as well [29], [35]. The ability of analogy to cross domain boundaries is critical in these functions, and this may be exactly what is needed for the integrative operation of cortical maps. Relating one sensory modality (say, sound) to another (e.g., vision), explaining a more abstract concept in terms of more concrete sensory-motor concepts, and imitation of visually perceived events to generate a similar motion are all good examples where a broader definition of analogy can be applied, and where cortical integration is needed.

There is experimental evidence suggesting that the thalamus and the TRN may in fact be involved in such an integrative activity. For example, Crabtree and Isaac [14] discovered through electrophysiological methods that there exists a rich set of functional pathways between different pairs of thalamic nuclei with different sensory/cognitive/motor modalities. More specifically, they showed that electrical stimulation of one nucleus in the dorsal thalamus can result in an inhibitory response in another nucleus of a different modality. These cross-modal influences are mostly inhibitory (GABAergic), and this is consistent with the view that the TRN may take part in such an interaction by conducting disynaptic inhibition [14]. These results can provide experimental grounds for the analogy hypothesis, which puts an emphasis on the cross-modal function of the thalamus and the TRN. Another important aspect of the thalamic function, besides the functional connectivity, is the diverse structural and physiological characteristics that can give rise to a complex temporal dimension to the functioning of this nucleus. For example, long membrane time constants in TRN neurons [36], [37], long axonal conduction delay in corticothalamic feedback due to the unmyelinated axons [38], and fast synaptic transmission between reticular neurons through gap junctions [39] are experimental results evidencing the rich temporal aspect of the thalamocortical network. In this paper, the role of this diversity in the temporal dimension will be investigated in relation to the function of analogy.

This paper presents a computational implementation of the hypothetical model described in [26], [27] and compare the results with the previous predictions. The predictions are related to the various temporal requirements of the model, and the goal is to show that the temporal dynamics we observe in experimental data have an important role to play under the framework of analogy.

The following sections will briefly summarize the analogy hypothesis, then provide details of the implemented model, together with the results. The paper will conclude with a discussion about issues to be resolved, relevant related work, and future directions.

II. Neurons as Active Elements

Neurons are commonly seen as processing information, i.e., producing output given a certain input. However, in this view, the output generated by neurons becomes passive, in the sense that they need further interpretation by something that actively computes from or processes that output. A scheme like this can potentially lead to an

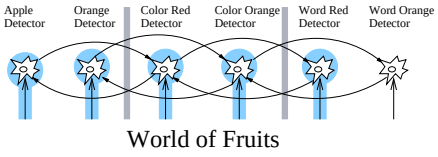
infinite regress where the final word on the semantics (or meaning) of the information is infinitely delayed, which can be a severe problem. Since passiveness has been identified as a problem, we can reverse our perspective a little bit and view neurons as *active elements*, in the sense that neurons actively *invoke* other neurons. In fact, this is one major function of the neurons, i.e., causing target neurons to activate.

A natural question that arises from this is, "what benefit can such a small change of perspective bring us?" From the simple observation that neurons are active, and focusing more on the active invoking nature of neurons, I showed that we can derive the function of *analogy* [26], [27]. The very act of invoking a pattern of activity in another neuron in a different modality can be seen as establishing semantics or a context through analogy, and this can potentially help mitigate the problems associated with the passive view. The papers above demonstrated that *completion* and *filtering* are necessary for such active elements to implement the function of analogy. In the following, an example will briefly illustrate how this works.

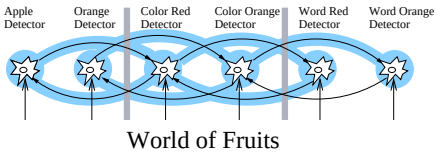
III. Analogy Through Active Completion and Filtering

Let us suppose we have a simple brain with active neurons (or population of neurons) responding to specific inputs in the environment (Fig. 1). The simple brain has detectors for different input features including fruit objects (apple and orange), colors (red and orange color), and spoken words (word-red and word-orange). The neurons are *active*, i.e., they *invoke* other neurons when they fire, and the invoking is done through relational (or associative) links which are learned through experience and embody frequently co-occurring events or stimulus pairs. For example, the apple-object detector may have a strong connection to the color-red detector because apples are mostly red.

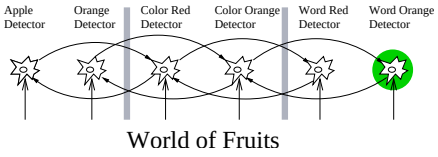
Given the above, consider the proportional analogy question `apple:orange::word-red:?`. Fig. 1 shows an activation sequence of the simple brain to this input question. Initially, the first 5 neurons in the left are activated in response to this input (Fig. 1a). Note that when an apple (object) is presented, its red color also activates the color-red detector, and the same goes for the orange (object). Next, through the relational links, other cortical neurons are invoked (Fig. 1b). This is where the *completion* occurs based on previously learned associations. It is kind of a replaying of the previously observed sequence of events, thus it can be seen as *predicting* what is to be expected next, given the current input. Last, after *filtering* for the purely cortex-driven activity in the cortex, only the `word-orange` detector remains active (Fig. 1c). In other words, the filtering process promotes newly formed



(a) Initial activation.



(b) Active completion.



(c) After filtering.

Fig. 1. **Analogy Through Active Completion and Filtering.** A simple brain with 6 detector neurons is shown. Environmental input comes in from the bottom (*World of Fruits*), and the neurons are linked with directed arcs that show the learned association between the detectors. The gray vertical bars partition the neurons in to separate maps: object, color, and word maps from the left to the right. From (a) to (c) shows the activation sequence of the brain in response to an analogy question "`apple:orange::word-red:?`." (a) Initial activation. (b) Active completion across relational links. (c) Remaining activity after filtering out input-driven cortical activity.

activity not directly caused by the input but somehow related to the current input. Interestingly enough, such a cortex-driven activity that survives the filtering (in this case `word-orange`) happens to be precisely the answer to the analogy question we posed above. Thus, in this simple case, associative completion and filtering can account for a primitive form of an analogical process.

