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Selective pattern recall in neural networks by chemical modulation

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Abstract. We present a simple neural network model in which the outputs of groups of neurons are chemically modulated. This feature takes into account the existence of neuromodulators which are known to have a strong effect on the behaviour of real biological networks. The effective (non-symmetric) connection matrix thereby becomes a function of the modulator concentrations. By deriving evolution equations for the pattern overlaps we show that, during recall, the system can select distinct subclasses from the stored patterns by varying these concentrations. Our model may increase our understanding of the function of neuromodulators.

1. Introduction

Neural network models of the Hopfield type (Hopfield 1982, Amit *et al* 1985a, b) provide a nice framework in which to obtain analytical results. The definition of neural networks in terms of Ising spins and pair interactions enables one to make extensive use of tools from statistical physics. The storage and retrieval of patterns (under various circumstances) is the topic that is studied most. Whereas the first models were not completely realistic from a neurological point of view, nowadays it is becoming possible to incorporate Nature's constraints, e.g. Dale's law (Shinomoto 1987) and finite connectivity (Derrida *et al* 1987, Canning and Gardner 1988, Coolen *et al* 1989) and still analyse the model behaviour analytically.

The purpose of our paper is to define a simple model along these lines which incorporates the fact that in biological networks there are a large number of neuromodulators which strongly affect the system's evolution in time. The role of these substances ranges from causing a switch between modes of operation of small pattern generators (Mardner 1988) to controlling the 'motivational' or 'emotional' state of the nervous system in a wide variety of animals (Kravitz 1988). The modulators can be hormones, neuropeptides or drugs. There is a large body of experimental work on neuromodulation effects at the level of single neurons (Kaczmarek and Levitan 1987), but we know of no model in which one can explicitly calculate the effect of neuromodulators on collective processes in large, densely interconnected networks. Our model illustrates how a considerable increase in flexibility of the network behaviour arises from adding a simple cell property which occurs naturally and which can be implemented easily in silicon chips. Still, analysis of the network performance only requires a moderate extension of existing methods.

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2. Motivation and biological background

The theoretical and technological relevance of our model can be understood without regard for biological considerations, but it may be interesting to quickly sketch its biological motivations.

Since at least 1849 (Berthold 1849), it has been observed that actually displayed behavioural repertoires are strongly dependent on compounds such as hormones, neuropeptides and many drugs, which are released slowly and reach large parts (or the whole) of the brain. Such neuromodulators affect (Kaczmarek and Levitan 1987) the action of the (fast and local) neurotransmitters by interfering with the biochemistry of their production, transport, release, etc. They may also affect the effectiveness of specific receptors or change thresholds. In our model we only consider modulation of the transmission strength. Note that modulation specificity depends on transmitter/receptor types, used by a cell, not (on our scale of interest) on its position in the network.

At present one of the main areas of experimental neurobiology concerns questions of how modulation of cells controls the behavioural repertoire of neural networks. In recent experiments (Bicker *et al* 1989, Kravitz 1988, Mardner 1988) on very small networks, it has become possible to study how spontaneous or stimulus-evoked behaviours can be activated, suppressed, or switched from one mode to another. Our model can be seen as a first attempt to understand these effects.

3. Definitions

As usual, we represent the N neurons by Ising spins s_i $(i = 1 \ldots N)$. If neuron i fires we put $s_i = 1$; if it is at rest $s_i = -1$. The network is fully connected. The stochastic evolution of the microscopic state $\boldsymbol{s} = (s_1, \ldots, s_N)$ is governed by the master equation

$$\frac{\mathrm{d}}{\mathrm{d}t} p_t(\boldsymbol{s}) = \sum_i p_t(F_i\boldsymbol{s}) w_i(F_i\boldsymbol{s}) - p_t(\boldsymbol{s}) \sum_i w_i(\boldsymbol{s}). \tag{1}$$

Here $p_t(\boldsymbol{s})$ is the probability of finding the system at time t in state $\boldsymbol{s}$, $w_i(\boldsymbol{s})$ is the probability per unit time that spin i will flip, and F_i is an operator: $F_i\Phi(s_1, \ldots, s_N) \equiv \Phi(s_1, \ldots, -s_i, \ldots, s_N)$. For $w_i(\boldsymbol{s})$ we make the usual choice

$$w_i(\boldsymbol{s}) = \tfrac{1}{2}[1 - \tanh(\beta s_i h_i)] \tag{2}$$

where β (the inverse 'temperature', $\beta \equiv 1/T$) is a measure of the rate of spontaneous spin-flips and h_i is the local field, or 'input', at site i:

