

ヒックの法則の神経基盤：
 多者択一過程における決定タイミングを説明する皮質回路モデル
 Neural basis of Hick's law:
 Cortical-circuit model accounting for decision timing of multi-alternative decision

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ヒックの法則は、複数のメニューから一つを選択するのに要する時間(反応時間)が選択枝数の対数に比例すると主張する。UI 設計・ユーザビリティ評価においてヒックの法則の実用的価値が広く認められているのとは対照的に、この心理物理経験則の神経基盤はほとんどわかっていない。我々はヒックの法則を再現する皮質回路モデルを構築した。このモデルはさらに、反応時間の変動係数(標準偏差/平均)が選択枝数によらず一定になることを予言する。

1. Introduction

Psychological timing of decision between multiple alternatives is known to follow Hick's law. This empirical law tells that the response time (the amount of time taken for choosing one alternative) is a log-linear function of the number of alternatives [1]. While Hick's law is widely used as a design principle in human-computer interaction literature, surprisingly little is established on its neural basis.

Several authors have proposed mathematical models to examine internal mechanisms underlying Hick's law [2, 3]. However, these models are built on highly abstract schemes with ad hoc assumptions, and their relevance to real brain processes is unclear.

Biophysically realistic cortical-circuit models have also been proposed [4, 5] to specifically account for electrophysiological and behavioural findings of the recent experiment study using two- and four-choice direction discrimination task [6]. Nevertheless, whether these models can reproduce Hick's law is unaddressed.

The purpose of the present study is to propose the first cortical-circuit model that reproduces Hick's law. We also examine statistical properties of response time of multi-alternative decision on the basis of this model.

2. Methods

2.1 Hick's law

Hick's law states that the mean of response time of multi-alternative decision increases log-linearly with the number of alternatives. Here, "mean" refers to the average over multiple trials. Mathematical expression of Hick's law is given by

$$\mu = b \log_2(P+1), \quad (1)$$

where μ , b and P are the mean response time, a suitable constant and the number of alternatives, respectively.

2.2 Model architecture

We consider multiple populations of excitatory neurons, with each population exclusively corresponding to one of multiple alternatives. Each population consists of M subpopulations of N excitatory neurons. These N neurons are located nearby each other in the cortex and are recurrently connected with strong excitatory synapses. Subpopulations in each population, which are not necessarily nearby located, interact with each other via horizontal, weaker excitatory connections.

For each subpopulation we consider inhibitory neurons associated with this subpopulation. These inhibitory neurons specifically project to excitatory neurons in the associated subpopulation that in return project to these inhibitory neurons. Thus these inhibitory neurons give feedback inhibition to the associated subpopulation. We refer to this inhibition as 'local inhibition'. In the real brain, this type of inhibition is mediated by dendrite-targeting, calbindin-containing interneurons [7].

We further consider a pool of inhibitory neurons of another type. All the excitatory neurons in the model project to these inhibitory neurons that in return nonspecifically project to all the excitatory neurons. These inhibitory neurons generate mutual inhibition between populations, which will be referred to as 'global inhibition'. This type of inhibition is mediated in the real brain by perisoma-targeting, parvalbumin-containing interneurons (basket cells) [7]. We further assume that each neuron is subject to background noise.

2.3 Neuronal dynamics

We refer to the n -th excitatory neuron belonging to the m -th subpopulation in the p -th population as 'neuron (n, m, p) '. Let $S(p, m, n)$ be a binary variable describing the state of neuron (n, m, p) : If this neuron is active, $S(n, m, p) = 1$; if it is inactive, $S(n, m, p) = 0$. Local and global inhibitions are modelled by linear feedback formulae (see the third and fourth terms in the right-hand side of Eq. (2)).

The state transition of excitatory neurons is defined by the following asynchronous stochastic dynamic.

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- (i) Select one excitatory neuron (say, neuron (n, m, p)) randomly from the pool of all the excitatory neurons.

