# Hypernetwork Model of Biological Information Processing

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**Abstract- A hierarchical architecture for information processing, the hypernetwork model, has recently been implemented. This is a three level model, inspired by biological systems, that includes representation of scale, vertical flow of information, and feedback control. All interactions are based on complementary relationships between molecular subunits. The system is molded to perform desired tasks through a variation-selection algorithm acting on the structure of the molecular subunits. The design of the system, the learning algorithm, and preliminary pattern classification results are presented.**

## 1 Introduction

Nature is organized hierarchically. Biological system hierarchies run through subatomic particles, atoms, molecules, cells, organs, individuals, communities, and ecosystems. Each level of organization has its own characteristics, but nevertheless the units at the different levels interact with each other to form a vast network whose space-time dynamics cross different physical scales, from the micro to the meso to the macro. All interactions are ultimately based on virtual particle exchanges between elementary particles. This atomistic interactional picture does not, however, preclude the existence of properties at higher hierarchical levels that emerge in collective fashion from the properties of the constituent elements (Scott, 1996). Scale and hierarchy go hand in hand.

The model described here, to be referred to as the hypernetwork model, addresses the role of this hierarchical organization in biological information processing. The term "hypernetwork" is intended to capture the interactional network aspect of multiscale information processing. The model treats biological systems as networks of interacting macromolecules. Shape complementarity is the operative interaction principle. Cells are networks of molecular interactions. The key feature of the hypernetwork model, consistent with the atomistic principle, is that units at all levels above the base molecular level are constructed from molecular interactions. The distinguishing feature is scale. Thus the organism is represented as a network of molecular interactions involving effector and receptor molecules of the cells. The interactions that define the organism level network have in general different scale properties. This represents the complementary collective aspect of biological organization.

Adaptive capabilities are clearly essential. The pattern of interactions must be molded by evolution or learning to perform requisite functions. Adaptation can be understood as a process of choosing a particular subnetwork to solve a given problem. Two basic time scales can be considered: the phylogenetic and the ontogenetic. Phylogenetic learning is essentially population based. Evolutionary computation techniques generally fit to this picture (Bremermann, 1962; Fogel, 1995; Fogel et al., 1966; Holland, 1975; Reynolds, 1994; Schwefel, 1995) The shorter time scale ontogenetic adaptation process resides in a single individual and may take many forms (including population based forms, as in the immune system).

Our specific purpose here is to describe the model and to illustrate its operation with preliminary experiments on simple signal classification tasks. At this stage we represent only a single organism. Learning is based on error feedback acting on the representation of molecular structure. Signals impinging on the network are integrated in space and time by complementary molecular interactions, viewed as representing either the dynamic formation of structure or as linking metabolic processes. Readout molecules interpret these dynamic patterns to activate output. The representation of molecular structure is varied first, then the location of readouts is varied. As the performance is improved, the extent of variation is decreased. Population-based evolution can be viewed as an extension that would allow memories of successful variants to be preserved. This is the intended next step.

## 2 The Percolation Network Model

The percolation network model forms the conceptual basis of the hypernetwork model. This is is a multi-scale model that stresses the flow of information among the different levels (Conrad, 1979, 1984, 1993, 1995a,b, 1997). Information percolates across scale if it is neither ignorable nor eliminable for the purposes of calculating the time development of the system as viewed at other scales. These influences go both bottom-up and top-down. Influences from the environment impinge on the top level of the system. The influences then filter down into lower levels (organs, cells) through "integrative dynamics", until they reach lower levels (meso and microscale). Specific influences percolate to upper levels by "selective amplification" until they again reach the environment at the top level (see Figure 1).