However, things can get complicated when combinations of objects are used in the question. Let us extend the simple brain to include concepts of small and big (not shown in Fig. 1). For such a case, simple filtering for only purely cortex-driven activity is insufficient because the answer can contain items that are already present in the question. For example, consider the analogy question `big apple:small apple::big orange:?`. The answer is `small orange`, but both `small` and `orange` appeared in the question, thus the simple filtering as above will not work (i.e., everything will be filtered out). In this case, relaxing the filtering criterion allows us to get

to the answer: Find *relatively less input-driven* cortical activities [26], [27] within each map (e.g., object map, color map, and word map shown in Fig. 1 divided by the gray vertical bars). Following a similar activation sequence as in Fig. 1, we can see that `word-red` and `word-orange` are the purely cortex-driven activities. In addition, because `big` and `apple` appeared in the input twice but `small` and `orange` appeared only once, the latter two can survive the filtering under the new criterion. Thus, even for cortex-driven activities that are also input-driven, those that are less input-driven can survive and the correct analogical response can still be found among such activities that are more cortically-driven within each map (or area). Note that not only `small orange` survives such a filtering, and thus the answer is not exact. However, what is more important here is that such a simple filtering process can generate a small set of *potential answers*. Although simple analogical questions may have straightforward answers, in more complex analogical problems, there can be multiple answers depending on the context [40], [41], and the flexibility shown above can be a desirable thing to have.

In summary, the examples demonstrated that simple analogies can be processed by active completion and filtering, and this observation was made possible by the initial change of perspective to view neurons as active elements. In the next section, we will see how such completion and filtering can be neurally implemented.

IV. Active Completion and Filtering in the Thalamocortical Loop

How can active completion and filtering be implemented in the neural networks of the brain? In previous papers, I proposed that the thalamus, thalamic reticular nucleus (TRN), and the cortex may be involved in completion and filtering [26], [27]. Fig. 2 shows a schematic diagram of the thalamocortical circuit based on known anatomy and physiology [6], [14]. In the following, we shall review how this circuit can carry out completion and filtering.

Cortico-cortical connections linking different maps in the cortex are ideally suited for active completion (see, e.g., [42]). Specific patterns of connections observed in animals (e.g. visual cortex of monkeys; [43], [4]) show how such patterns can implement specific completion functions.

A more difficult issue is how can filtering be done, i.e., how can input-driven cortical activity be distinguished from cortex-driven cortical activity? As suggested in [26], [27], the TRN is a promising location where such a filtering can occur.

The basic idea is that the reticular neurons receive both ascending thalamic input and descending cortical feedback,

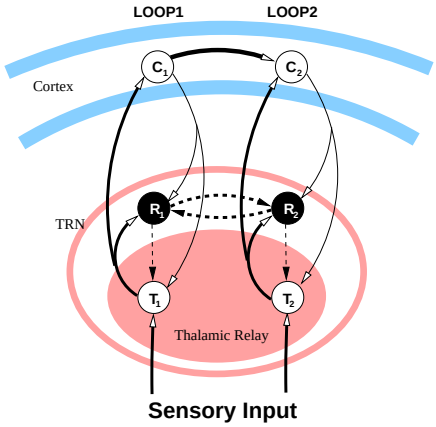


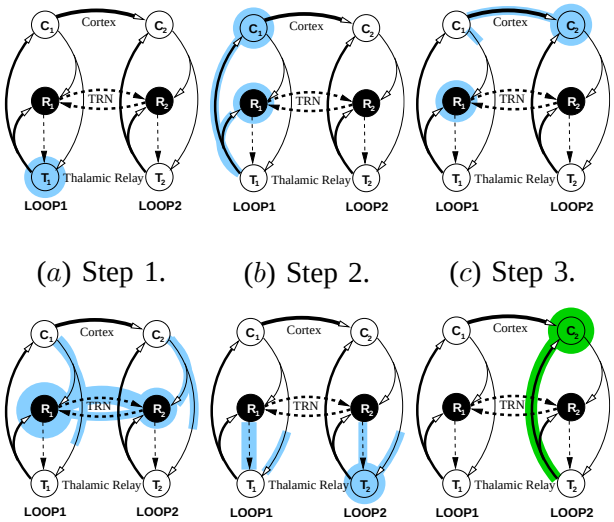
Fig. 2. **The Thalamocortical Network.** A schematic diagram of the thalamocortical network is shown (see [6] for a review). Solid edges with open arrows are excitatory connections, and dashed edges with closed arrows are inhibitory connections. The thalamus is a centrally located nucleus in the brain, and the dorsal part of it is covered with an inhibitory network of neurons called the Thalamic Reticular Nucleus (TRN). Ascending and descending connections all branch out and stimulate reticular neurons, and the reticular neurons send inhibitory connections to the thalamic relay neurons. Two thalamocortical loops, loop1 (T_1-R_1-C_1) and loop2 (T_2-R_2-C_2), are shown. The two loops are connected to each other in the cortex and within the TRN.

and reticular inhibition cancels out cortical feedback to the thalamic relays when both ascending and descending spikes are received at the TRN. On the other hand, when only descending (i.e., corticothalamic) feedback is received, TRN's inhibition on the thalamic relay is weak, and the relay neuron is allowed to fire in response to the cortical feedback, thus invoking the cortical neuron for the second time around.

Fig. 3 shows a detailed activation sequence of this mechanism. The example shows a case where only one of the thalamocortical loops (loop1) received sensory input, thus demonstrating how *purely cortex-driven* activity can be singled out: The cortical feedback to the thalamus that survives the filtering is allowed to reactivate the cortex.

As for promoting the *relatively less input-driven* activity, we can think of a case when loop1 receives a strong sensory input and loop2 receives a weak sensory input. This time, both reticular neurons R_1 and R_2 will be highly activated, but due to the *disinhibition* (inhibition of an inhibitory neuron results in less inhibition at the target neuron) between the two, R_2 (which is strongly inhibited by R_1) cannot cancel out cortical feedback from C_2 to T_2. On the other hand, R_1, with its strong activity, will cancel out feedback from C_1 to T_1.

