Towards a comprehensive Alife-model of the evolution of the nervous system and adaptive behavior

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Abstract

The potentials and tools that are offered by Alife for biology in modeling the nervous system and animal behavior are mainly unexploited. There is no consistent Alife model of the biological evolution of the nervous system as yet, whereas the modeling tools are at hand and their application for this purpose seems evident. In a biologically grounded model we have to make every possible effort to use principles known from biology, and to minimize the arbitrarily employed organizing rules. The aim of our work is to create a biologically accurate Alife model of the formation and evolution of the nervous system in connection with the adaptive behavior. In this article we concentrate on the structure of the modeled genome, which is the basis of playing a double biological role: to ensure an open-ended evolutionary process, as well as to direct the ontogenesis. The main questions we examined are: what are the basic rules of construction that are sufficient to create a workable nervous system and how can we model them in a biologically realistic way?

Introduction

Adaptive behavior and its neurophysiological background is a complex enough phenomenon to be a serious challenge for the modelers, and the tools of Alife – neural nets, genetic algorithms (GAs), animats and the combinations of them – are excellent for this purpose.

We have to distinguish two ways of modeling adaptive behavior, as they apply these tools in different manners (Wilson 1991; Meyer and Guillot 1994). The aim of models motivated by biology is to study the neural control mechanisms of adaptive behavior of animals (Collett and Land 1975; Beer 1990; Cliff 1992), while models of evolutionary robotics mainly have an “engineering motivation”, according to which the most important goal is not biological authenticity but creating an autonomous robot which can perform a special task without human direction (Dorigo and Schnepf 1993; Floreano and Mondada; 1994). The latter ones can apply the biological principles freely and arbitrarily in the interest of success. These models are not those of biological evolution, but they are meant to search for optimal solutions of a special problem with the help of GA. The biological value of these models is doubtful, but it is a problem only if they are supposed to lead to biological conclusions beyond their competence.

The motivations of the two research groups are obvious. On one hand a practical way to understand the organization and development of intelligent systems is to study and to model their existent natural forms (animal behavior and its control mechanisms), because the successful modeling helps us understand the organizing and working principles. On the other hand, if we know the principles of forming intelligence during the biological evolution, we have some chance to create similar systems in an artificial way. The elaboration of successful models has a crucial practical importance, because difficulties of planning grow along with the advance of the complexity of the system. Up to now “real” Artificial Intelligence has successfully resisted the efforts of human designing...

The synthetic approach of Alife has been largely influenced by the results of analytic science, mainly neurobiology and genetics. At the same time we find that the potentials and tools that are offered by Alife for biology in modeling the nervous system are mainly unexploited. Perhaps we can state without exaggeration that neurobiology have not realized the potential that Alife-tools can offer them (Risan 1997). Computational neuroethology is a good example indicating the direction of progress, where there are advanced results by now (Beer 1990; Cliff 1994). But up to the present there is no Alife model of the biological evolution of the nervous system, whereas the modeling tools are at hand and their application for this purpose seems evident. Notwithstanding, there are no signs of applying them to create a consistent model of this evolutionary process. Perhaps this statement is surprising at first hearing, because there really are very interesting and successful models of adaptive behavior and its evolution (Ray 1992, 1994; Harvey, Husband and Cliff 1993; Sims 1994a, 1994b; Nolfi and Parisi 1994a, 1994b, 1995), which illuminate and illustrate important aspects of the biological evolution; and without intending to diminish the importance of the achievement of them we are only saying that they do not lead us any closer to understanding how the nervous system of animals had been created and developed during the evolution – as these models had not been created to study this question. However, we do not
have much evidence in connection with the formation and early evolution of the nervous system. In these cases modeling could help us to supply missing links and to test the theories. If we want to create a biologically relevant model of this subject, we have to keep some considerations in view, which normally do not arise in modeling adaptive behavior.

The aim of our work is to create an ALife model of the formation and evolution of the nervous system in connection with adaptive behavior. The following questions stand at the central point of our interest: how came the nervous system and the most primitive forms of intelligence into being? What are the basic rules of construction that are sufficient to create a workable nervous system without specifying the details of the construction? What kind of advantages can be ensured by a primitive protoneural system that is just forming?

