

Processing of Analogy in the Thalamocortical Circuit

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Abstract—The corticothalamic feedback and the thalamic reticular nucleus have gained much attention lately because of their integrative and modulatory functions. A previous study by the author suggested that this circuitry can process analogies (i.e., the *analogy hypothesis*). In this paper, the proposed model was implemented as a network of leaky integrate-and-fire neurons to test the *analogy hypothesis*. The previous proposal required specific delay and temporal dynamics, and the implemented network tuned accordingly functioned as predicted. Furthermore, these specific conditions turn out to be consistent with experimental data, suggesting that a further investigation of the thalamocortical circuit within the *analogical framework* may be worthwhile.

I. INTRODUCTION

Understanding how cortical maps in the brain *interact* with each other to generate complex 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 [2, 11], we still lack the understanding of how these maps work as an integrated system.

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 [23] for a review). The thalamus was previously thought of as a passive relay station for sensory-motor signals, however, this explanation was not satisfactory because of the existence of massive feedback from the cortex and a thin inhibitory network covering the thalamus called the thalamic reticular nucleus (TRN; see [6] for a review).

Previous work by the author suggested that the interplay between the thalamus, TRN, and the cortex may be implementing a function of analogy (the *analogy hypothesis*; [3, 4]). Analogy plays an important role in human perception and cognition [10, 12, 13, 16, 20], and its ability to cross domain boundaries may be critical in the integrative operation of cortical maps. Experimental observations by Crabtree and Isaac [6] nicely demonstrate the cross-modal nature of interaction in the thalamus and in the TRN, and these results can provide experimental grounds for the analogy hypothesis.

This paper presents the first computational implementation of the hypothetical model by the author initially described in [3, 4] and compare the results with the previous predictions. 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.

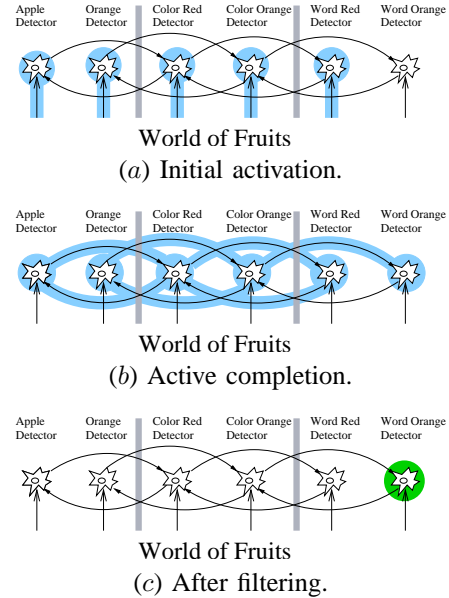


Fig. 1. **Analogy Through Active Completion and Filtering.** A simple brain with 6 detector neurons is shown. Environmental input comes in from the bottom (*World of Fruits*), and the neurons are linked with directed arcs that show the learned association between the detectors. From (a) to (c) shows 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.

II. ANALOGY THROUGH ACTIVE COMPLETION AND FILTERING

Neurons can be seen as processing information, i.e., producing output given a certain input. However, in this perspective, the output generated by neurons is *passive*, in the sense that they need further *interpretation* as all information or data does. A slight change of perspective allows us to view neurons as *active elements*, in the sense that neurons *actively invoke* other neurons. From this, it was shown that we can derive the function of analogy [3, 4]. The previous papers showed that *completion* and *filtering* are necessary for such active elements to implement the function of analogy. In the following, an example will briefly illustrate the proposed mechanism.

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. For example, the apple-object detector has a strong connection to the color-red detector, etc.

Now consider the proportional analogy question `apple:orange::word:red:?`. Fig. 1 shows an activation sequence of the simple brain to the input question. Initially, the first 5 neurons are activated in response to the input (Fig. 1a). Next, through the relational links, other cortical neurons are invoked (*completion*; Fig. 1b). Last, after *filtering* for the purely cortex-driven activity in the cortex, only the word-orange detector remains active (Fig. 1c). We can see that word-orange is precisely the answer to the analogy question we posed in the beginning, and completion and filtering produced that answer.