The description of the procedure sounds straightforward, but in fact there are hidden assumptions (mostly regarding the temporal aspect) that are not quite obvious from the above. The next section will explicate these



(a) Step 1. (b) Step 2. (c) Step 3.

(d) Step 4. (e) Step 5. (f) Step 6.

Fig. 3. **Analogical Completion and Filtering in the Thalamocortical Circuit.** An activation sequence of two thalamocortical loops (loop1 and loop2) are shown. Active parts of the circuit at each step are highlighted. (a) Initially, only T_1 receives an afferent sensory input. (b) T_1 invokes R_1 and C_1. (c) The cortical neuron C_1, through fast connections, invokes another cortical neuron C_2. C_1 also sends out feedback to R_1 and T_1, but these connections are slow and the spike can only travel a short distance in the same time. Note that R_1 retains the level of excitation because of its slow dynamics. (d) Cortical feedback from both C_1 and C_2 arrives at the TRN, and adds to the existing activity at TRN. Reticular neurons R_1 and R_2 inhibit each other through fast connections. (e) At the time the cortical feedback arrives at T_1 and T_2, the reticular neurons exert inhibition on the thalamic relays. R_1, driven by both afferent input and cortical feedback exerts strong inhibition on T_1, effectively canceling out the cortical feedback from C_1. On the other hand, R_2 was only driven by the cortical feedback, and it is not enough to cancel out feedback from C_2 at the thalamic relay T_2. Thus T_2 is permitted to fire again. As a result, C_2 will be the only active neuron in the cortex in the next iteration.

hidden assumptions and provide justifications for them.

V. Functional Requirements of the Proposed Model

As mentioned above, there are specific assumptions that need to hold for the scenario described in the previous section to work (cf. Fig. 3):

1) reticular neurons must have a slow dynamics (Fig. 3*b*–*d*);
2) inhibition between reticular neurons must be strong (Fig. 3*e*);
3) either the cortico-cortical connections must be very fast or the corticothalamic feedback connections must be slow (or both), compared to each other (Fig. 3*c*–*d*); and

4) interaction between reticular neurons must be fast (Fig. 3*d*).

These conditions must hold due to the following functional requirements of the proposed model (in the same order as above):

1) reticular neurons need to retain the ascending excitation level to strongly inhibit the thalamic relay later when the cortical feedback comes around (R_1 in Fig. 3*b*–*d*);
2) reticular activity must not be strong enough to cancel out cortex-driven cortical feedback to the thalamic relay (R_2 in Fig. 3*e*);
3) both input-driven cortical activity (C_1) and cortical activity (C_2) driven by that input-driven cortical activity must send feedback to the thalamus and TRN at approximately the same time (Fig. 3*c*–*d*); and
4) reticular neurons must rapidly adjust their activity level before inhibiting the thalamic relays (R_1 and R_2 in Fig. 3*d*).

Most of these conditions were described in [26], [27], but some of those that were only implicit in the earlier description are made more explicit here.

As it turns out, all of these conditions have experimental support (in the same order as above):

1) reticular neurons activate and deactivate on a slow timescale compared to thalamic relays [36], [37];
2) reticular neurons are harder to depolarize than thalamic relays [37], which may be due to inhibition between reticular neurons;
3) corticothalamic feedback connections are unmyelinated (i.e., very slow) [38]; and
4) gap junctions have been found between reticular neurons [39], suggesting that the interaction between reticular neurons may be rapid.[1]

In the following, the conditions listed above will be tested in a computational implementation of the model.

[1]It is controversial whether gap junctions can directly carry out disinhibition as required in here. However, the existence of gap junctions shows that reticular neurons need to communicate at a high speed, suggesting that other connections between reticular neurons may have to be fast as well.

VI. Model Description

A network of leaky integrate-and-fire neurons [44] was constructed to test the *analogy hypothesis* summarized in the previous section.

Six neurons of three types (T: thalamic relay; R: thalamic reticular neuron; C: cortical neuron) were connected according to the diagram in Fig. 2 (with the additional reciprocal connection from C_2 to C_1). For each neuron i, the membrane potential V_i evolved according to the following dynamic equation:

$$C_i \frac{dV_i}{dt} = I_i(t) - \frac{V_i}{R_i}, \tag{1}$$

where C_i is the membrane capacitance, R_i the resistance, and $I_i(t)$ the input contribution to neuron i at time t. When V_i reaches a threshold value θ_i, a spike is generated and V_i is reset to the baseline (0.0). A spike generated by a presynaptic neuron j results in a postsynaptic potential (PSP) s_{ij} at a target neuron i, which is set to 1.0 at the moment the spike is received and is decayed over time as follows:

$$\frac{ds_{ij}}{dt} = -\frac{s_{ij}}{\tau_i}, \tag{2}$$

where τ_i is the membrane time constant of the PSP of the neuron i. The input contribution $I_i(t)$ to the neuron i at time t is defined as follows:

$$I_i(t) = \sum_{j \in \mathcal{N}_i} w_{ij} s_{ij}(t - \delta_{ij}), \tag{3}$$

where $\mathcal{N}_i$ is the set of neurons sending spikes to neuron i (see Fig. 2 for the connectivity); w_{ij} is the connection weight from neuron j to i (the sign is negative if j is an inhibitory neuron); and $s_{ij}(t - \delta_{ij})$ is the PSP generated in neuron i by a spike from neuron j with a conduction delay of δ_{ij}. See Section VII, Tables I and II for the exact parameter values.

VII. Experiments and Results

Six experiments were conducted with the model described above to test the assumptions and predictions in the hypothetical model (Fig. 2; [26], [27]). The common setup for these experiments is summarized in Section VII-A. The first three experiments (Sections VII-B to VII-D) tested if the model tuned according to the conditions listed

in Section V can filter out input-driven or less input-driven cortical activity and just leave either the purely cortex-driven or relatively more cortex-driven activity in the cortex. The remaining experiments (Sections VII-E to VII-G) systematically tested the effects of violated conditions on the function of analogy.