### The importance of anatomy

An animal – even if we have to simplify it when it is modeled – cannot be viewed as a robot where the body can be handled in separation from the neural network. In the case of an animal body the outputs of the motoneurons cannot be valued by themselves, but only as elements of responses of a more complex system. The role of a certain output unit is not a result of an arbitrary definition, but depends on the anatomy and the position of the muscle that is innervated by that neuron. Moreover, we have to take into account that in a real animal even the simplest movements require the co-operation of many muscle cells, so their innervation has to be carried out in harmony as well. So the behavioral response to an environmental stimulus is influenced not only by the inputs and the outputs of the nervous system, but the anatomical construction of the body as well (Bullock and Horridge 1965; Lawn 1982; Spencer and Arkett, 1984).

If we examine the development of the adaptive behavior, it is not sufficient to model merely the formation and evolution of the neural control mechanisms. We have to consider the whole organism as an evolutionary unit, because keeping contact with the environment and performing the responses are the tasks of the whole living being (Albert 1999a, 1999b). It follows from the foregoing that the bodily construction of a certain animal cannot be a negligible feature in the modeling of adaptive behavior (Mackie 1990). Modeling the evolution of adaptive behavior cannot be reduced to modeling the evolution of the neural network.

A crucial point is to choose the suitable type of animal that can be the basis of the construction of the animal. In our opinion a model of the biological evolution of adaptive behavior and its nervous control cannot be based upon modeling evolved animals with advanced bodily constructions. These animals are too specialized to serve as a starting point for an evolutionary model. Advanced anatomy needs a highly specialized moving control system both in insects and vertebrates. In this case the modeling of a GA to test the optimal solutions of the nervous control of this specialized “machinery” (Ijspeert 1999). This task is analogous with evolutionary robotics and not with biological evolution. If we want to model the latter, then we cannot leave out of consideration that the bodily construction, the locomotor organs and their nervous control evolved together, so we cannot model one of them regardless of the others (Sims 1994a, 1994b).

### The problem of encoding

In a biologically established model we have to make every effort to avoid or at least to minimize the arbitrarily employed organizing rules. We have to make an attempt to use principles known from biology and not high-handed theories. Therefore in our model the connection of the genotype and phenotype has to differ from that simple relation that is common in models working with neural nets connected to GAs, and we have to bring this relation close to reality. In the case of “traditional” GAs the genome is only a plan containing the solution of a certain problem encoded in it. It is merely the subject of the working of GA, which is able to modify it via mutation and recombination. Then the program reads the data from the genome and creates the phenotype, the solution of a certain problem (Holland 1975). In this case genome is only a passive lump of data that is modifiable and readable, but has no active role creating the phenotype. Consequently GAs use only one aspect of the biological role of the genome, namely the function of a modifiable plan. In this model the flow of information is a one-way process during the life of an individual. Feedback from the phenotype to the genotype is only possible through selection (Balakrishnan and Honavar, 1995) and not during the life of an individual. It is not adequate for biological evolutionary processes, where genome has an active role in forming the phenotype and controlling the development.

Creating the genome, raises the crucial question what properties of the model-organism should be encoded and how to do it, because it determines the working of the model. The higher level properties are encoded in the genes, the easier it is to survey the connection between genotype and phenotype, and the more considerable the effect of the mutations is (Nolfi and Parisi 1995; Albert 1999a, 1999b). At the same time, however, there is no more considerable chance for new properties to arise, only the preprogrammed ones can become better adapted via quantitative changes. If there is no chance for qualitative changes, there will be no chance for new properties to emerge that are not preprogrammed – so the evolutionary process cannot cause a “surprise”. This lack of qualitative evolution is a serious problem, because we lose one of the most important aspects of the evolutionary process. We have to make some effort to preserve the possibility of an open-ended evolution, otherwise the model will only mirror the willowy built-in limitations of the programmer. Modelers in evolutionary robotics are also faced with this problem (Harvey 1992, 1993).
common feature in these attempts is the search for lower level properties encoded in the genome. We have to find the sub-properties or "primitives" that are building elements of higher level properties and take part in building more than one special property (Koza 1992). In real biological systems these encoded units are the amino acids, which build proteins, the real carriers of the properties.

