Development and Evolution of Neural Networks in an Artificial Chemistry

Jens C. Astor* and **Christoph Adami**

Computation and Neural Systems and Kellogg Radiation Laboratory
California Institute of Technology
Pasadena, CA, 91125 (USA)

Abstract

We present a model of decentralized growth for *Artificial Neural Networks* (ANNs) inspired by the development and the physiology of real nervous systems. In this model, each individual artificial neuron is an autonomous unit whose behavior is determined only by the *genetic information* it harbors and *local concentrations* of substrates modeled by a simple artificial chemistry. Gene expression is manifested as axon and dendrite growth, cell division and differentiation, substrate production and cell stimulation. We demonstrate the model's power with a hand-written genome that leads to the growth of a simple network which performs classical conditioning. To evolve more complex structures, we implemented a platform-independent, asynchronous, distributed *Genetic Algorithm* (GA) that allows users to participate in evolutionary experiments via the *World Wide Web*.

1 Introduction

Ever since computational neuroscience was born with the introduction of the abstract neuron by McCulloch and Pitts in 1943 [1], we have witnessed a gap between the mathematical modeling of neurons—inspired by Turing's notions of universal computation—and the physiology of biological neurons and the networks they form. The current state of affairs reflects this dichotomy: neurophysiological simulation test beds [2] cannot solve engineering problems, while sophisticated ANN models [3] do not explain the miracle of biological information processing.

Compared to real nervous systems, classical ANN models have a serious shortcoming owing to the fact that they are engineered to solve particular classification problems, and analyzed according to standard theory based mainly on statistics and global error reduction. As such, they can hardly be considered universal. Hence, such models *define* the network architecture *a priori* which is in most cases a fixed structure of homogeneous computation units.

Some models support problem-dependent network

changes during simulation [4, 3]. In these models, global decisions lead to network structures adapted to the problem at hand. Other approaches try to shape networks for a particular problem by evolving ANNs either directly [5], or indirectly via a growth process [6]. More recently, approaches like Ref. [7] include a kind of artificial chemistry which allows a more natural development. Still, in these models neurons are unevolvable homogeneous structures in a more or less fixed architecture which, we believe, limits their relevance to natural nervous systems.

In this paper we investigate the idea that interesting information-processing structures can be grown from a model which follows four basic principles of molecular and evolutionary biology, listed below. While models for ANNs currently exist that implement a selection of them, the inclusion of all four opens the possibility that, given enough evolutionary time, novel structures can emerge that are comparable to natural nervous systems.

- CODING. The model should encode ANNs in such way that evolutionary principles can be applied.

- DEVELOPMENT. The model should be capable of growing an ANN by a completely decentralized developmental process, based *exclusively* on the cell and its interactions.

- LOCALITY. Each neuron must act autonomously and be determined only by its genetic code and the state of its *local* environment.

- HETEROGENEITY. The model must have the capability to describe different, *heterogeneous* neurons in the same ANN.

One of the key features of a model implementing those principles will be the absence of explicit activity functions, learning rules, or connection structures. Rather, such characteristics should emerge in the adaptive process and lead to ANNs with open architectures and more universal artificial neurogenesis.

While keeping in mind that the model is not designed to reproduce real neural systems, we posit that an ad-

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*On leave from Dept. of Medical Informatics, University of Heidelberg/School of Technology, Heilbronn, Germany, jastor@stud.fh-heilbronn.de

herence to the fundamental tenets of molecular and evolutionary principles—albeit in an artificial medium—represents the most promising unexplored avenue in the search for intelligent information-processing structures.

2 Model

In this section we introduce our model of neurogenesis starting with the artificial physics and biochemistry, and go on to explain how local gene expression ultimately results in information-processing structures. This gene expression takes place exclusively in artificial neurons which are embedded in a tissue-like structure. As this model is inspired by the concepts of molecular cell biology, we use the nomenclature of this science unabashedly while issuing the caveat that they are analogical in nature only.

