Evolving Artificial Neural Networks that Develop in Time

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Abstract

Although recently there has been an increasing interest in studing genetically-based development using Artificial Life models, the mapping of the genetic information into the phenotype is usually modeled as an abstract process that takes place instantaneously, i.e. before the creature starts to interact with the external world and is tested for fitness. In this paper we show that the temporal dimension of development has important consequences. By analyzing the results of simulations with temporally developing neural netwoks we found that evolution, by favouring the reproduction of Os which are efficient at all epochs of their life, selects genotypes which dictate early maturation of functional neural structure but not of nonfunctional structure. In addition, we found that development in time forces evolution to be conservative with characters that mature in the first phases of development while it allows evolution to play more freely with characters that mature later in development. Finally, characters that mature in the first phases of development tend to be phylogenetically older than characters that mature later.

Keywords: Development, Recapitulation, Alife.

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Introduction

Development in natural organisms is a temporal phenomenon. There is not a single phenotype that is the final result of the genotype-to-phenotype mapping - the mature or adult form of the phenotypical individual - but the individual passes through a sequence of phenotypical forms from the initial cell (the egg) to the adult form. Therefore, the information contained in the genotype is reflected not only in the initial state of the organism (at birth or conception) but also - and crucially - in the succession of changes that occur in the organism throughout the organism's life (although these changes may be more pronounced during the organism's developmental age).

Such developmental issues have potentially important evolutionary consequences. Because in many animals (including humans) the gametic cells are sequestered from the embryology affecting the rest of the somatic cells, the "Weissmann doctrine" has often been invoked to decouple developmental issues from population genetics. Like all other characters, however, the developmental process itself evolved and the wide range of developmental "strategies" employed across the various phyla points to important interactions between development and evolution (Buss, 1987). For example, the effective in utero insulation of the mammalian fetus in some species has allowed the developmental process much more flexibility during this stage than in species (frogs, for example) where the juvenile's viability is costantly being tested. Gould has been a particularly vocal advocate of ways in which apparently minor "heterochronic" mutations affecting the relative timing of subprocesses of development can have drammatic consequences (Gould, 1977). At a more local level, features of a species' evolved "life history strategy" such as when individuals reach reproductive maturity and the evolutionary consequences of senescence must also be considered part of any developmental account.

Recently, there has been an increasing interest in studing development using Artificial Life models. Wilson (1987), Kitano (1990), and Belew (1993) have proposed models that include a process resembling the cellular duplication and differentiation process. Nolfi and Parisi (in press) have proposed a model in which the connectivity of the nervous system of artificial creatures grows in a way that has similarities to neural development in natural organism. Cangelosi, Parisi, and Nolfi (1994) and Dellaert

and Beer (1994) have proposed still more complex models in which both the cellular duplication and differentiation process and the neural development process are simulated. In Dellart and Beer, in particular, the molecular level is also simulated (even if, of course, in a very simplified way) with a boolean network that emulates genetic regulatory processes (see also Kauffman, 1969). However, in all these works and, as far as we know, in all published works in Artificial Life, the mapping of the genetic information into the phenotype is viewed as an abstract process that takes place instantaneously, i.e. before the creature starts to interact with the external world and is tested for fitness.

Several published works that use neural networks to represent the nervous system of artificial creatures describe models that entail some form of learning that produce after-birth changes in evolving creatures (e.g. Parisi, Nolfi, and Cecconi, 1991; Ackley and Litmann, 1991; Nolfi, Elman, and Parisi, in press). However learning is esogenous change, i.e. change caused by the interaction of the individual with the external environment. Development is endogeneous change, i.e. change due to genetically inherited information which is already "inside" the organism. We are aware of the fact that learning and development are interconnected processes and that it may be difficult to separate them in real biological organisms. However, the possibility to study development without learning in artificial organisms is an advantage if one wants to understand the influence of development on evolution. (For a model that incorporates both development in time and learning, cf. Nolfi, Miglino, and Parisi, 1994).

If development is realized as a temporal succession of phenotypic forms a number of important research questions that cannot be posed with nontemporal mapping models are open to research with simulations.