Another problem that can limit the possibilities of a modeled evolution is that the lengths of the genes are predefined in most of the models. It is because of the encoding mechanism, since modeled genes are sequences in which all characters define a certain property of the phenotype. So if somewhere in the genome an additional character gets wedged in or falls out of the genome, all of the following characters shift, and this event can change the inner structures of the genes, causing not only a change in the meaning - as it is in reality - but perhaps making impossible the reading of the genes. In most of the evolutionary models genes are rather rigid structures without any flexibility. A model of an open-ended evolution process would require more flexible, error-tolerant encoding methods that can handle not only the preprogrammed properties, but allow changes of lengths and inner structures of the genes without making impossible the reading of genes. In this case the size of the genome as well as the single genes can vary freely, so this mechanism can serve as a basis for modeling an open-ended evolution.

The structure and the function of the genome

We endeavored to apply the biological principles (Smith-Keary 1991) consistently in our model. The model-genome is built of four types of characters (0, 1, 2, 3), similarly to the four nucleotides of the DNA (adenine, cytosine, guanine and thymine). In most of the Alife models genes consist of a predefined number of characters, and their meaning depends on their position inside the gene. In reality genes are homologous nucleotide-sequences, in which there are no specially positioned characters, so the "meaning" of a gene is not hidden in the single nucleotides, but in the protein, which results from the translation of the whole gene. We applied this principle, therefore the meaning is not assigned to the position of a certain character. In our model the result of the translation of a certain gene is a set of sub-properties. The composition of this set defines the property and its quantitative value encoded in that gene (also, in reality the amino acid sequence defines the property of a certain protein). As the meaning of a gene is not assigned to the positions of the characters inside, the genome can change more flexibly (e.g. the number of the characters of a gene, the length of a single gene and also, the whole genome can vary freely, moreover, the genes can change their position inside the genome) without losing its functionality. This model-genome does not have a strictly organized sequential structure, it can be created as a totally random sequence, and there is no need to predefine the lengths of the genes and their order in the genome. This structure usually contains less information than it could, but it is also similar to reality, because in the real genomes there are large meaningless sequences among the genes. So this kind of encoding is closer to reality than the common strict way.

The characters of the genome are read in twos, so the codons consist of two characters. Because we have four "nucleotides" (0, 1, 2, 3), the codons are double figures in the base four numerical system, so we have 4 x 4 = 16 different codons. All of them can be connected to a hexadecimal figure (0 – F), which are the results of the translation (the "amino acids"). On the basis of this simple coupling rule we can easily create the "genetic code" of the model (see Fig.1.). In fact real codons consist of three characters, so there are 4 x 4 x 4 = 64 triplets, but because of the degeneracy of the genetic code they encode only 20 amino acids. For this reason mutations in the third letter of the triplets rarely cause real changes. Using two letter codons our encoding system does not have degeneracy, so the ineffective mutations are eliminated. This modification leads to the acceleration of the evolutionary process, but apart from this feature the organizing principles resemble reality, and the number of encoded units is also similar (16 and 20).

![The genetic code of the model](image)

Fig. 1. The genetic code of the model

The genome does not have special positions in the sequence, all of them are equivalent, regardless of their position. Because of this there must be a "start" and a "stop" codon to sign the forepart and the end of the genes, just like in reality. In our model the "start" codon is "1 1" (corresponding to AUG codon in reality), while the "end" codon is "00" (corresponding to UAA, UGA and UAG). The "1 1" in the base four numerical system has the hexadecimal equivalent "5", similarly to the real genetic code where the "start" triplet (AUG) also encodes an amino acid (Metionin). This kind of encoding makes possible the emergence of overlapping sequences of genes, which means that "1 1" marks the starting point of the genes, but if in a sequence of a gene we find another "1 1" codon (i.e., before a "00" closes that certain gene), then this new "1 1" codon has two different meanings. In the original gene that is under translation this codon means a
sub-property ("5"), while at the same time this codon serves as a "start" sign for another gene. The sequence of this latter gene is the same as the sequence of the original from this point, and all of them continue until the first "00" codon. So the same part of the genome can encode more than one set of sub-properties (more than one "protein", see Fig. 3). This interesting phenomenon – which increases the efficiency of information storage – is also known in biology, we can find examples of multiple readings for genes in viruses and Prokaryotes.