$$h_i = \sum_j J_{ij} M_j (s_j + 1) \tag{3}$$

The matrix elements J_{ij} represent the strengths of the synaptic connections between the neurons; if $J_{ij} > 0$, neuron j has an excitatory effect on neuron i, if $J_{ij} < 0$ the effect is inhibitory. All information is stored in the values of these connections. The $M_j \in \{0, 1\}$ reflect the effect of the neuromodulators which can prevent neurons from transmitting information. A specific modulation state is represented by the N-bit vector $\boldsymbol{M} = (M_1, \ldots, M_N)$, which we call a 'mask'. These masks have only a finite number of degrees of freedom since there is only a finite number of classes of neurons that can respond differently to neuromodulators. We assume that the network has learned a

number of patterns $\boldsymbol{\xi}^{(\mu)} = (\xi_1^{(\mu)}, \ldots, \xi_N^{(\mu)})$, $\mu = 1 \ldots p$, according to Hebb's rule (Hebb 1949). During the learning stage we must again take into account the presence of neuromodulators: we will denote by $\boldsymbol{M}^{(\mu)}$ the mask that was present during the learning of pattern μ. If we write $\boldsymbol{c} = (c_1, \ldots, c_n)$ for the concentrations of the n neuromodulators during recall we can write for the local fields:

$$h_i = \sum_j T_{ij}(\boldsymbol{c}) s_j \tag{4}$$

where

$$T_{ij}(\boldsymbol{c}) = \frac{1}{N} \sum_\mu \xi_i^{(\mu)} \xi_j^{(\mu)} M_j^{(\mu)} M_j(\boldsymbol{c}).$$

The (non-symmetric) effective connection matrix T_{ij} can be controlled reversibly by varying the concentrations $\boldsymbol{c}$ (the changes in $\boldsymbol{c}$ are of course much slower than the changes in the microscopic state $\boldsymbol{s}$.

4. The macroscopic behaviour

In order to analyse the behaviour of this system, we consider the usual macroscopic description in terms of the state overlaps $q_\mu(\boldsymbol{s})$:

$$q_\mu(\boldsymbol{s}) = \frac{1}{N} \sum_i \xi_i^{(\mu)} s_i. \tag{5}$$

Because of the non-symmetry of the effective connection matrix, we cannot compute the equilibrium values of the q_μ by applying equilibrium statistical mechanics; however, we can cast the problem in the form studied in Coolen and Ruijgrok (1988), where evolution equations were derived for the pattern overlaps which also hold for non-symmetric connectivity. We introduce dummy patterns $\boldsymbol{\eta}^{(\mu)} = (\eta_1^{(\mu)}, \ldots, \eta_N^{(\mu)})$ with corresponding overlaps $k_\mu(\boldsymbol{s})$:

$$\eta_i^{(\mu)} \equiv M_i^{(\mu)} M_i \xi_i^{(\mu)} \qquad k_\mu(\boldsymbol{s}) = \frac{1}{N} \sum_i \eta_i^{(\mu)} s_i \tag{6}$$

(note that in Coolen and Ruijgrok (1988) the 'patterns' need not be real spin configurations). In the limit $N \to \infty$ we find deterministic flow equations for the macroscopic variables $\boldsymbol{k}$ and $\boldsymbol{q}$. If the stored patterns are chosen at random these equations are:

$$\frac{\mathrm{d}}{\mathrm{d}t} q_\mu = \langle \xi_\mu \tanh[\beta \boldsymbol{\xi} \cdot \boldsymbol{k}] \rangle_{\boldsymbol{\xi}} - q_\mu \qquad \frac{\mathrm{d}}{\mathrm{d}t} k_\mu = \lambda_\mu(\boldsymbol{c}) \langle \xi_\mu \tanh[\beta \boldsymbol{\xi} \cdot \boldsymbol{k}] \rangle_{\boldsymbol{\xi}} - k_\mu.$$

Here $\langle\,\rangle_{\boldsymbol{\xi}}$ denotes the average over the distribution from which the patterns were chosen; $\lambda_\mu(\boldsymbol{c})$ is the overlap between the present mask $\boldsymbol{M}$ and the mask during the learning of pattern μ:

$$\lambda_\mu(\boldsymbol{c}) \equiv \lim_{N \to \infty} \frac{1}{N} \sum_i M_i^{(\mu)} M_i.$$