A. General Experimental Setup

TABLE I

NEURON PARAMETERS

Parameter	Thal. Relay (T_i)	TRN (R_i)	Cortex (C_i)
Capacitance C_i	0.3	**0.6**	0.3
Resistance R_i	3.0	3.0	3.0
Threshold θ_i	0.25	0.25	0.25
PSP time constant τ_i	0.05	0.05	0.05

TABLE II

CONNECTION PARAMETERS

Weight w_{ij}	T_i	R_i	C_i
T_j		1.0	1.0
R_j	**2.0**	**10.0**	
C_j	1.0	1.0	**0.9**

Delay δ_{ij}	T_i	R_i	C_i
T_j		2.0	2.0
R_j	2.0	**0.2**	
C_j	**4.0**	2.0	**0.2**

Tables I and II list the neuron and connection parameters (the units are arbitrary). The parameters are fairly uniform, except for the values in **bold-type** indicating a deviation from the default parameters. The deviations are not arbitrary, and are specifically required as discussed in Section V (in the same order):

1) the membrane capacitance C_i of a reticular neuron R_i must be large ($= 0.6$, twice the default value) so that the membrane has slow dynamics;
2) the synaptic weight between reticular neurons R_j to R_i must be large ($= 10.0$, ten times the default value);
3) the conduction delay δ_{ij} from a cortical neuron C_j to a thalamic relay T_i must be large ($= 4.0$, twice the default value); the conduction delay δ_{ij} from a cortical neuron C_j to another cortical neuron C_i must be very small ($= 0.2$, 1/10th the default value); and

4) the conduction delay δ_{ij} from a reticular neuron R_j to R_i must be small ($= 0.2$, 1/10th the default value).

The two exceptions are the R_i to T_i connection weight ($= 2.0$, twice the default value) and the cortico-cortical connection weight ($= 0.9$, 90% of the default value). These parameters were found empirically, and the significance of the relative magnitude of these parameters needs to be further investigated. A possible reason for R_i to T_i connection weight being high is that this inhibitory influence should be strong enough to cancel out input-driven cortical feedback. As for the cortico-cortical connection weight, it has to be lower than 1.0 to avoid a positive feedback loop that can potentially make the activity to go out of control.

The same set of parameters were used without modification in all the experiments, except for the following: (1) in Sections VII-C and VII-D, where the R_i to T_i connection weight was increased; and (2) in Sections VII-E to VII-G, the temporal parameters were altered to systematically test the effects of violated conditions.

B. Experiment 1: Input-Driven vs. Cortex-Driven Activity in a Single Loop

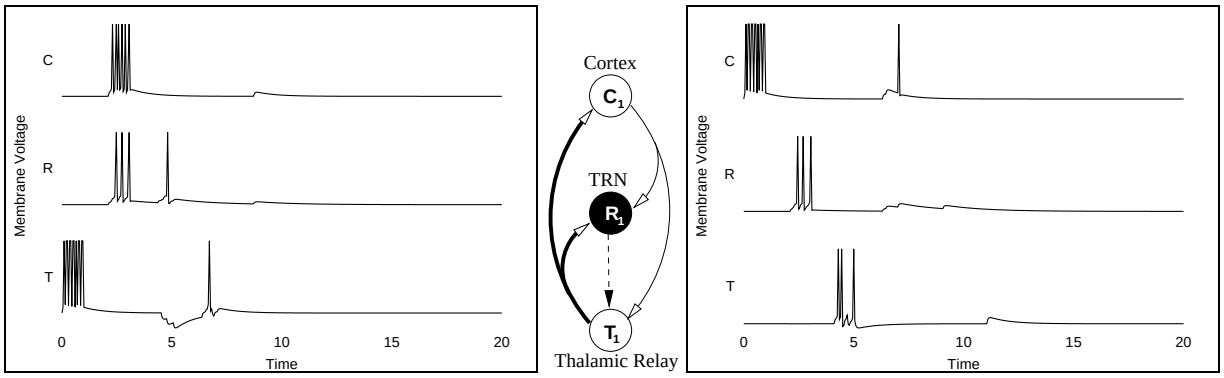
To independently test the function of a *single* thalamocortical loop, depolarizing currents were injected at two different locations of the loop, (1) the thalamic relay T and (2) the cortical neuron C, in two separate experiments. The thalamic injection emulated the input-driven condition, and the cortical injection the cortex-driven condition. The results are shown in Fig. 4.

In the input-driven case (Fig. 4*a*), the cortex activates at time $t = 3$ in response to the thalamic drive, but the corticothalamic feedback at time $t = 7$ is canceled out (i.e., filtered out) by the reticular inhibition. As a result, the cortical neuron fails to reactivate.

In contrast, for the cortex-driven case (Fig. 4*b*), the corticothalamic feedback to T at time $t = 5$ is strong enough to survive the weak reticular inhibition and successfully reactivates the cortex at time $t = 7$. These initial results demonstrate the ability of the model to implement a primitive form of analogical process.

C. Experiment 2: Input vs. No-Input Condition in a Connected Pair of Loops

In this experiment, a pair of connected thalamocortical loops was simulated to test whether *purely cortex-driven* activity can be singled out in the model. The two loops were connected as in Fig 2 with the addition of a reciprocal cortico-cortical connection from C_2 to C_1. To suppress hyperactivity in the thalamus caused by the strong feedback



(a) Thalamus-driven (b) Cortex-driven

Fig. 4. **Thalamus- vs. Cortex-Driven Activity.** Membrane potential traces for a thalamic relay (T), a reticular neuron (R), and a cortical neuron (C) in a *single* loop are shown from the bottom row to the top row in each panel. The x-axis is time and the y-axis is the membrane potential. A depolarizing current of magnitude 1.0 and duration 1 was either injected in (a) the thalamic relay, emulating an input-driven condition, or in (b) the cortical neuron, emulating a cortex-driven condition. Only for the cortex-driven case (b), the initial cortical burst of activity can reactivate the cortical neuron through the corticothalamic loop ($t = 7$). Note that the plot shows the V_i trace (equation 1) with spikes added at the moment of threshold crossing. Also note that (a) and (b) are from two independent experiments.