2.1 Artificial Physics

The physical world is a two-dimensional grid of hexagons. Each such site harbors certain concentrations of substrates, measured as percentage values of saturation between 0 and 1. As cells are equidistant in a hexagonal lattice, the diffusion of substrate k in cell i can be modeled discretely as

$$C_{ik}(t+1) = \frac{D}{6} \sum_{j=1}^{6} \left(C_{ik}(t) - C_{N_{i,j}k}(t) \right) \qquad (1)$$

where $C_{ik}(t)$ is the concentration of substrate k in site i, D is a diffusion coefficient ($D < 0.5$ to avoid substrate oscillation), and $N_{i,j}$ represents the jth neighbor of grid element i.
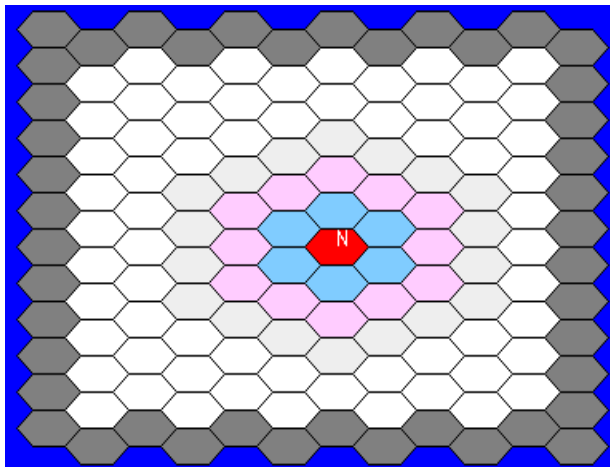

Figure 1: Hexagonal grid with boundary elements. Diffusion occurs from a local concentration peak at grid element N.

Accordingly, a local concentration of substrate will diffuse through the tissue under conservation of mass. The tissue itself is surrounded by special boundary elements which absorb substrates (Figure 1), thus modeling diffusion in infinite space. We caution at this point that the hexagons are sites that may *harbor* cells, but otherwise only represent a convenient equidistant discretization of space to facilitate the distribution of chemicals via diffusion.

2.2 Artificial Biochemistry

We distinguish four different classes of substrates:

- EXTERNAL PROTEINS: Diffusive substrates which can be produced by neurons if expressed.

- INTERNAL PROTEINS : Produced by neurons, but non-diffusive as they cannot cross cell membranes.

- CELL-TYPE PROTEINS: Each neural cell harbors an external protein that defines its type. Like any external protein it is diffusive.

- NEUROTRANSMITTER: Special type of internal protein used for directed information exchange between neurons.

2.3 Artificial Cell

Cell types We distinguish three kinds of neurons on the cellular level: actuator cells, sensor cells, and common neurons. The first two types are special versions of the third and are used as interface to a (simulated) environment to which the network adapts and on which it computes. These neurons can be excited to a real-valued level between 0 and 1, and take part in the information transfer via dendritic or axonal connections, respectively (see below). Each type of cell is also characterized by its own cell-type protein, which it produces continuously at a certain rate. These cell-type proteins diffuse over the tissue (Figure 1) and therefore signal cell existence to other cells. They can be compared to *growth factors* known from the development of real nervous systems.

Actuator and sensor cells do not carry genetic information; they are used solely as interfaces to the environment (input-output units, see Section 2.4.) They represent *sources* and *sinks* of signal. Consequently, their behavior is hardwired and does not depend on transcription as for common neurons. The latter can receive a flux of neurotransmitter from dendrites with a particular weight. However, this does not imply an automatic stimulation of activity unless such behavior is explicitly encoded in the neuron's genome. Table 1 summarizes the cell types and how they interact with other computational elements used in our model.

Genetic code and gene expression in artificial neurons Each neural cell carries a genome which completely encodes its behavior. Genomes consist of genes which can be viewed as a genetic program that can either be executed (*expressed*) or not, depending on a gene *condition* (akin to the regulator/operator genes in the

Type	[1]	[2]	[3]	[4]	[5]	[6]
Neuron	x	x	x	x	x	x
Sensor	x	x		x		x
Actuator	x	x			x	x
Grid element	x					
Boundary element						

Table 1: Features of different cell types building the artificial organic tissue: [1] participates in diffusion, [2] can be stimulated, [3] depends on gene expression, [4] can have axons, [5] can have dendrites, [6] produces a diffusible cell-type protein.