**The process of translation**

In our model there are 16 “hexadecimal amino acids” (0 – F) that are the equivalents of the codons. Their determination is inevitably high-handed, since they are not influenced by the laws of physics and chemistry, which determine the properties of real amino acids. The features have to be defined by the modeler, but we have to endeavor not to restrict the potentials of the model.

In reality a certain amino acid does not have a special “meaning”, its function depends on the protein which the amino acid molecule is built in. So we cannot order a special function to a single hexadecimal amino acid (and its codon that corresponds to it). The carrier of a function is the whole protein, and a single amino acid can only modify it. Therefore we have to order somehow the functions to the whole proteins (or genes) and not to their separate parts. We connect sub-properties to larger functional units of the proteins (that are called “domains” in real protein molecules). So we have to find these domains in a certain molecule, and they determine its function, but the quantitative features of this function will be modified by the single amino acids of the domains. We defined the repeated hexadecimal amino acid sequences as separators between the domains. If a protein does not contain at least three uniform amino acids successively, then it comprises only one domain. The functions of the domains are determined by the total of the values of its hexadeimals.

Determining the features of the sub-properties we have to keep in view the most general abilities and attributes of a living cell that can be combined with each other and result in different cell-types.

<table>
<thead>
<tr>
<th>Domains</th>
<th>Type of sub-property</th>
<th>Description of sub-property (The proteins containing these units have the required properties)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 3</td>
<td>MEMB</td>
<td>Membrane proteins</td>
</tr>
<tr>
<td>4 – 7</td>
<td>IONCH</td>
<td>Ion channel proteins (determine the passive and active electric properties of the cell)</td>
</tr>
<tr>
<td>8 – B</td>
<td>CONTR</td>
<td>Gives the possibility of active contraction in a certain part of the cell</td>
</tr>
<tr>
<td>C – F</td>
<td>SEQ</td>
<td>Create identifier sequences in a protein molecule</td>
</tr>
</tbody>
</table>

Fig. 2. The sub-properties and their characteristics

<table>
<thead>
<tr>
<th>Type of protein:</th>
<th>Regulatory molecule</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ligand dependent ion channel</td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 3. The process of translation**
Regulation of the cells

More or less biologically inspired modelers of neural networks often try to get their models close to reality by planning not an accomplished, well-formed neural net, but an "embryonic-state" one that goes through an ontogenesis and develops by itself (Nolfi and Parisi, 1994a). Some of these models try to simulate ontogenesis without taking into account the active role of the genome in it. In these models turning on and off a certain gene is solved by the mutation of a regulatory gene, so the state of a gene is not influenced directly by the environment, therefore it is not changeable during the ontogenetic process. This solution is not realistic, although it can be suitable for robotic models (Nolfi, Miglino and Parisi 1994; Nolfi and Parisi 1994a, 1994b). Other models try to imitate a more realistic controlling role of regulatory genes inspired by the operon-theory of molecular genetics (Eggenberger 1996, 1997).

Every cell of a living organism contains the same genetic information, but cells are different from each other, because the environmental stimuli activate different genes in them. Most of these stimuli have their origin in the cells themselves. So cells create their own environment inside the body, which influences their further development. In biological systems this regulation is carried out by regulatory genes (Smith-Keary 1991). This needs special molecules called transcription factors, which can be attached to these genes turning them on and off. The number of active genes is changing continually because of the feedback between the environment and the genome, so the phenotype is continuously adapting to this environment. In this model the plasticity has two levels, just like in reality: genotypic and phenotypic plasticity. The former makes possible the evolutionary adaptation of the "species", while the latter makes possible the adaptation during the lifetime of a certain individual.