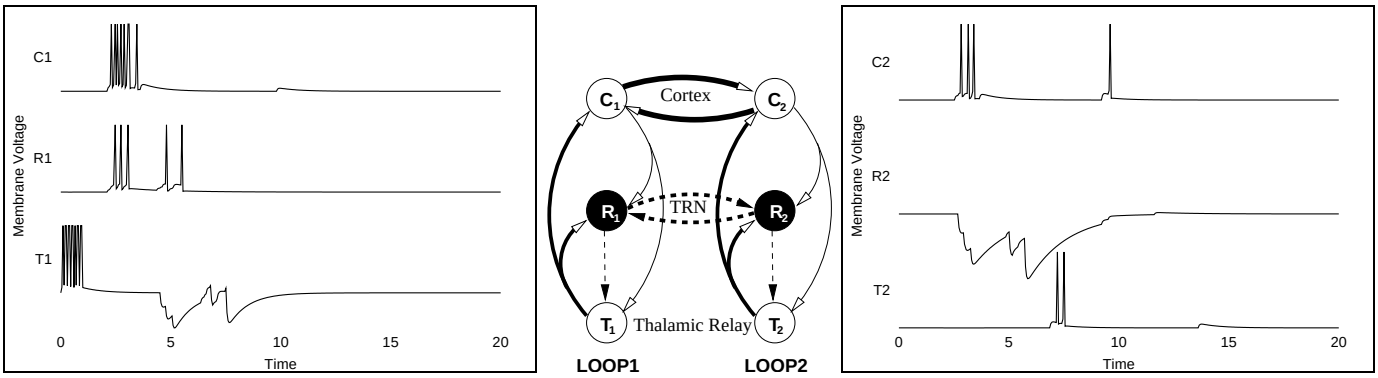
from the cortex due to this recursive cortico-cortical activation, the connection weight from R_i to T_i was increased to 5.0. All other conditions were identical to that of Section VII-B.

Input current was only injected into loop1 thalamic relay T_1 to test the input (loop1) vs. no-input (loop2) condition. The results are shown in Fig. 5. For the initially input-driven loop (loop1; Fig. 5a), the cortical burst of activity at time $t = 3$ is unable to reactivate the cortex. However, for the initially cortex-driven loop (loop2; Fig. 5b), the cortical burst at time $t = 3$ induced by loop1 is able to reactivate the cortex at $t = 10$ through the corticothalamic feedback. Thus, the model demonstrates selectivity for purely cortex-driven activity.

D. Experiment 3: Strong- vs. Weak-Input Condition in a Connected Pair of Loops

The setup in this experiment was identical to Section VII-C, except for the input condition. For this experiment, loop1 thalamic relay T_1 was injected with a depolarizing current of magnitude 2.0, and T_2 of loop2 with a lower magnitude ($= 1.0$). Thus, a strongly input-driven vs. weakly input-driven condition was setup to test whether *relatively less input-driven* cortical activity can be promoted in the model. The results are shown in Fig. 6.

The results are similar to those in Section VII-C. Cortical activity in the more input-driven loop1 is unable to



(a) Loop1: Input=1.0 (b) Loop2: Input=0.0

Fig. 5. **Input vs. No-Input Condition.** Membrane potential traces for neurons in *two* connected thalamocortical loops are shown, one in (a) and the other in (b). The two loops are wired as shown in Fig. 2, with the addition of a reciprocal cortico-cortical connection from C_2 to C_1. The voltage traces for the neurons in the loops are numbered accordingly. (a) A depolarizing current of magnitude 1.0, duration 1 was injected in T_1. (b) No current was injected anywhere in the loop, thus all activities were initially driven by the cortico-cortical connection from C_1 to C_2 at time $t = 3$. Only the cortex-driven cortical activity (C_2) is able to reactivate the cortex through feedback to the thalamus ($t = 10$ in b). Note that the above are results from a single experiment, unlike in Fig 4 where there were two.

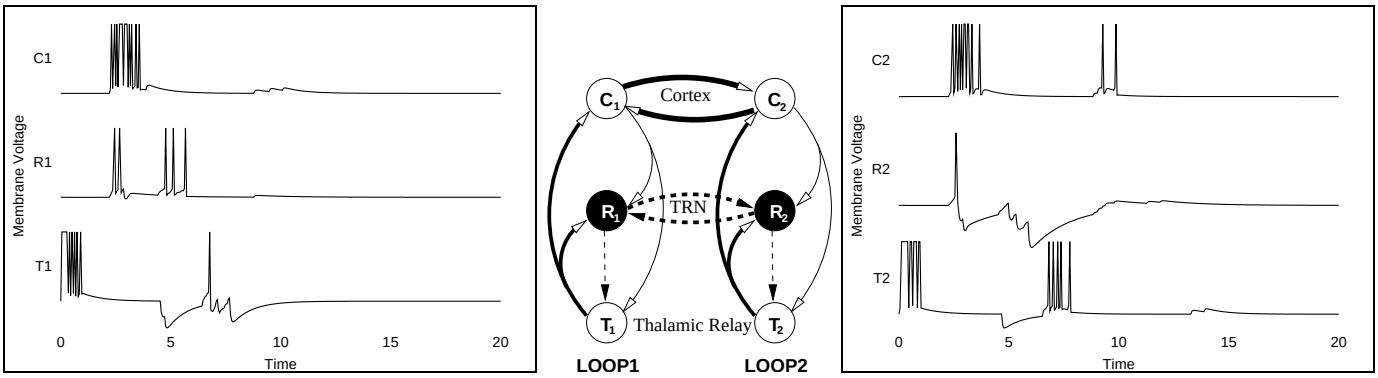
to reactivate the cortex (Fig. 6a), but the less input-driven loop2 is able to reactivate the cortex ($t = 10$; Fig 6b). These results show that the model has selectivity for *less* input-driven cortical activity.

Note that in this case, disinhibition between reticular neurons plays an important role in allowing loop2 to reactivate the cortex, despite the fact that loop2 was also input-driven: Loop1 reticular neuron R_1 fires more vigorously than R_2 of loop2, and when they fire, they strongly inhibit R_2 ($t = 3$ and beyond; Fig. 6b), thus weakening the inhibition exerted by R_2 on T_2. Compare this result to Fig 5a, where the same amount of input current injected in the thalamic relay resulted in the failure of the cortical neuron to reactivate.