The cyclic AMP system of neurons provides an example of cross-scale interactions and of the transduction-amplification process. Neurotransmitter molecules (e.g.,

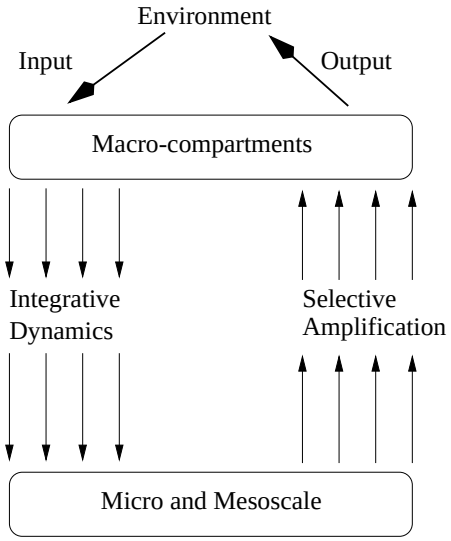



Figure 1: Schematic representation of a percolation network. Modified from Conrad (1995b).

epinephrine, vasopressin) bind excitatory receptors that activate G-proteins; these are first stage amplifying components that activate adenylate cyclase, the enzyme that produces the second messenger cAMP from ATP. The cAMP in turn activates target proteins (protein phosphatases) that activate effector proteins. These might be associated with DNA, with cytoskeleton, with synaptic vesicles, or may be ion channel proteins that control nerve impulse activity (Shepherd, 1994). The net result is a change in the internal network of the cell that can percolate up to higher levels of organization.

The cyclic nucleotide system is ubiquitous in the cells of higher organisms. Its role in controlling the firing behavior of central neurons suggests that the percolation network principle is operative in the brain (Liberman et al., 1985). The immune and developmental systems can also be viewed in this manner. The general feature is this: external influences (e.g., photons, messenger molecules) filter down to alter internal networks of molecular interactions within the cell. The influences are combined in space and time through these interactions, leading eventually to activation of cellular effector molecules. The interactions percolate up from the molecular to the cellular level through interactions between effector and receptor molecules of different cells. Integration and selection of information occurs at all stages. Multiple scales contribute synergistically to this process. The key underlying mechanism is molecular conformation. The structure and function in biological systems is most essentially controlled by shape interactions among macromolecules.

## 3 Model Description

The overall architecture comprises three hierarchical levels, to be referred to as molecular, cellular, and network levels, as well as an external environment from which the organism receives influences (Figure 2). The internal environment of the organism is defined by the relationships among its constituent components.

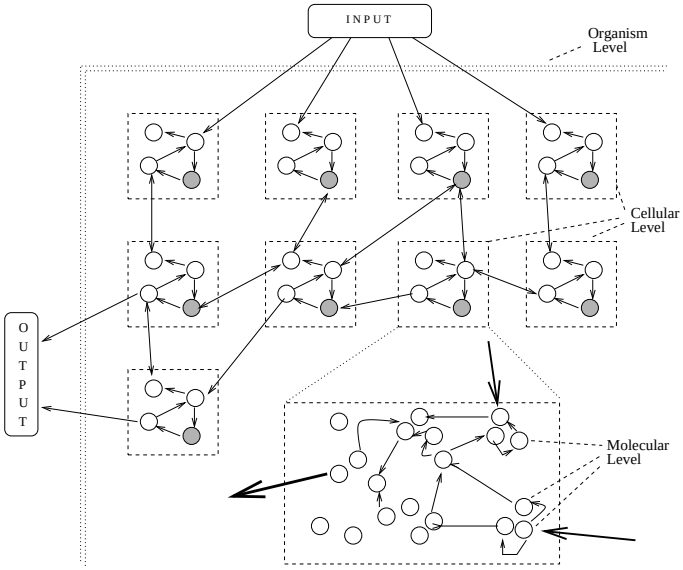
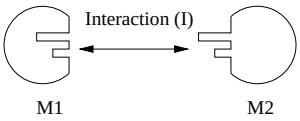
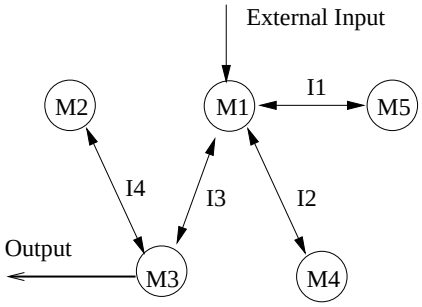



Figure 2: The hypernetwork model. Circles represent molecules and arrows interactions among molecules. Dashed rectangles around collections of molecules demarcate cellular units.