Condition	Description
ADD [EP]	$\Phi_{new} = \Phi_{before} + $ [EP]
MUL [eNT]	$\Phi_{new} = \Phi_{before} \times $ [eNT]
SUP [CTP0]	suppresses gene if cell not of type CTP0
AND [IP]	fuzzy AND: $\Phi_{new} = \max($[IP]$, \Phi_{before})$

Table 2: Condition atoms and their interpretation. Condition atoms build a gene condition, which is obtained by evaluating its condition atoms in the given order. Here, [XY] means 'the current local concentration of substrate XY inside of the cell to which the gene condition belongs'. Φ is the evaluation result of this gene condition.

Jacob-Monod-model). A gene condition is a combination of several condition *atoms*, usually related to local concentrations of substrates. The expression of a gene can result in different behaviors such as the production of a protein, cell division, axon/dendrite growth, cell stimulation, etc. Figure 2 clarifies the structure of the genetic code. Thus, gene conditions model the influence of external concentrations on the expression level of the gene, i.e., they model activation and suppression sites. To evaluate a gene condition, each element of its chain of
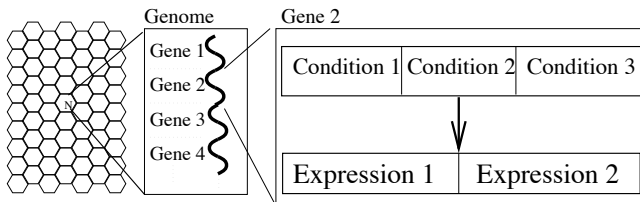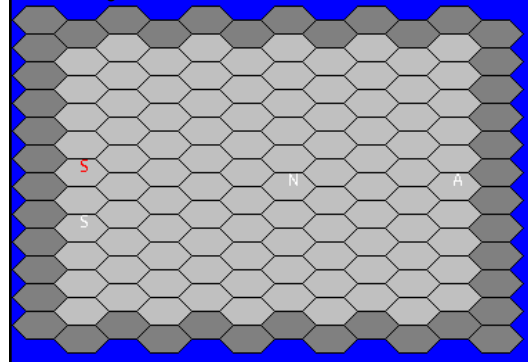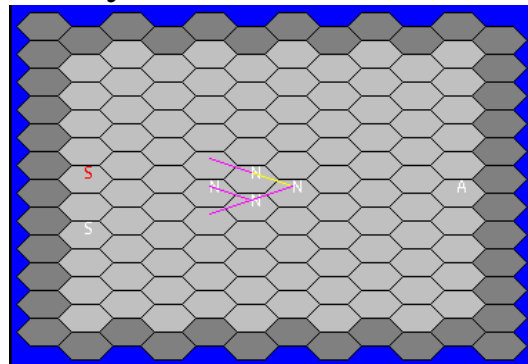

Figure 2: Genetic structure of neural cells. Local concentrations of substrates (condition atoms) trigger gene expression.

condition atoms contributes to obtain a real-valued expression level between 0 and 1, describing the strength with which the respective gene expression will take place.
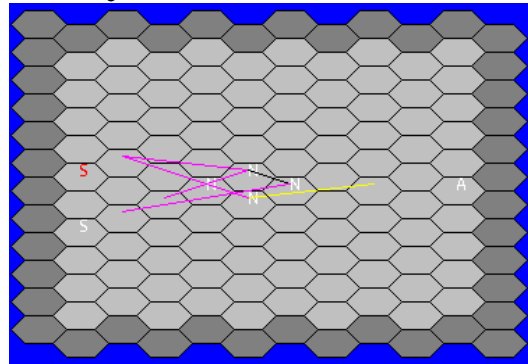
t=0 cycles:

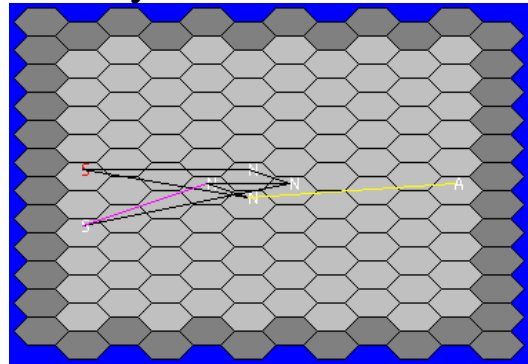t=6 cycles:

t=8 cycles:

t=10 cycles:

Figure 3: Development of the network for classical conditioning.

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Expression	Command Description	Influence of Condition Value
PRD[XY]	produce substrate XY	production quantity
GDR[XY]	grow dendrite following gradient of XY	growing probability
GRA[XY]	grow axon following gradient of XY	growing probability
SPL[CTPx]	divide to CTPx-type cell	probability
EXT	excitatory stimulus	increase rate
INH	inhibitory stimulus	decrease rate
MOD+	increase connection weights	strengthening factor
MOD-	decrease connection weights	weakening factor

Table 3: Overview of expression commands. Growing axons/dendrites follow the substrate gradient until the local maximum is reached, then connect to the cell (if it exists). Strengthening/weakening is a percentage increase/decrease of connection weights, determined by the product of the last neurotransmitter influx at each connection and the value of the gene condition. The cell-type protein assigned to a cell division command determines the future type of the offspring cell. In this example, the daughter will be of type CTPx and therefore produce cell-type protein CTPx continuously.

Consider for example substrates with local concentrations [ep0, ip, ep1]=(0.3, 0.5, 0.5). Then, the evaluation of gene condition ADD[ep0] ADD[ip] MUL[ep1] would lead to an overall expression level 0.4 (this value is modded back into range between 0 and 1 if it falls outside it). Table 2 illustrates a few examples of such conditions, while Table 3 gives an overview about the different gene *expression* commands. The evaluation result of the gene condition has different meanings depending on the gene expression command.

2.4 Simulation of the Artificial Organism

The tissue of cells produced by gene expression and cell growth is termed the *artificial organism*. It receives input from the environment (the outside world) and can act on it by signaling to the environment via its actuators. In the simplest case, thus, the organism receives and generates patterns.

A simulation always starts by creating sensor and actuator cells. Their number is determined only by the complexity of the outside world and is not coded for in the genome. In other words, these cells really represent *possible* signals and actuations in the world, not actual signals and actuations performed by the organism. An organism chooses to receive input or perform an actuation by connecting to these cells. If needed, an additional reinforcement cell can be created. This is a special sensor cell (with its own cell-type protein) used to provide a reinforcement signal from the world about the behavior of the organism. Whether or not this signal is used is determined by the organism's genome. Furthermore, at the start of each simulation, one initial neuron is placed in the center of the grid. After initialization, the simulation can begin. Input from the world is provided to the sensor cells, diffusion of produced cell-type proteins and external proteins takes place, and neurons execute their genetic code synchronously.

Depending on its gene expression, a neuron starts growing axons and dendrites, produces offspring cells and might initiate cell differentiation. Gene expressions may lead to protein production cascades, stimulation, and ultimately information exchange between neurons. After every simulation cycle the network's 'fitness' is determined by comparing any inputs and outputs to what is expected in this particular world, producing a real-valued reinforcement signal between 0 (punishment) and 1 (reward). This signal can be used by the organism if a reinforcement sensor is present and if the organism chooses to connect to it.