Chemical regulation has three basic types: intracellular regulation, intercellular regulation directly through the cell membrane and receptor-mediated intercellular regulation. These types of communication are successfully modelled in evolutionary robotics to create and format simple neural nets, and in a model of morphogenetic processes (Eggenberger 1996, 1997). Chemicals have three important features in this context: the type of the molecule (this is important in binding to a receptor), the weight of the molecule (determines the diffusion constant) and the half-life (determines the duration of its effect).

Intracellular regulation (see Fig. 4a): Some sequences produced by the cell itself can connect to a certain gene and work as a regulatory unit repressing the expression of that gene. In our model, differently from others, there are no predefined regulatory genes. To become a regulatory gene two requirements have to be met. One is a "protein sequence" (product of another gene) that can link to that certain sequence of the genome. But this alone is not enough, because if the gene sequence is a meaningless part of the genome (so it is not between a "start" and a "stop" codon) the linking has no effect. But if this gene-sequence is a real gene, then the linking prevents it from being expressed.

Intercellular regulation (see Fig. 4b-c): cells can emit chemical signals into the intercellular matrix. Naturally these molecules are also the products of the cells (the features of these chemicals are determined by their subproperties). If a cell has a proper receptor in its membrane (the identifier sequences of the receptor and the signal correspond to each other), then this cell is sensitive to this signal. The effect of linking is determined by the receptor, and not the signal. There is a different type of intercellular signal that has an effect directly on the genes of another cell without linking to a receptor. (This kind of signal can go through the membrane of the cells and can have an effect on the genes in the same way as the intracellular regulatory molecules.)

![Fig. 4. Levels of the gene regulation](https://example.com/figure4)

This model of regulation does not define the number of the regulatory genes and the signal molecules in advance; every feature can change by mutation and recombination. So it is not certain whether at the start of a simulation every signal will have a receptor, or if there will be any receptors at all. So in the beginning it is probable that there will be many individuals that are incapable of living, but the fortunate ones have almost unlimited chances to evolve.

Properties of nerve cells

Nerve cells have the most important role in the regulation of the behavior of an animal (or an animat), so in the process of creating a model of adaptive behavior one of the most important steps is to choose a suitable type of model-neuron. There are two restrictions that influence our decision. On the one hand we want to create a biologically relevant model, so the modeled nerve cells cannot be...
oversimplified. On the other we have to take into consideration that if we want to model an evolutionary process as well, the nerve cells have to have parameters that are well encodable in that kind of genome we outlined above. In consideration of these requirements we created a model neuron which functions based on the Goldman-Hodgkin-Katz equation:

$$E = \frac{RT}{F} \ln \frac{P_{Na}[Na]_{out} + P_K[K]_{out} + P_{Cl}[Cl]_{in}}{P_{Na}[Na]_{in} + P_K[K]_{in} + P_{Cl}[Cl]_{out}}$$

$E$ is the actual value of the membrane potential, while $R$, $T$ and $F$ have their usual meanings, $P$ is the permeability of the sodium ($Na$), potassium ($K$) and the chloride ($Cl$) ions, in the square brackets there are concentrations of these ions inside (in) and outside (out) the cell. The concentrations have values that are usual in nerve cells, so they are not encoded. The active electric properties of a nerve cell that functions based on this equation depend on the values of three parameters: $P_{Na}$, $P_K$ and $P_{Cl}$. We had to find a solution to encode them in the genome in a way that is biologically reasonable.

The biological subjects of membrane permeability are the ion channel proteins. Their kinetic properties determine the flow of the ions through the membrane, therefore they are the bases of the active electric properties of the nerve cells (Ganong 1987; Prosser 1991). The kinetics of these ion channels can be well described by built-in gating particles. These are hypothetical parts of the ion channel molecules, but in all probability they have equivalents in reality. E.g., in a common voltage-dependent sodium channel there are two types of them, one is an opening ($m$), while the other is a closing ($h$) particle. The channel is opened only if they are in a suitable position. As $m$ and $h$ are probability variables, their value is between 0 and 1, and the probability of being open for a sodium channel is $m^3h$, because there are three pieces of $m$ particles connected to a channel.