The design of the model is bottom-up, based on molecular interactions at the lowest hierarchical level. Two molecules react if their recognition sites are complementary to each other. The basic unit can be schematically represented like this:




Here M1 and M2 are molecules and I is the interaction between these two molecules. The network of interactions is constructed from many such binary interactions. For example in the next schematic molecule M1 interacts with molecules M3, M4 and M5, through the interactions I3, I2 and I1 respectively.



The network of interactions can be thought of as a highly abstracted representation of reaction cascades in the cell. The network can change according to the given input and its current state. At present scale is represented by density of interactions. This is greater within the cell than among cells. However, interaction strength and timing could also be used to represent scale features.

Each cell is represented as a grid (without wraparound). Each grid location contains one molecule. Molecular shapes are represented by binary strings. Interactions are defined by complementary matches between strings. At present these strings run from two to eight bits, but for the preliminary studies reported here we used two bit strings. Every molecule has two states: active (if it is ready to interact with its neighbors) and inactive. A molecule can be activated by external influences or by a neighboring activated molecule that interacts with it (by complementary matching). Partial matches are possible, but not used in the present study.

Molecules can serve receptor, effector, metabolic and readout functions. Receptor molecules receive signals from external inputs or through the interaction with effector molecules from other cells. Metabolic molecules form internal networks within the cell. These networks combine the input signals in space and time. The cells contain a subset of effector molecules. The activation of the molecules in this subset controls the interactions with other cells. Cells can also contain readout molecules that are activated by local patterns of activity, but at present these are restricted to an output layer of cells (as described below).

All activities of the cell are based on the behavior of its constituent molecules (see Figure 3). The intercellular interactions that define the cell level network are formed through complementarity interactions between effector and receptor molecules. This is the organism level, the top level of a single system.

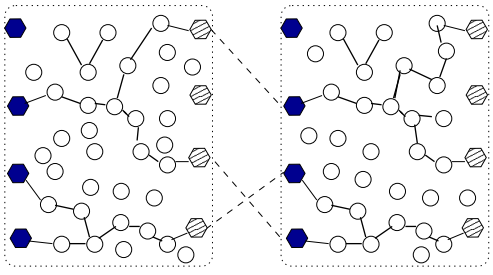


Figure 3: Two cells with their molecules. Receptors are black hexagons, effectors are dashed hexagons. Metabolic molecules are circles. Dotted lines are effector-receptor interactions.

The network level comprises input cells, intermediate cells, and output cells. Input and intermediate cells form multiple connections to other cells that can be roughly pictured as multifunctional synapses. Effector (or output) cells have readout molecules that can activate these cells to yield a 0 or 1 output. Output cells will fire if a readout molecule is activated. In the present version of the model activation occurs if the readout molecule sits in the same location as an activated molecule of the output cells. Also, for the experiments done so far, the number of readout molecules has been rather arbitrarily restricted to five per output cell.

Input cell receptors receive external influences in the form of input vectors. The activated receptors trigger cascades of interactions in the input cells that activate effector molecules that in turn interact with receptor molecules of metabolic cells. These interactions, as indicated above, form the cellular level network. This is a dynamically changing network, depending on the particular input vector (Figure 4).

In this way external influences are translated and amplified first into intracellular dynamic signal flows, and then into less dense intercellular signal flows. The distribution of input to the molecules in the metabolic cells can be thought of as downward filtration and spreading of signals. The amplification of these signal patterns to form output can be thought of as an integration and upward percolation process.