3 An Example Genome

Figure 3 documents the development of a simple ANN from a hand-written genome. Starting from a single initial neuron, cell division takes place and connections (axons and dendrites) start to grow along the gradient of diffusing cell-type proteins. After a while, sensors, neurons and actuator cells are connected in a particular manner. In fact, this network displays conditioned reflex behavior as in Pavlov's classical experiment [10]. Suppose the sensor on the lower left side in Figure 3 is stimulated (active) at the sound of a bell. Further, suppose the upper left sensor is an *optical stimulus* representing the presence (or absence) of food. Finally, let us imagine that the actuator on the right side triggers a salivary gland if food is present. This behavior is the hardwired unconditioned reflex. The above network can learn to associate the reflex with a condition: the sound of the bell. If presence of food and the ringing of the bell are associated repeatedly, the network will learn to trigger the gland even if *only* the bell rings. If the bell rings after the conditioning without the presence of food, the association will gradually, but steadily, weaken. Such a

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1. NNY(ip) SUP(cpt) ANY(spt0) -> SPL(acpt0) PRD(ip) SPL(acpt2) GDR(spt0) DFN(NT1)
2. NNY(ip) SUP(acpt0) ANY(spt1) ANY(cpt) -> PRD(ip) GDR(spt1) GRA(cpt) DFN(NT1)
3. ANY(spt1) SUP(acpt2) NNY(ip) ANY(apt0) -> SPL(acpt1) GDR(spt1) PRD(ip) GRA(apt0)
4. ANY(acpt2) SUP(acpt1) ANY(spt0) NNY(ip) -> GRA(acpt2) GDR(spt0) GDR(cpt) PRD(ip)
5. ANY(ip) -> PRD(ip)
6. NSUP(cpt) NSUP(acpt1) ADD(eNT) -> EXT
7. SUP(acpt1) ADD(NT1) MUL(eNT) -> EXT
8. ADD(eNT) -> PRD(ip1)
9. ADD(ip1) -> PRD(ip2)
10. SUP(cpt) ADD(NT1) MUL(ip2) -> PRD(ep)
11. SUP(cpt) ADD(ep) -> EXT
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Figure 4: Genome for development and behavior of network exhibiting classical conditioning

behavior can be modelled using different kinds of cell-types. The "C" cell is activated if the network is in the conditioned state, which means that acoustical and optical stimuli have been present together before. Cell "E" is activated if the acoustical stimulus is currently present and the network is in the conditioned state at the same time. If so, cell "G" representing the trigger of the salivary gland is activated. Of course, cell "G" is also activated if only food is present. This is the "hardwired" reflex. A schematical drawing of the network is shown in Figure 5. The genome which encodes the development and behavior of this network is shown in Fig. 4.
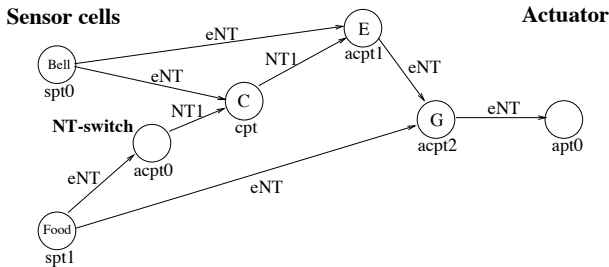

Figure 5: A schematical representation of the network for classical conditioning. The types of neurotransmitter used are shown next to the axons. The cell-type protein used by each cell is indicated near the cell body.

It is beyond the scope of this paper to go into the details of this genome and its function (see [8, 9] for a more thorough description). However, the explanation that follows still gives an idea of the type of information necessary to grow networks with particular characteristics.

The genome consists of 11 genes, each of which has its condition (left-hand side) and its expression (right-hand side). Genes 1 to 4 control cell division into the different types that are needed, as well as the growth of axons and dendrites. The first gene is only expressed by the initial cell, and only if no internal protein ip is present (the sequence NNY(ip) SUP(cpt)). In addition, gene 1 is