The values of these probability variables are calculable with the help of some simple equations described by Hodgkin and Huxley (Finkelstein and Mauro 1977; Hille 1984). The equations calculate the values of $\alpha$ (activity constant) and $\beta$ (inhibitory constant), which are used to calculate $\tau$ (time constant), which then is needed to calculate the probability variable. In the general forms of the equations we substituted variables ($a$, $b$, $c$, $d$) for the numbers connected to a certain ion channel. Knowing their values we can derive $P$, which determines the kinetics of a modeled ion channel. These parameters seem suitable for encoding the properties of the ion channels, because changing their value by mutation and recombination results in a number of ion channels that have more or less different properties ($m$ is the value of the probability variable after a long term, $m(t)$ is its value at $t$ time).

$$\alpha = a_{\alpha} \exp \left( \frac{-V}{c_{\alpha}} \right)$$

$$\beta = a_{\beta} \frac{b_{\beta} - V}{\exp \left( \frac{b_{\beta} - V}{c_{\beta}} \right) + d_{\beta}}$$

$$m_\infty = \frac{\alpha}{\alpha + \beta} \quad \tau_m = \frac{1}{\alpha + \beta}$$

$$m(t) = m_\infty - (m_\infty - m_0) \exp(-t/\tau_m)$$

This way of encoding is indirect enough to simulate the genetic process, in which a certain biological feature is the result of the functioning of a number of different factors, and not only a single number which directly determines that property. Being the model-genome a randomly generated character-set, the number of domains of a certain protein molecule is not predefined, and so some of these parameters can be missing. These "mutants" can be workable molecules, yet they have different properties. In our model the protein-domain type (or sub-property) that encodes the values of these parameters is called IONCH. So if a protein contains this domain-type, it can become an ion channel, but the resulting function depends on the other domains built in that protein as well.

Fig. 5. The values of the parameters in the case of a sodium ion channel.
An ion channel in a real cell can be not only voltage- but ligand dependent as well. In this case the activity depends on the binding of a ligand molecule, and not on a voltage threshold. This type of ion channel needs a receptor sequence that can bind a suitable ligand. If the result of a gene translation is a molecule that has IONCH domain(s) and a SEQ domain (that carries the receptor sequence), then it is defined as a ligand dependent ion channel molecule. Ligands are simpler molecules, which also have a SEQ domain. If the domain of the ion channel corresponds to the sequence of the ligand, they get bound to each other, and it results in the activation of the ion channel molecule.

Nerve cells have passive electric properties as well: the time constant (τ) and the space (or length) constant (λ) of the conduction. (This time constant is not the same as the previous one discussed above.) These passive electric properties serve as a basis for the appearance of the active ones. The time constant is the length of time during which the value of membrane potential decreases to its 1/e. The space constant of the membrane is the distance where the membrane potential decreases to its 1/e (Ganong 1987; Prosser 1991). Both of them are derived from the cable-equation, which describes the passive electric properties of biological membranes. These properties play an important role in the formation of behavior during the early evolution of the nervous system (Albert 1999a, 1999b), because in this stage there is no action potential, so passive conduction is the only way to pass electric stimuli. In the model there is a protein-domain type that has the capability of modifying the time- or space constant (they are signed as MEMB in the table, see Fig. 2).

The third important feature of the nerve cells is the ability to move during the early stages of the ontogenesis and to grow processes later. These two activities have similar biological mechanisms. Both of them need contractile molecules built in the cell membrane, and their working is the result of the change of the membrane potential. In our model there is a protein-domain type signed as CONTR (see Fig. 2), which carries this ability of a protein molecule. There is a parameter encoded in this type of domain that determines the voltage threshold of the activity. So the migration or forming processes are not a directly encoded and predefined property of a cell, but the result of its inner state.