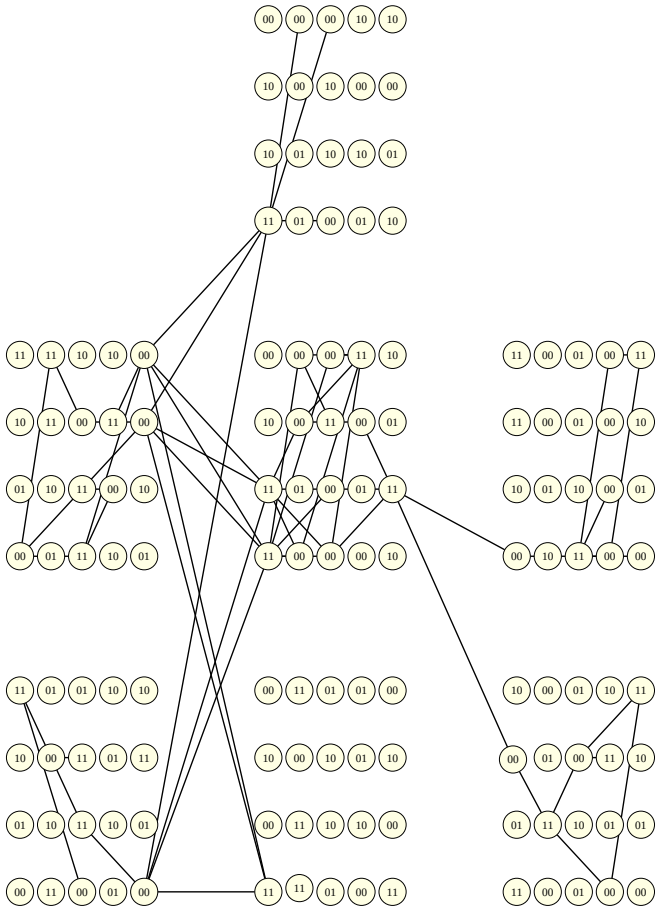


Figure 4: An example of an organism composed of, from left to right, two input cells, three intermediate cells, and two output cells. Each cell has 20 molecules (4 receptor, 12 metabolic, and 4 effector). Circles represent molecules. Lines represent interactions resulting from a given input.

## 4 Adaptation Algorithm

Adaptation is based on the feedback acting on the molecular structure. The difference between the output vector and the desired output associated with the input pattern determines the magnitude of an error signal. If the difference is significant, feedback lines are activated in a way that randomly modifies the bit string representation of the molecules. The hypernetwork architecture along with the adaptive feedback loop is illustrated in Figure 5.

The learning process begins with translation of the binary input data into a representation of intracellular molecular information. The binary input is segmented. Every segment influences one input cell. Each input cell contains all the possible receptors, thereby ensuring the activation of one receptor molecule in each input cell. This is not realistic, but is appropriate given the small number of cells in the present implementation. Every activated receptor molecule triggers the formation of a network of intracellular interactions, and therefore a wave of activity that passes through the cell until it reaches effector molecules. The effector molecule activity is in turn broadcast to receptor molecules of neighboring cells to form the cellular level network.

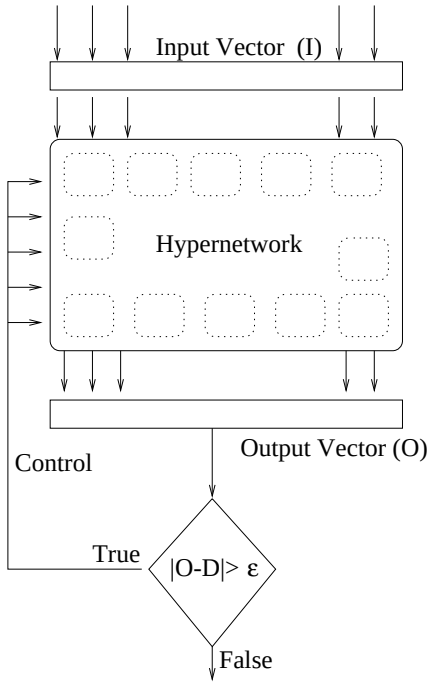



Figure 5: The hypernetwork and the self-adaptive loop.