However, simple filtering for only purely cortex-driven activity is insufficient for analogy answers containing items 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. In this case, relaxing the filtering criteria allows us to get to the answer: find *relatively less input-driven* cortical activities [3, 4].

In summary, simple analogies can be processed by active completion and filtering. In the next section, we will see how such completion and filtering can be neurally implemented.

III. ACTIVE COMPLETION AND FILTERING IN THE THALAMOCORTICAL LOOP

How can active completion and filtering be implemented in the neural circuits of the brain? Previous work by the author proposed that the thalamus, thalamic reticular nucleus (TRN), and the cortex may be involved in completion and filtering [3, 4]. Fig. 2 shows a schematic diagram of the thalamocortical circuit based on known anatomy and physiology [6, 21]. 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. However, 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 [3, 4], 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, 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

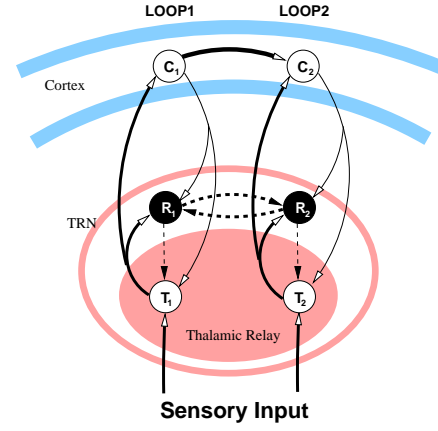


Fig. 2. **The Thalamocortical Circuit.** A schematic diagram of the thalamocortical circuit is shown (see [21] for a review). Solid edges with open arrows are excitatory connections, and dashed edges with closed arrows are inhibitory connections. Two thalamocortical loops (T_1 - R_1 - C_1 and T_2 - R_2 - C_2) are shown. 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.

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*¹ 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 .

A. Functional Requirements of the Proposed Circuit

There are specific assumptions that need to hold for the scenario described above to work:

- 1) reticular neurons must have a slow dynamics (Fig. 3b-d);
- 2) synaptic strength between TRN neurons must be strong (Fig. 3d);
- 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. 3c-d); and
- 4) interaction between reticular neurons must be fast (Fig. 3d).

These conditions must hold due to the following functional requirements (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. 3b-d);

¹Inhibition of an inhibitory neuron results in net excitation at the target of that neuron.

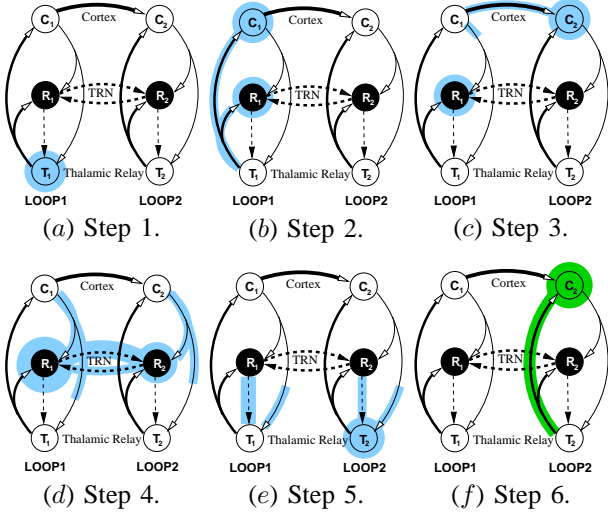


Fig. 3. Analogical Completion and Filtering in the Thalamocortical Circuit. An activation sequence of two thalamocortical loops (loop1 and loop2) are shown. Active parts of the circuit at each step are highlighted. (a) Initially, only T_1 receives an afferent sensory input. (b) T_1 invokes R_1 and C_1 . (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.