The results suggest that relatively less input-driven cortical feedback can be detected and promoted in the proposed model, without changing any parameter (cf. Section VII-C). In the following, we will see how violating model assumptions alter the behavior of the thalamocortical network.

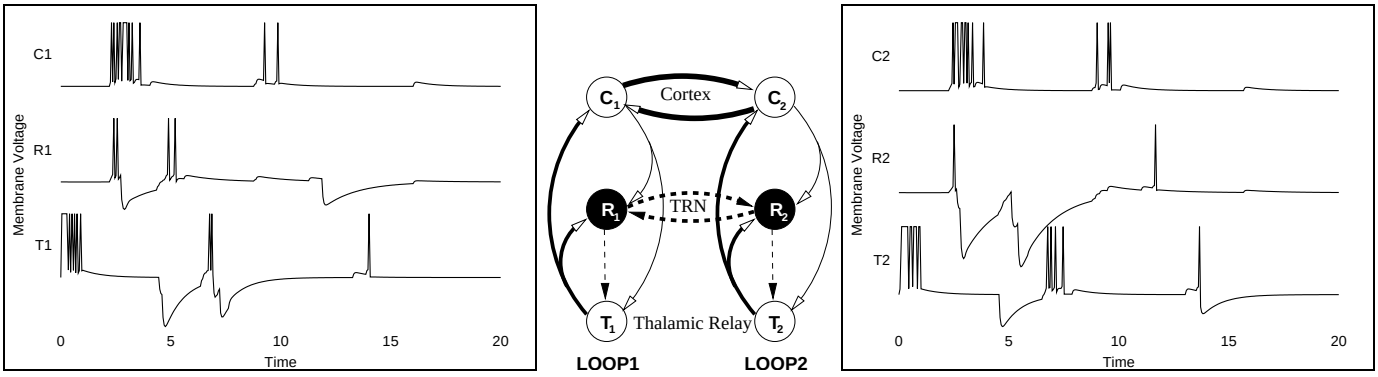
E. Experiment 4: Fast Reticular Neuron Dynamics

Previously in Section V, we reasoned that the reticular neurons need to operate on a slow timescale because of the need to integrate both ascending thalamic contributions and descending cortical feedback. In this experiment,



(a) Loop1: Input=2.0 (b) Loop2: Input=1.0

Fig. 6. **Strong- vs. Weak-Input Condition.** Membrane potential traces for two thalamocortical loops with an identical setup as in Fig. 5 are shown. The only difference was the magnitude of depolarizing current injected in loops 1 and 2. (a) A depolarizing current of magnitude 2.0, duration 1 was injected in T_1. (b) A depolarizing current of magnitude 1.0, duration 1 was injected in T_2. Thus, both loops were input-driven, but to a different degree. Only the less input-driven cortical activity (C_2) is able to reactivate the cortex through feedback to the thalamus ($t = 10$ in b).



(a) Loop1: Input=2.0 (b) Loop2: Input=1.0

Fig. 7. **Fast Reticular Neuron Dynamics.** Membrane potential traces for two thalamocortical loops with an identical setup as in Fig. 6 are shown. The only difference was the lower membrane capacitance ($C_i = 0.5$) in the reticular neurons (R_i), which makes the membrane dynamics faster. The reticular neurons fail to integrate the thalamic and cortical contributions, and thus timely inhibition is interrupted.

we will investigate the effect of faster reticular dynamics on the behavior of the model. The prediction is that analogical filtering, which depends on such an integration, will not work properly.

To test the effect, the capacitance of the reticular neurons was lowered to 0.2. All other simulation conditions were identical to that of Section VII-D. The resulting behavior, shown in Fig. 7, is a rapid activation and deactivation of the reticular neurons which causes the reticular neurons to fail to integrate the contributions from the ascending and

feedback connections in a timely manner. This leads to the failure of the reticular neurons to inhibit the thalamic relay neurons at the time cortical feedback arrives at the relay neurons (Fig 7a at $t = 7$ and 14). Compared to Fig. 6, the firing of the thalamic relay neurons are markedly more robust, thus it suggests that the reticular neurons are not performing the analogical filtering function properly. The results confirm that reticular neurons having a slow dynamics is important for integrating thalamic contribution and cortical feedback to perform analogical filtering.

F. Experiment 5: Weak Synaptic Strength Between Reticular Neurons

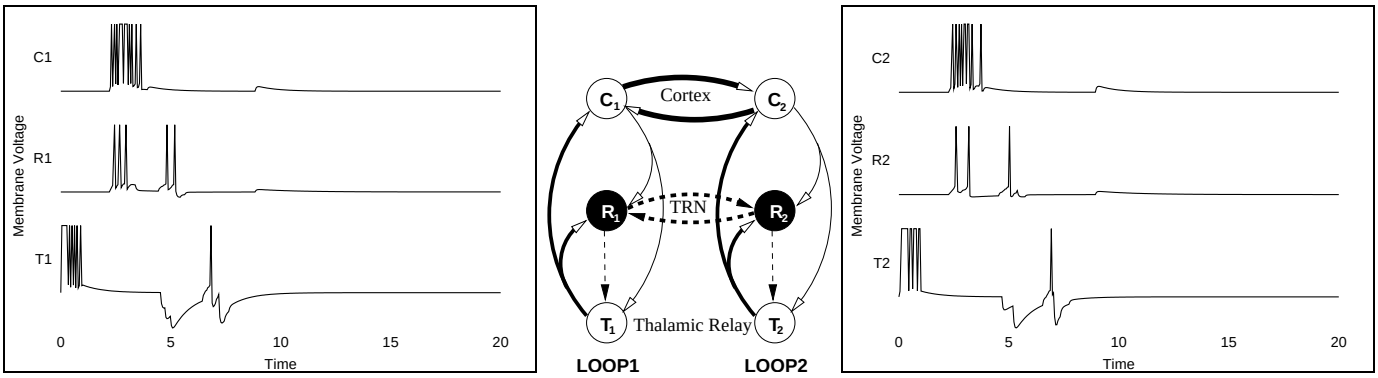
If the reticular inhibition exerted on the thalamic relay neurons is too strong, cortical feedback can never survive the filtering at the thalamus whether or not that feedback was input-driven or cortex-driven. To ensure that the inhibition is not too strong, the reticular activity must not be too strong. For this, mutual inhibition within the TRN can play a significant role, and in Section V, it was speculated that this inhibition should be strong. To test this prediction, the connection weight between the reticular neurons was decreased to 2.0. All other conditions were the same as in section VII-D.