The output vector is formed by the activity of the output cells. Output is binary. It is 1 if one of the readout molecules has been activated, 0 otherwise. The structure of molecules (i.e., their binary string representation) is changed randomly in response to the error signal. The extent of mutation is a sigmoidal function of the learning rate at the previous iteration. In the experiments reported molecules were chosen at random for mutation. Adaptation proceeds in two distinct phases (at present without repetition). The first is mutation of molecules other than readouts and receptors. The second is relocation of readouts. See Figure 6 for a pseudocode description of the learning algorithm.

**repeat**
  **for** Every input vector $I_i$, desired output vector $D_i$ **do**
    Read input vector $I_i$ into **input cells**
    **repeat**
      Propagate interactions through cells
      Form cell to cell interactions
    **until** Effectors of output cells are activated
    Read output vector $O_i$ from the **output cells**
  **end for**
  **if** $(\sum_1^n (O_i - D_i)/n \ > \ \epsilon)$ **then**
    Alter molecular structures (Phase I) or
    Relocation of readout molecules (Phase II)
  **end if**
**until** $(\sum_1^n (O_i - D_i)/n \ \leq \ \epsilon)$ or Termination condition

Figure 6: The adaptive algorithm for hypernetwork learning.

## 5 Preliminary Results

Previously, a multilevel neuron architecture was implemented to solve task navigation problems (Chen, 1993; Chen and Conrad, 1994), face recognition (Jeffries and Conrad, 1994), and categorization of Chinese characters (Chen and Conrad, 1997). The hypernetwork architecture has also been implemented to solve categorization problems, but at present of a much simpler nature. Data sets of 2, 4, and 6 bit length were used. The number of classes that must be recognized range from 2 to 64 (in the 6-bit case). See Tables 1-3 for preliminary results.

For a given architecture the difficulty of the task increases with the number of classes to be recognized, as might be expected given the hyper-exponential character of pattern grouping problems. The results show that the hypernetwork architecture exhibits adaptive properties despite the simplicity of the learning algorithm used. We expect that these capabilities will increase as the size of the system is enlarged and as more sophisticated versions of the learning algorithm are implemented.

Table 1: Two bit data set (4 vectors). I is the number of input cells, N the number of intermediate cells, and O the number of output cells.

| No. of Classes | No. of Cells I,N,O | Number of Molec/Cell | Iterations | Learning Rate (%) |
|---|---|---|---|---|
| 2 | 1,2,2 | 20 | 18 | 100 |
| 4 | 1,2,2 | 20 | 6000 | 100 |

Table 2: Four bit data set (16 vectors).

| No. of Classes | No. of Cells I,N,O | Number of Molec/Cell | Iterations | Learning Rate (%) |
|---|---|---|---|---|
| 2 | 2,10,4 | 20 | 6000 | 99 |
| 4 | 2,10,4 | 20 | 6000 | 88 |
| 16 | 2,10,4 | 20 | 6000 | 75 |

Table 3: Six bit data set (64 vectors).

| No. of Classes | No. of Cells I,N,O | Number of Molec/Cell | Iterations | Learning Rate (%) |
|---|---|---|---|---|
| 2 | 3,12,4 | 20 | 6000 | 74 |
| 8 | 3,12,3 | 20 | 6000 | 70 |
| 64 | 3,12,6 | 20 | 6000 | 65 |

## 6 Concluding Remark

The hypernetwork model has only recently been implemented. Work with it has just begun. The main objective is to elucidate the operative principles of hierarchical information processing in biological systems from a consistently interactional point of view. Consistent here means that all hierarchical features are formulated in terms of interactions of elementary macromolecular subunits. Many questions are open: what factors control how signals are distributed to the parts of a system; how do signals percolate from macro to micro scale; how are they integrated at different levels in space and time; how are they selected for amplification to the macro level; and how are they molded for function on ontogenetic and phylogenetic time scales? Our hope is that formal modeling from an interactional point of view will provide insight into these questions.

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