- 2) reticular neurons must inhibit each other strongly to effectively disinhibit the thalamic relay for the weakly input-driven case (R_2 in Fig. 3d);
- 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. 3c–d); and
- 4) reticular neurons must rapidly adjust their activity level before inhibiting the thalamic relays (R_1 and R_2 in Fig. 3d).

Most of these conditions were described in [3, 4], 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 [5, 14];
- 2) reticular neurons are harder to depolarize than thalamic relays [14], which may be due to the strong mutual inhibition between reticular neurons;
- 3) corticothalamic feedback connections are unmyelinated (i.e., very slow) [25]; and
- 4) gap junctions have been found between reticular neurons [18], suggesting that the interaction between retic-

ular neurons may be rapid.²

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

IV. MODEL DESCRIPTION

A network of leaky integrate-and-fire neurons [9] 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 connection from C_2 to C_1). For each neuron i , the membrane potential V_i evolved according to the following dynamic equation:

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

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 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:

$$\frac{ds_{ij}}{dt} = -\frac{s_{ij}}{\tau_i}, \quad (2)$$

where τ_i is the time constant of the PSP in the neuron i .

The input contribution $I_i(t)$ to the neuron i at time t is defined as follows:

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

where \mathcal{N}_i is the set of neurons sending spikes to neuron i (see Fig. 2); 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 V, Tables I and II for the exact parameter values.

V. EXPERIMENTS AND RESULTS

Three experiments were conducted with the model described above to test the assumptions and predictions in the hypothetical model (Fig. 2; [3, 4]). The experiments tested if the model tuned according to the conditions listed in Section III-A 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.

A. General Experimental Setup

Tables I and II below list the neuron and connection parameters (the units are arbitrary). The parameters are fairly uniform, except for the figures in **bold-type** indicating a deviation from the default parameters.

The deviations are not arbitrary, and are specifically required as discussed in Section III-A (in the same order):

²It is controversial whether gap junctions can 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.

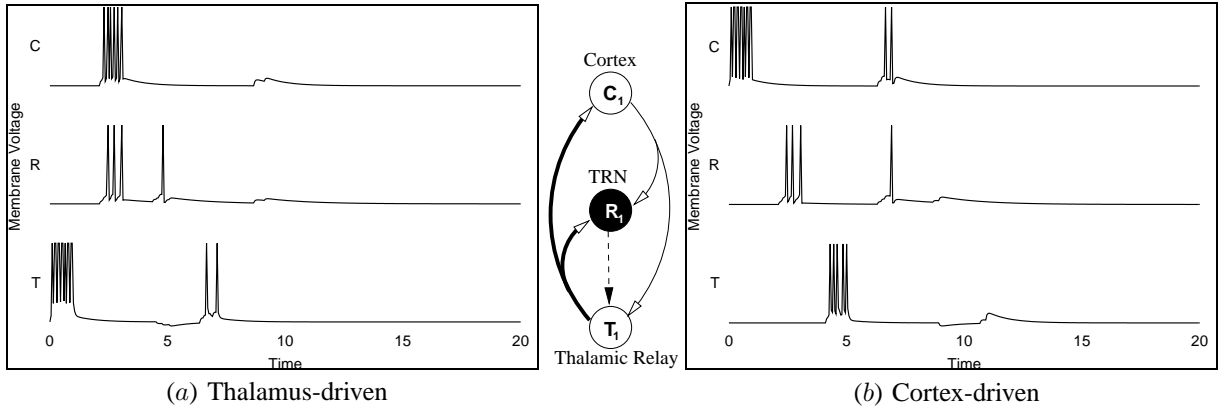


Fig. 4. **Thalamus- vs. Cortex-Driven Activity.** Membrane potential traces for a thalamic relay (T), a reticular neuron (R), and a cortical neuron (C) in a *single* loop are shown from bottom to top in each panel. The x-axis is time and the y-axis is the membrane potential. A depolarizing current of 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. Note that the plot shows the V_i trace (equation 1) with spikes added at the moment of threshold crossing. Note that (a) and (b) are from two independent experiments.