The animat

At the recent stage of our model the execution of morphogenesis is not perfect yet (these are only technical problems). So we temporarily applied an animat from our previous model that is well-tried and suitable for testing the behavior resulting from this new control mechanism. This animat is a Hydra-like being that is able to perform feeding behavior (Albert 1999a, 1999b; see Fig. 6). The body of the animat consists of three cell-types, epithelial cells, muscle cells and nerve cells. Epithelial cells give the "framework" of the body, defining its shape and boundaries. The layer of these cells provides points for the muscle cells to anchor, and the movements of the animat will result in the change of position of the affected epithelial cells, so these cells serve as a kind of skin. Nerve cells and muscle cells are embedded in the layer of epithelial cells. Muscle cells have fixed positions, while nerve cells can move in the "matrix" of epithelial cells, if they have the capability of movement. Chemical signals emitted by the nerve cells also spread in this matrix by diffusion. The number of nerve cells results from the division of cells, so it can change during the lifetime of an animat. When the "life" of an animat begins, it has only a few "multipotent" cells. If they have the ability of division (it is an encoded property), they start multiplying, but this is not enough to form a nervous system, as it requires the ability to establish connections as well. During the tests the most successful individuals had about 600 – 800 nerve cells, but the fitness also depended on the properties of these cells. In the final version of the recent model the bodily construction will be the result of an ontogenesis, similarly to the nervous system, which directs the behavior.

The environment is modeled as a box. As the animats are sessile, the interaction between the individuals would be minimal, so we perform the tests with only one individual in the box at a time. The stimulus is the appearance of a piece of food, which slowly sinks to the bottom of the box. This is a chemical stimulus for the animat and its strength decreases exponentially with the distance. If the animat is able to catch at least one piece of food, the test is continued. The feeding is successful if the animat not only touches the piece of food, but it also "eats" it, in other words, it gets food particles into its coelenteron through the mouth. The fitness depends on the success of feeding (for details see Albert 1999a, 1999b).
Testing the model

During the tests we examined the success of the feeding behavior of the animat. Then we performed some special tests examining the capabilities of the ontogenetic process (naturally we could only study the ontogenesis of the nervous system and not of the body).

First we created some sets of genomes generating totally random sequences of numbers, only their lengths were defined in advance (There were sets of three different lengths: 100, 200, 300, 400, 500 characters per genome). This was followed by the translation, during which the program read the genes of a certain genome (sequences started with “11” and ended with “00”) and determined the encoded sets of sub-properties. At the same time this step was a screening test: we rejected those individuals that were obviously not viable (e.g. they did not have real genes starting with “11” and ending with “00”, only meaningless sequences). There were only a few of individuals of this kind. We also rejected those ones that have real genes, but these genes could not produce viable individuals (e.g. they had no receptors or tools to respond to a stimulus). Since genomes were totally random sequences, there were a lot of individuals in this group. The rest of the genotypes were qualified as potentially viable. (They were about 10% of the total amount of genotypes. The longer the genomes were, the more viable individuals were resulted.) We tested the feeding behavior only of these ones.

Results and discussion

The results of the tests show that only a small part of the potentially viable individuals of the first generation were able to control successfully the feeding behavior of the animat. It is not surprisingly few if we consider the structure of the genome and the way of generating them. Moreover, the successful ones shed light upon the self-organizing ability of the genome structured by this principle, because the genes and their functions arose by themselves from totally random character sets. If there are more or less successful individuals, then the advantages of the flexible genome could manifest themselves during the later phase of the evolutionary process. This is probably because the preprogrammed properties of the “traditional” genomes can evolve easily by using GA, but this evolution has a limit, because when the best genomes are found in a certain environment, there is no other chance for new, better features to appear; the evolution “gets exhausted”. The advantage of the flexible genome is that it has the ability to ensure an open-ended evolution including the emergence of new properties. Under these temporary testing circumstances it is the body construction of the animat and the simple environment that limited the possibilities of evolution, and not the genome.