The results are shown in Fig. 8. When we compare these results to Fig. 5 (where the intra-reticular weight was high) we can notice that loop2 fails to reactivate the cortex. This is because the reticular disinhibition was not strong enough to allow the C_2 to T_2 feedback to activate T_2 strongly enough. The results suggest that reticular neurons need to inhibit each other strongly to satisfy the functional requirement of the analogy framework, and this can possibly be the reason why it is experimentally observed that reticular neurons are hard to depolarize [37].

G. Experiment 6: Altered Delay Conditions

As shown in Section V, the model requires specific delay conditions in two cases: (1) larger delay in corti-cothalamic feedback compared to cortico-cortical connections and (2) small delay between TRN neurons. In this section, the effect of violating these delay conditions will be investigated. Two experiments were conducted where all parameters were identical to those in Section VII-D, except for the particular delay condition being considered.

In the first experiment, the cortico-cortical connections were made slower ($= 1.0$) so that the delay is similar to that of the thalamocortical feedback. Fig. 9 summarizes the results. Because of the longer delay in the cortex, the cortical feedback arrives at the thalamus out of sync with the reticular inhibition, and this causes the tightly



(a) Loop1: Input=2.0 (b) Loop2: Input=1.0

Fig. 8. **Weak Synaptic Strength Between Reticular Neurons.** Membrane potential traces for two thalamocortical loops with an identical setup as in Fig. 6 are shown. Only one parameter was altered: The connection weight between reticular neurons was decreased to 2.0. Due to the weaker disinhibition effect, loop2 reticular neuron generates more activity to suppress the thalamic relay. As a result, loop2 fails to reactivate the cortex.

coupled behavior of the thalamocortical network to break down. For example, the cortical and thalamic activities beyond $t = 5$ in the two loops are out of phase.

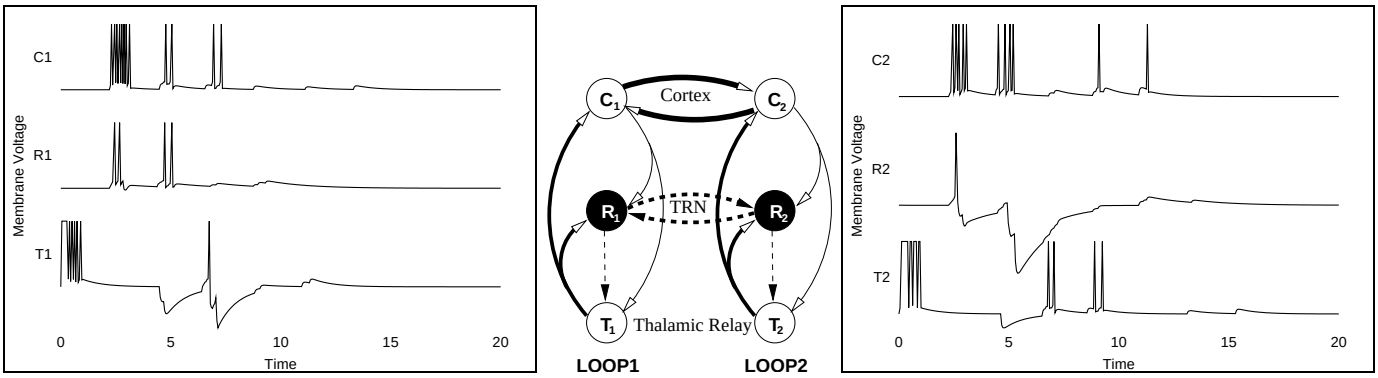
On the other hand, in the second experiment, the axonal delay between reticular neurons within the TRN was made longer. As shown in Fig. 10, delayed inhibition within the TRN allows for a large amount of initial activity in the TRN ($t = 3$ to 5), causing the reticular neurons to strongly inhibit the thalamic relays. Because of this, the cortical feedback to the thalamic relays are totally suppressed ($t = 7$). These two experiments show why the cortico-cortical connections and intra-TRN connections should be fast, if the analogy model is to properly function.

The results show that specific delays found in the thalamocortical network support analogical completion and filtering, and altering these delays can cause the function of analogy to break down.

H. Summary of Results

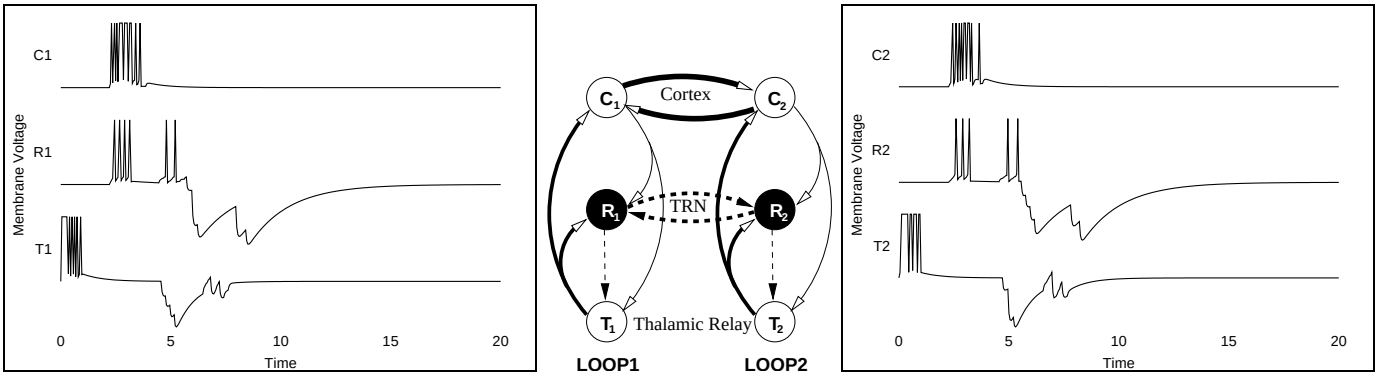
In summary, the thalamocortical model functioned as predicted by the *analogy hypothesis*, with a set of parameters derived from functional, anatomical, and physiological considerations. The model was successful in detecting and promoting (1) purely cortex-driven cortical activity, and (2) relatively less input-driven cortical activity, which forms the basis of the proposed model of analogical processing in the thalamocortical network (Sections VII-B to VII-D).