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 |

- 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 connection weight from R_j to R_i must be large enough to have a disinhibitory effect ($= 10.0$, 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).

There are two exception, one is (1) the connection weight C_j to C_i , which was less than 0.0 to avoid hyperactivation through a positive feedback loop; and the other is (2) the connection weight from R_i to T_i , which needs to be strong enough to suppress cortical feedback.

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

sites of the loop in two separate experiments: (1) the thalamic relay T and (2) the cortical neuron C. The thalamic injection tested 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. 4a), 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. 4b), 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 $t = 7$.

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

In this experiment, a pair of 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 an addition of reciprocal cortico-cortical connection from C_2 to C_1 . The parameters were the same as in Section V-B, except for R_i to T_i weight which was increased to 5.0 to counter the increased level of activity due to the recursive corticocortical connection. Input current was only injected to loop1 thalamic relay T_1 . 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 $t = 3$ 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 Pair of Loops

The setup in this experiment was identical to Section V-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 was injected with a current, but with a lower magnitude of 1.0. Thus, a strongly input-driven

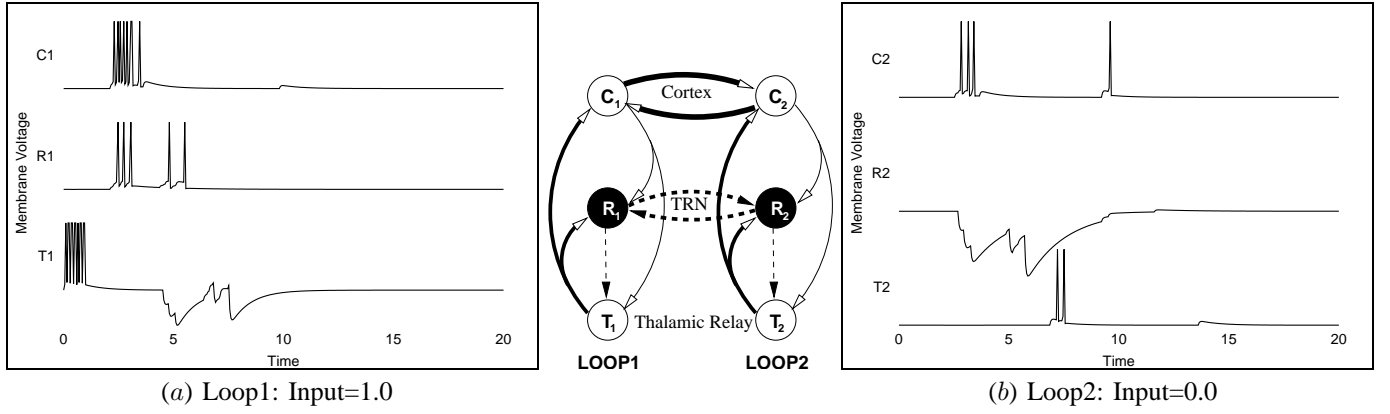


Fig. 5. **Input vs. No-Input Condition.** Membrane potential traces for neurons in *two* connected thalamocortical loops are shown, one in (a) and the other in (b). The two loops are wired as shown in Fig. 2, with the addition of a reciprocal cortico-cortical connection from C_2 to C_1 . The voltage 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 (panel b, time $t = 10$). Note that the above are results from a single experiment.