only expressed if it senses nonzero concentrations of cell-type protein spt0 (ANY(spt1) ANY(spt0)) which is emitted by one of the sensor cells and has diffused. Under these circumstances the initial cell will divide and produce off-spring of type acpt0 and acpt2 (SPL(acpt2) SPL(acpt0)), grow a dendrite that follows the gradient of the sensor protein spt0 (GDR(spt0)), and produce the internal protein ip. Once ip is produced, this will continue to happen (gene 5) which prevents that gene 1 can ever be turned on again. Genes 2 to 4 work just as gene 1, but for other cell types. While gene 5 takes care of the hardwired-reflex stimulation, genes 10 and 11 control the conditioning (expressed only by cell-type cpt). If food is present and the bell rings, gene 10 is expressed. It produces certain amounts of external protein ep. The concentration of ep influences cell stimulation (gene 11 exhibits stimulation via EXT). Due to diffusion, ep diminishes over time, so the conditioning decreases accordingly. As pathways to the "C" cell are not equally long, a production cascade of internal proteins in genes 8, 9 and 10 is necessary that delays the input of the acoustical sensor. The behavior of the resulting phenotype network is documented in Figures 6, 7 and 8.

4 WWW-based Genetic Algorithm: Community of Artificial Organisms

While it is not difficult to write genomes which lead to simple networks with desired characteristics, one of the main features of the system is its evolvability. Certainly, the search space for such genomes is immense, and it is unreasonable to hope that interesting genomes can be found without massive parallelism. Rather than choosing to implement this system on supercomputers, we opted to allow users on the Internet to donate their CPU time by participating in a global evolutionary experiment.

Using Sun's Java™ technology, we developed an asynchronous, distributed GA system which allows a massive parallel search for new genomes based on evolutionary principles [8, 9]. It consists mainly of a central server
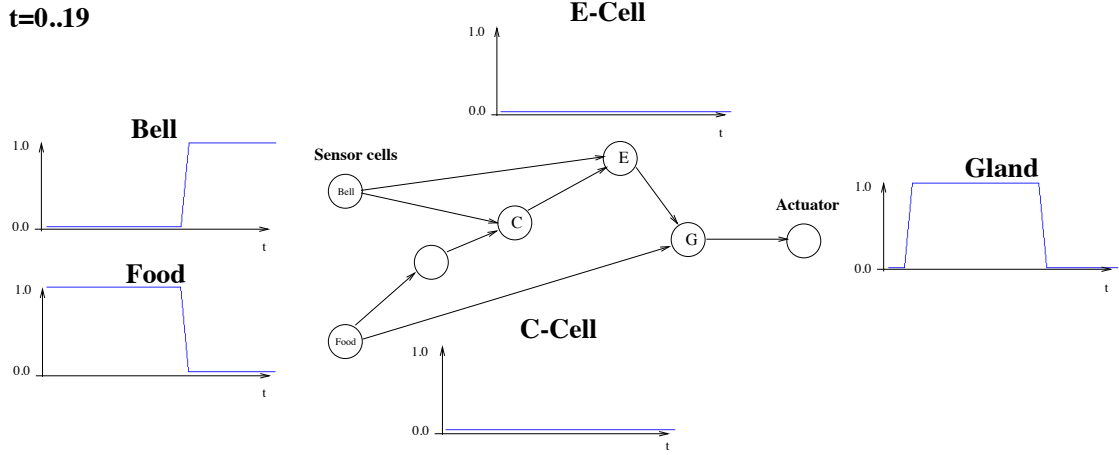
Figure 6: First, only food is present. This triggers the gland because of the hardwired reflex, while the "C"-cell and "E"-cell remain inactivated. Later, only the bell signal is present. Due to the fact that the network is not yet conditioned, none of the cells becomes active as a result.
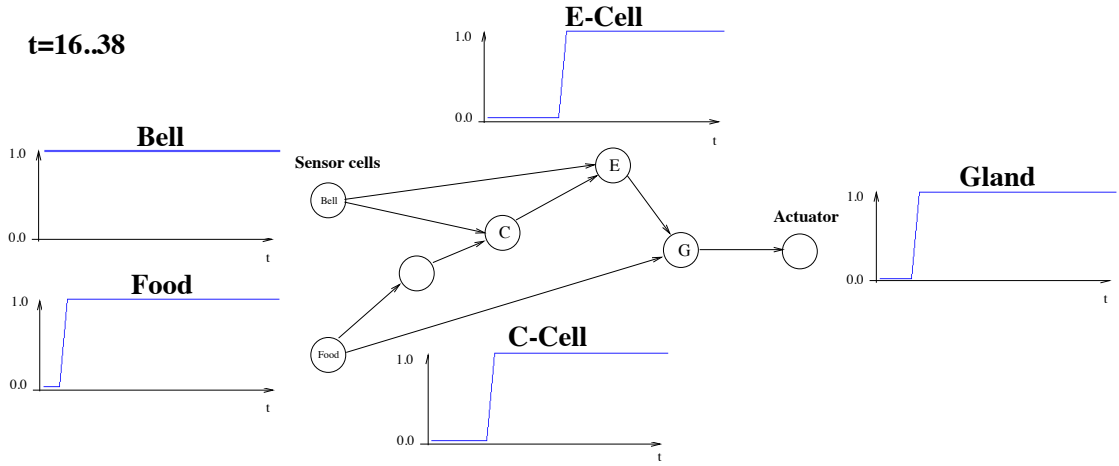