Violated model assumptions regarding temporal parameters such as membrane time constant and axonal delay



(a) Loop1: Input=2.0 (b) Loop2: Input=1.0

Fig. 9. **Longer Cortico-Cortical Axonal Delay.** Membrane potential traces for two thalamocortical loops with an identical setup as in Fig. 6 are shown. The only difference was the cortico-cortical axonal delay, which was longer ($= 1.0$). The result shows that the phases of loop1 and loop2 activities start to drift and become irregular.



(a) Loop1: Input=2.0 (b) Loop2: Input=1.0

Fig. 10. **Long Axonal Delay Between Reticular Neurons.** Membrane potential traces for two thalamocortical loops with an identical setup as in Fig. 6 are shown. All parameters were the same except for the delay between reticular neurons, which was increased to 1.5. Because of the delayed inhibition, the disinhibition effect did not happen in time to allow loop2 to reactivate the cortex.

caused the model to fail to function properly as required by the framework of analogy (Sections VII-E to VII-G). These results suggest that the temporal parameters found in experimental observations may have such specific values for a very good reason, i.e., to promote the processing of analogies through completion and filtering.

VIII. Discussion

The main contribution of this paper was establishing a connection between (1) activeness, (2) analogy, (3) thalamus and TRN, and (4) temporal properties of neural circuits. Taken apart from each other, we can see that an

enormous amount of research is going on in parallel in all of the four areas above. For example, active schemas [45] and the role of action in perception [46], [47] have gained much interest lately, where the feedforward, passive view was critiqued [48]. In analogy research, several different perspectives have emerged: analogy as a higher level perception [30], [34], [35], analogy through structure mapping [49], analogy by constraint satisfaction [33], and analogy using holographic representations [29]. On the other hand, a separate line of research is going on in neuroscience where specific anatomical and physiological measures are gathered from the thalamus and its surrounding structures [14], [5], [18], [19], [21], [8], [12]. Based on these, various theories have been advanced, including attention, prediction, action selection, sleep/wake, and awareness and consciousness as discussed in Section I. The temporal properties of neurons and network of neurons are also heavily studied in both the experimental and the modeling and theory communities. Various temporal aspects of neuronal signaling is studied, such as the role of synchrony [50], [51], [52], [53], [54], and how to understand and decode the temporal code of the neurons [55], [56]. Although all of the four research areas above look rather unrelated at first, under the framework of analogy presented in this paper, things come together nicely and a coherent explanation becomes possible. If all parties involved can recognize the relevance of the developments in the other areas, a productive collaboration can be initiated.

The proposed model is not without limitations, realizing which naturally leads into several promising future directions. The first thing to notice is that the model only supports a limited form of analogy, thus is not able to deal with structural analogies [57], [26], [27]. Also, it produces a set of potential answers and not the exact answer, thus further investigation may be necessary if the model is to be applied in solving common analogy problems. To efficiently deal with structural analogies, the ordering of input must be taken into account, and for this, the involvement of the prefrontal cortex must be assessed [58]. This leads to the question about the role of higher-order relays in the anterior thalamus [21]. Higher-order relays do not receive any afferent input from the peripheral nervous system which is quite distinct from the model in this paper. What would it mean to ask “what is the less input-driven activity” in this case, when there is no input at all? It would be quite interesting to see how the model of analogy can be adjusted to that kind of anatomy. Another limitation is the scale of the model: The maximum number of neurons used in the current study was only 6. To study a larger scale behavior, scaling

up is inevitable. This brings up another interesting point. In the current model, each loop consisting of individual neurons took on a specific function, but is it also possible to have synchronized populations of thalamocortical loops working as group? Can completion and filtering be carried out among these synchronized populations? The thalamus plays a critical role in modulating synchrony in the cortex [13], [59], and these synchronized populations can be seen as a higher abstraction of a single thalamocortical loop. Thus, extending the current model to treat synchronized populations as a single unit may also be possible.

Finally, we can derive several predictions from the model presented in this paper. The specific assumptions on connection weight and temporal parameters described in Section V and the computational results already form a concrete prediction. Experiments similar to these can be conducted on live tissue blocks in vitro or even in vivo, and the course of activity in the thalamocortical network can be compared to the results presented in this paper. The model makes very specific predictions on the expected behavior when the normal parameters are disrupted (Sections VII-E to VII-G). At a higher level, the model predicts that purely or more cortically driven activity in the cortex will reactivate while input-driven activity in the cortex will not reactivate. This kind of prediction can be tested using fMRI devices while the subject is performing a cognitive task involving analogical abilities. Activities at the thalamus and the cortex can be measured simultaneously, and to get a better temporal resolution, EEG studies can be done in parallel. Through such a study, cognitive scientists can gain crucial insights into the neural basis of analogy, and neuroscientists can better understand the function of the brain, within the framework of analogy.

IX. Conclusion

In this paper, the thalamocortical model of analogy [26], [27] was tested computationally. A network of integrate-and-fire neurons was built and tuned based on functional, anatomical, and physiological considerations. The results showed that active completion and filtering for less input-driven activity, which forms the basis of analogy, arise in the model. Various temporal parameters, tuned based on anatomical and physiological data, turned out to be crucial in enforcing the proper function of the model. This suggests that these parameters need to be studied together, not separately, under a general functional framework, such as the analogy model. The model suggests that analogy can potentially be the mode of integration among multiple interacting cortical maps, and that further investigation into the thalamocortical network under the framework of analogy may be a worthwhile endeavor.

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