Finally we define $m_\mu \equiv k_\mu / \lambda_\mu$, in terms of which the evolution equations are:

$$\frac{\mathrm{d}}{\mathrm{d}t} q_\mu = \left\langle \xi_\mu \tanh\left(\beta \sum_\nu \lambda_\nu \xi_\nu m_\nu \right) \right\rangle_{\boldsymbol{\xi}} - q_\mu \tag{7}$$

$$\frac{\mathrm{d}}{\mathrm{d}t} m_\mu = \left\langle \xi_\mu \tanh\left(\beta \sum_\nu \lambda_\nu \xi_\nu m_\nu \right) \right\rangle_{\boldsymbol{\xi}} - m_\mu. \tag{8}$$

With (7), (8) the system behaviour can be understood. The evolution is fully determined by the m_μ (apart from a scaling factor, m_μ is simply the pattern overlap computed over the subset of all neurons for which there is a match between the actual mask $\boldsymbol{M}$ and the mask $\boldsymbol{M}^{(\mu)}$). The overlaps q_μ are simply being 'dragged along'. Note that (8) is the same equation one would find if one were to compute the evolution in time for the overlaps in Viana's (1988) model, where the connection matrix is:

$$J_{ij} = \frac{1}{N} \sum_\mu \lambda_\mu(\boldsymbol{c}) \boldsymbol{\xi}_i^{(\mu)} \boldsymbol{\xi}_j^{(\mu)}.$$

Our system will always reach equilibrium, in which case $\boldsymbol{q} = \boldsymbol{m}$. However, in Viana's model the weights λ_μ (which determine how strongly the patterns are stored) are fixed numbers; they are determined solely by the number of times the patterns were presented during the learning stage. In our model the weights $\lambda_\mu(\boldsymbol{c})$ are not fixed, but can be varied by changing the concentrations $\boldsymbol{c}$ of the neuromodulators.

The recall of one of the stored patterns corresponds to the macroscopic state $q_\mu = q(T)\delta_{\rho\mu}$. The critical temperature T_ρ below which such a state is an equilibrium solution of (8) is simply λ_ρ. Given a noise level T, the system can only recall patterns for which $\lambda_\rho > T$. Put differently, only those patterns can be recalled for which the mask used during the learning stage bears a sufficient resemblance to the present mask. In addition, the 'suppressed' state are unstable in a finite range below their critical temperature; this further extends the selectivity. A detailed analysis of the equilibrium properties of (7), (8) for a given chemical setting is implicitly given in Viana (1988).

5. Discussion

Our model illustrates how a simple modulation scheme, based on neuron types with different transmitters, leads to selective pattern recall at the network level. During pattern recall, by varying the concentrations of the neuromodulators, the system selects from the set of all stored patterns those patterns which were learned with a modulation state (mask) that resembles the present modulation state. We have concentrated here on selective recall from a repertoire of static patterns but the analysis can be extended to networks with stored sequences of patterns or to layered networks that perform transformations.

Again, as with, e.g., the models by Mezard *et al* (1986) and Nadal *et al* (1986), the collective behaviour in a specific neural system is found to be formally equivalent to the macroscopic evolution in Viana's model (1988) for pattern storage with unequal weights (if these weights are properly chosen).

References

Amit D J, Gutfreund H and Sompolinsky H 1985a *Phys. Rev.* A **32** 1007
—— 1985b *Phys. Rev. Lett.* **55** 1530
Berthold 1849 *Arch. Anat. Physiol. Wiss. Med.* **16** 42
Bicker G and Menzel R 1989 *Nature* **337** 33
Canning A and Gardner E 1985 *J. Phys. A: Math. Gen.* **21** 3275
Coolen A C C, Denier van der Gon J J and Ruijgrok Th W 1989 *Artificial Neural Networks* (London: IEE)
Coolen A C C and Ruijgrok Th W 1988 *Phys. Rev.* A **38** 4253
Derrida B, Gardner E and Zippelius A 1987 *Europhys. Lett.* **4** 167

Hebb D O 1949 *The Organization of Behaviour* (New York: Wiley)
Hopfield J J 1982 *Proc. Natl Acad. Sci., USA* **79** 2554
Kaczmarek L K and Levitan I B 1987 *Neuromodulation. The Biochemical Control of Neuronal Excitability* (Oxford: Oxford University Press)
Kravitz E A 1988 *Science* **241** 1775
Mardner E 1988 *Nature* **335** 296
Mezard M, Nadal J P and Toulouse G 1986 *J. Physique.* **47** 1457
Nadal J P, Toulouse G, Changeux J P and Dehaene S 1986 *Europhys. Lett.* **1** 535
Shinomoto S 1987 *Biol. Cybern.* **57** 197
Viana L 1988 *J. Physique* **49** 167