We found that in the beginning several fortunate circumstances are necessary to create a more or less viable individual by generating random genomes. This is the reason of the numerous unviable individuals (~90%) after generating the starting genomes of the first generation. The members of the second and further generations are the offspring of merely the viable starting individuals (~10%), so the fitness grows rapidly. After about 300 generations the success of feeding behavior stops increasing. We have to stress that the animat we used in this test was originally created for a previous model and it has strong limitations in an evolutionary respect. This is only a stopgap arrangement that is applicable to the first test of this new genetic encoding and the neural control of behavior. During the evolutionary process some characteristic types of nerve cells appeared in the modeled nervous systems (see Fig. 7.)

![Fig. 7. Some interesting firing patterns of the modeled nerve cells](image-url)
development of the nervous system, but this process was only successful in the "best" individuals. The lives of individuals begin with the ontogenesis and this process lasts throughout the whole of their lifetime, just like in reality. So the ontogenesis is not separated from the "working" of the individuals, which results in the behavior. (Most of the models handle ontogenetic process — if it exists at all — as a separated beginning part of the life, during which the neural network forms, but after this there is no other chance to change the anatomical structure of the network.) The successful animats must have the ability of adaptive behavior from their "embryonic state". In this developmental state there are two successful strategies. One is to develop in a way that continuously ensures the adaptive behavior, the other is to pass rapidly this first period (Nolfi and Parisi 1994a, 1994b).

One of the most interesting qualitative features was that there were individuals which had a "fluid" nervous system, in which the cells were in continuous migration during their lifetime, while their nervous systems preserved the capability of controlling the feeding behavior. The synapses of the cells also changed continuously, but because of the large number of cells they could form new connections instead of the ceased ones. It is very similar to the nervous organization of Hydras, because in these simple animals nerve cells continuously come into being by cell division during the lifetime and they migrate along the body, nevertheless this simple network-like nervous system can preserve its operating ability (Bode 1992). Our previous model (Albert 1999a, 1999b) also showed that in animats at the starting point of nervous evolution with a simple bodily construction (e.g. Hydra and other Cnidarians) and therefore not having special locomotor organs, which would need an advanced nervous control, the large number of nerve cells in a randomly connected network-like nervous system can control the behavior successfully. This result is in harmony with the physiological data (Robson 1975; Spencer 1991).

Another interesting feature that is the result of the lifelong-ontogenesis is the ability of adaptation to the changes of anatomical relations. If we "cut asunder" an animat, in some cases it has the ability to regenerate its neural net, and the resulting "child-animats" can survive this interference and feed more or less successfully. (During the simulation of this process we did not take care of the details of the anatomy, we only pulled apart the tube-like animat at its centre, and the resulting half-animats were considered as two smaller ones.) We only studied the half nerve systems and the behavior produced by them. This is a simple model of the asexual propagation of Hydras, during which an individual produces offspring by gemmation (Bullock and Horridge 1965; Robson 1975).

The "growing together" of two animals had similar results; in some cases this process led to workable animats, which could feed successfully. This surprising regenerating ability is possible because of the simple diffuse structure of the nervous systems in which there are no specialized connections between the cells and there are no special locomotor organs that would need these connections for their proper working. These simulation results are in agreement with the physiological data, because Hydras and other Cnidarians, which have the most ancestral network-like nervous system, have a very good self-regenerating ability.

Conclusion

All of these early state simulation results demonstrate that our model could be suitable to become a biologically well established model of the evolution of the nervous system. One of the crucial factors of this model is that genome has to exceed its common, passive role and the rigid structure, and has to have the ability to perform the tasks consisting of two parts they have to perform in biological systems. On the one hand it has to make possible developing properties that are not preencoded in the genome, on the other hand it has to ensure the chance to perform the ontogenetic processes.

If we can create a biologically accurate and well-established Alife model of the evolution of the nervous system in relation to the behavior, then it can help Alife to become a useful modeling method for neurobiology. This would help us to make the information flow to a really two-directional process between Alife and biological research. This fruitful relation should accelerate the process that leads to the better understanding of the mysterious phenomenon called intelligence.

References


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