The fact that development occurs in time implies that all the successive phenotypical forms which constitutes development may be subject to evaluation in terms of fitness. While in nontemporal mapping schemes it is only the single adult form whose fitness is measured and which determines the individual's reproductive chances, if one adopts a temporal genotype/phenotype mapping each successive developmental form must demonstrate its value in terms of fitness. In fact, the global fitness of an individual is likely to be a complex function of the separate fitnesses of each of the successive developmental forms of the individual. On one side, a particular developmental form has the role of preparing in the best possibile way the future forms. On the other side, an intermediate developmental form is not only a way-station to the final adult form but it is something which must possess properties that allow it to survive and to contribute independently to the global fitness of the organism. Therefore, evolution won't shape only the final adult form of a particular organism but also all the intermediate developmental forms. And it will shape the intermediate developmental forms by taking both their role as way-stations to the adult form and their intrinsic fitness into consideration. Therefore, a first important question that can be asked is how evolution shapes the particular sequence of developmental forms that characterizes a particular population of organisms. Is there acceleration or retardation of developmental changes from one generation to the next? What is the role of heterochrony, i.e. the differential speed of development of the various parts and traits of an organism, in determining the final adult form?

But it is not only the case that evolution shapes development. Development also constrains evolution. Since an individual is a particular succession of developmental forms, evolution cannot just change a single developmental form in isolation. The possible change in fitness caused by the new developmental form depends on the entire succession of forms in which the changed form is included. Hence, the entire succession of developmental forms of an organism will constrain the evolutionary changes that will be retained or discarded. A second research question is then how the particular succession of forms that characterizes the development of an organism constrains the types of evolutionary changes that can occur at the population level. Since evolution shapes the genotype and the genotype controls development, evolution is crucial to understand development. But since, development constrains evolution, development is crucial to understand evolution.

A final question is a classical one: What is the relationship between evolutionary changes and developmental changes? Are there similarities between the two? Does development (ontogeny) recapitulates evolution (phylogeny)?

The model

Let us begin by assuming that our ultimate goal is to create an organism (O) which is able to find and

eat food in its environment. O includes two components: (a) a phenotypical neural network which controls O's behavior in the environment, and (b) some genetic material (genotype) encoding developmental instructions which generate a certain number of neurons and control the growing and branching process of the neurons' axons (Nolfi and Parisi, in press). The result of this growing process is a neural network that represents the nervous system of the corresponding O. The architecture and connection weights of such a network determine the way in which O responds to environmental stimuli, i.e. its behavior. O's behavior determines its fitness, i.e. its reproductive chances, through O's interaction with the environment to which it is exposed.

Each O lives in a simulated environment which is a two-dimensional square divided up into cells. At any particular moment O occupies one of these cells. A number of food elements are randomly distributed in the environment with each food element occupying a single cell. O has a facing direction and a rudimentary sensory system that allows it to receive the angle (relative to where O is currently facing) and the distance of the nearest food element as input. O is also equipped with a simple motor system that provides it with the possibility, in a single action, to turn any angle from 90 degrees left to 90 degrees right and then move from 0 to 5 cells forward. Finally, when O happens to step on a food cell, it eats the food element which disappears.

O's genotype is represented as a string of 0 and 1. It has a fixed length (1600 bits) and is divided up into 40 blocks, each block corresponding to a single neuron. Since some blocks may not be expressed, the mature phenotypical network may include less than 40 neurons. Each block encodes developmental instructions specifying a number of properties of the corresponding neuron (Figure 1).