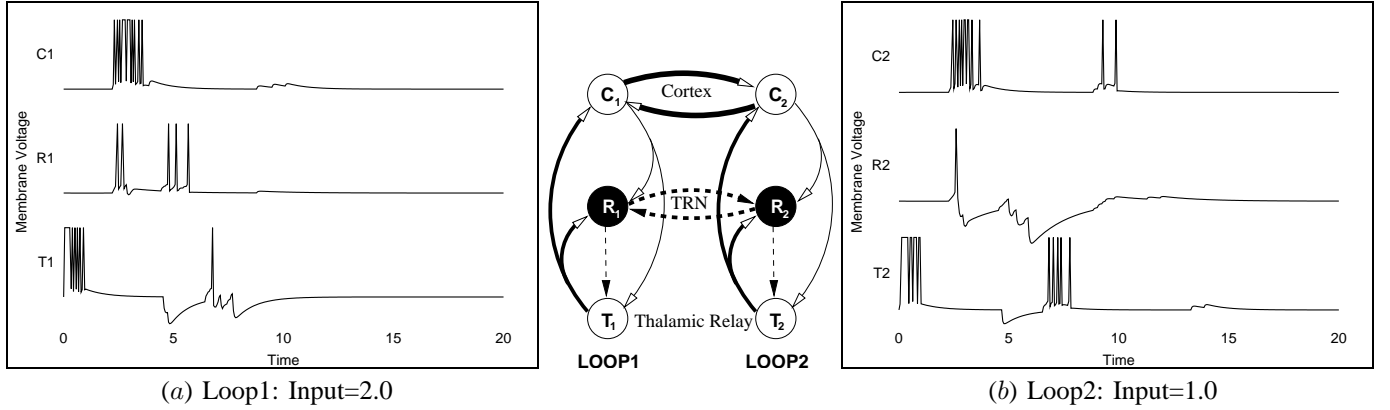


Fig. 6. **Strong- vs. Weak-Input Condition.** Membrane potential traces for two thalamocortical loops with an identical setup as in Fig. 5 is shown. The only difference was the magnitude of depolarizing current injected in loops 1 and 2. (a) A depolarizing current of magnitude 2.0, duration 1 was injected 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 panel b).

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 V-C. Cortical activity in the more input-driven loop1 is unable to reactivate the cortex (Fig. 6a), but the less input-driven loop2 is able to reactivate the cortex ($t = 10$; Fig 6b). Again, these results show that the model has selectivity for less input-driven cortical activity.

Note that in this case, disinhibition between reticular neurons play 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 strongly than R_2 of loop2, and when it fires, it inhibits R_2 ($t = 3$ and beyond), thus abolishing the inhibition exerted by R_2 on T_2 (compare T_1 and T_2 at $t = 7$). Thus, the model demonstrates the ability to promote relatively less input-driven cortical feedback.

E. Summary

In summary, the thalamocortical model functioned as predicted by the *analogy hypothesis*, with a fixed set of parameters

derived from 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 are requirements for the processing of analogy.

VI. DISCUSSION

Two major emphases of this paper are *activeness* and the integrative role of *analogy* in brain function. The two rather abstract notions turn out to have a firm biological ground (i.e., the thalamocortical loop), and through this connection we can begin to take a fresh look at how the diverse cortical maps can interact to give rise to an integrated behavior. Action [1, 15], analogy [10, 12, 13, 16, 20], and the attentive and integrative role of the thalamocortical circuit [6, 17, 19, 21–23] are all related in that respect, and a collective effort in understanding the relationship between these areas will become necessary.

The current model can only address a limited range of analogy problems [3, 4], and these issues need to be resolved. The most prominent issues are that of structured analogy and that of temporal or spatial order. Investigations into these issues will inevitably involve the prefrontal cortex [8], which is believed to be dealing with sequences of events.

Another issue not addressed in the previous papers [3, 4] is that of synchrony [24, 26]. Thalamus plays an important role in synchronization [19], and how analogical processes can interact with synchronized populations of neurons will become an important issue.

VII. CONCLUSION

The current work computationally tested an earlier proposal by the author that the thalamocortical circuit may be performing analogies across cortical maps. A network of integrate-and-fire neurons was built and tuned based on functional 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. These results suggest that further investigation into the thalamocortical mechanism of analogy may be worthwhile.

ACKNOWLEDGMENTS

I would like to thank Bard Ermentrout for his help with XPPAUT [7], in which the model was implemented. This research was supported in part by Texas A&M University, by the Texas Higher Education Coordinating Board ARP/ATP program (#000512-0217-2001), and by the National Institute of Mental Health Human Brain Project (#1R01-MH66991).

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