Figure 7: Both sensors, food and sound, are stimulated. The ANN becomes conditioned ("C"-cell) and the gland is triggered due to the presence of food.
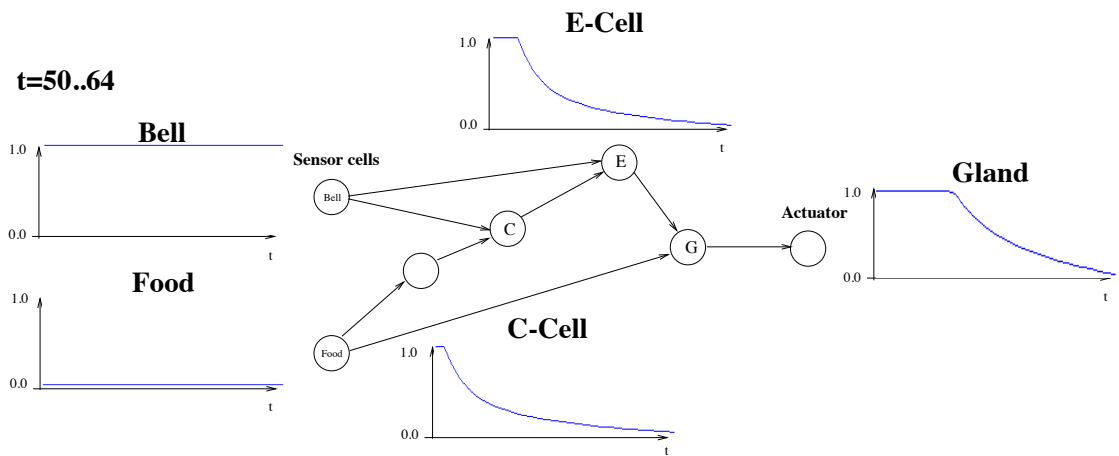