- (ii) Calculate the input to the selected neuron,

$$I(n, m, p) = (G_e / (N - 1)) (\sum_{n'} S(n', m, p) - S(n, m, p)) + (G_E / (M - 1)N) (\sum_{n', m'} S(n', m', p) - \sum_{n'} S(n', m, p)) - (G_i / N) (\sum_{n'} S(n', m, p)) - (G_l / NM) (\sum_{n', m', p'} S(n', m', p')). \quad (2)$$

Here, G_e is the strength of recurrent connection between excitatory neurons in each subpopulation; G_E is the strength of horizontal excitatory connection between subpopulations in each population; G_i and G_l represent the strengths of local and global inhibitions, respectively.

- (iii) Update $S(n, m, p)$ by the following probabilistic rule:

$$S(n, m, p) = \begin{cases} 1 & \text{with probability } p = 1 / (1 + e^{-\beta I(n, m, p)}) \\ 0 & \text{with probability } 1 - p \end{cases} \quad (3)$$

- (iv) Repeat (i)-(iii).

2.4 Parameter values

Parameter values used in the present study are listed as follows:

$$N = 5; M = 100; G_e = 2.0; G_E = 2.0; G_i = 2.0; G_l = 1.75; \beta = 45.$$

3. Results

Simulation analysis of the model revealed a characteristic time course of neuronal activation (Fig. 1). At first the activity of one population, which is defined by $\sum_{n, m} S(n, m, p)$, is gradually ramped up, while those of the other populations gradually decrease. Then at a certain time the former sharply increases to the maximum, while the latter ones fall to the minimum.

We regard the alternative corresponding to the maximally activated population as the chosen alternative and define the response time (RT) by the timing of the onset of this maximal activation. The simulated RT varies from trial to trial (data not shown); this is attributed to the probabilistic nature of the model. Hence we calculated the mean of simulated RT. We found that the simulated mean RT increases log-linearly with the number of alternatives (Fig. 2), thus following Hick's law.

In practical psychological experiment, subject's decision timing also varies from trial to trial. However, statistical properties of RT for multi-alternative decision are not yet fully established so far. To get insight into them, we examined the coefficient of variation (CV) of simulated RT (i.e. the ratio of the standard deviation to the mean) as a function of the number of alternatives. We found that CV of simulated RT is constant with the number of alternatives (Fig. 3).

4. Discussion

Hick's law is a hallmark of multi-alternative decision making. Nevertheless, neural basis of Hick's law is little established up to now. Here we have proposed a cortical-circuit model that reproduces Hick's law.

The model also gives testable prediction to psychophysical experiment: The coefficient of variation of the response time of multi-alternative decision is kept constant with the number of

alternatives. Interestingly, preliminary data from a recent psychophysical experiment is consistent with this prediction (see Fig. 6 of [8]).

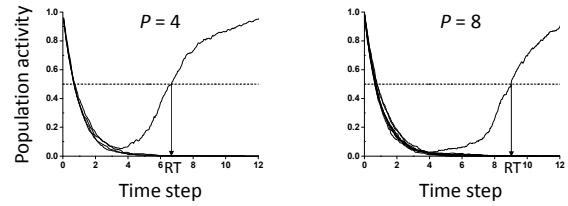


Figure 1: Time course of population activity for four (left) and eight (right) alternatives

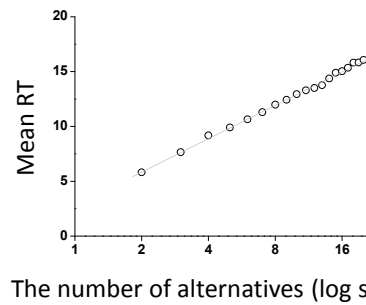


Figure 2: The simulated mean RT (500 trial average) increases log-linearly with the number of alternatives.

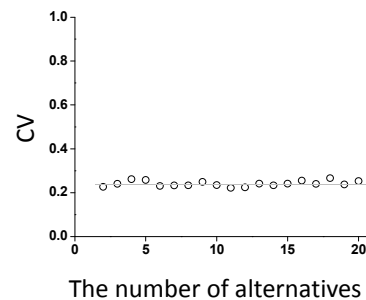


Figure 3: The CV of RT is constant with the number of alternatives.

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