Figure 8: Being in the conditioned state, the food sensor suddenly becomes deactivated while the bell keeps on ringing. Thus, the activation of the "C"-cell becomes weaker. This implies a decrease of activation of the "E"-cell which finally results in a decline of gland activity.
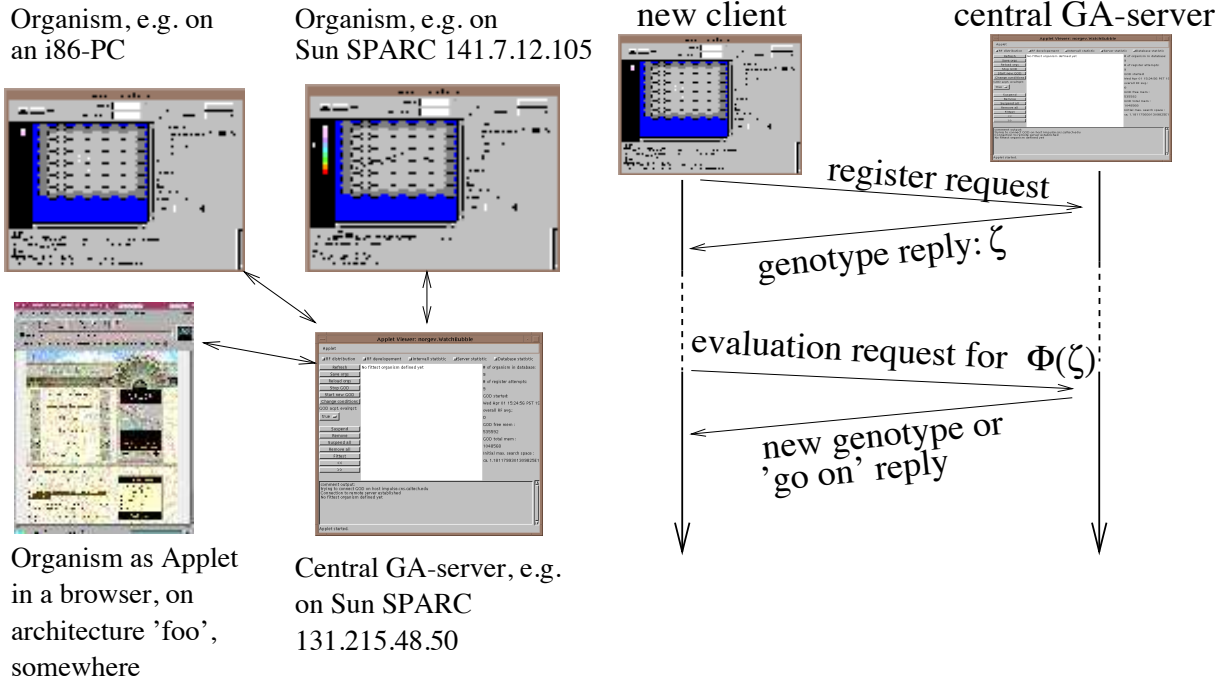
Figure 9: Left: genotype evaluation in clients of different architecture, using TCP/IP for communication with the asynchronous genetic algorithm. Right: communication between the GA-server and a client hosting an organism.

application and clients, each of which hosting *one* individual of the current GA population.

As Java is supposed to be platform independent, clients can be started from every computer for which an accurate Java 1.1 virtual machine or browser exists (Figure 9). The clients are hybrids, which means that they can be started as Java Applet by choosing the html page from our WWW-server, or with the help of a boot-loader program which dynamically downloads the client and starts it as a Java application (no browser necessary).

A client automatically sends a request-to-register to the central GA-server after it was started, and receives from the server a genotype ζ. The client then starts up a simulation as described in Section 2.4. After a certain number of simulation cycles, it sends the genotype's fitness $\Phi(\zeta)$ (average reinforcement signal during simulation) to the server. By comparing $\Phi(\zeta)$ to the fitness of other genotypes in the database, the server decides if it is worth to keep this genotype or if the client should be assigned a new one. If the server has to send a new genotype, it either takes a suspended one out of its database, or *constructs* one through the processes of recombination and/or mutation from genotypes of known fitness already present in the population (Figure 9). Fitter genotypes are more likely to be selected for recombination and/or asexual copying then genotypes of lower fitness. This leads to an increase of the average fitness over time.

5   Conclusion

We introduced a developmental and behavioral model based on artificial gene expression which shares key properties with natural neural development. Within this model we succeeded to construct simple systems with properties which are believed to be essential [11, 12] for higher self-organizing information processing systems, such as deterministic structure development, self-limiting growth, growth following diffusion gradients, computation of logical functions, pacemaker behavior and simple adaptation (sensitization, habituation, associative classical conditioning) [8, 9]. Furthermore, we showed how an evolutionary search for genomes coding for information-processing network structures can be distributed in a platform-independent manner such that the unused CPU power of the Internet can be tapped to search for ANNs that reduce the gap between the abstract models and neurophysiology.

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