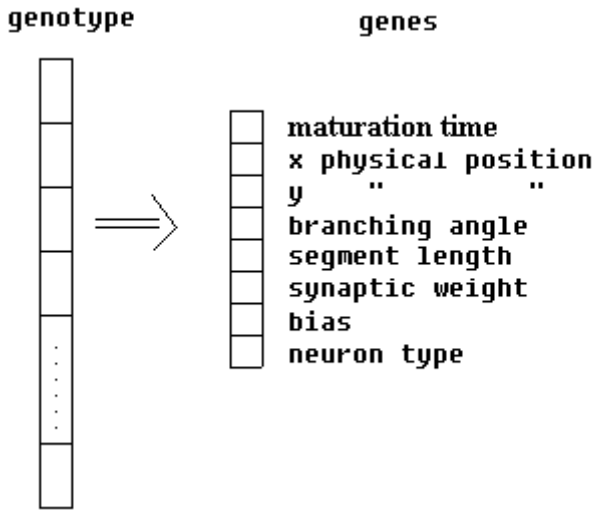

Figure 1: O's genotype. Each block contains a set of developmental instructions for the corresponding neuron. The developmental instructions specify when during ontogeny the block will be expressed (this is the 'maturation time gene'), the x and y spatial coordinates of the neuron in the nervous system's bidimensional space, the angle of branching and the length of the branching segments, the weight of the connections departing from the neuron (same weight for all connections), the activation bias of the neuron, the type of the neuron: sensory, internal, or motor.

When a growing axonal branch of a particular neuron reaches another neuron a connection between the two neurons is established. Figure 2 shows the growth of axons resulting from a random genetic string on the left and the resulting neural network on the right. Functional neurons and connections, i.e. neurons and connections that actually map input into output and therefore can influence the O's behavior, are represented with larger circles and thick lines.

The functional network determines O's behavior through the interaction with the environment. At each time step, O receives a pattern of activation values on its sensory neurons encoding the position of the nearest food element relative to O. Such an input determines, through a spreading activation process, the activation value of the internal and output neurons. These last neurons determine O's motor reaction

to the current input, i.e. O's behavior.

Since different blocks in an individual's genotype may contain different 'maturation time genes', different neurons will grow their neurites at different times during the life of the individual. The branching axons will establish connections that can vary from one epoch to the successive epoch of life because new connections can be added. Therefore, the behavior of the individual in the environment will be controlled by a succession of different phenotypical forms of its nervous system and, as a consequence, the behavior itself will be different in various epochs of life.

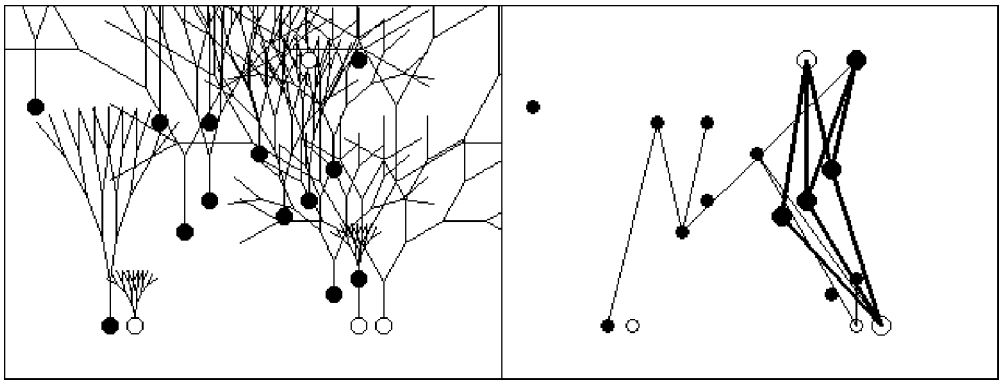

Figure 2: Development of a neural network from a randomly generated genetic string. The growing and branching process of axons is shown on the left side of the Figure. The resulting neural network after removal of nonconnecting branches is shown on the right side. Isolated or nonfunctional neurons and connections are represented by small circles and thin lines. Functional neurons and connections are represented as large circles and thick lines. The bottom layer contains sensory neurons, the upper layer motor neurons, and the remaining layers internal or hidden neurons.

In order to obtain adapted Os, i.e. Os that exhibit an efficient food collecting behavior, we simulate an evolutionary process using a genetic algorithm. (Holland, 1975). We ran 20 simulations each starting with 100 different randomly generated genotypes. This is generation 0 (G0). Os of G0 are allowed to develop and "live" for 8 epochs, each epoch consisting of 250 actions in 5 different environments (50 actions each). The environment is a grid of 40x40 cells with 10 pieces of food randomly distributed in it. Os are placed in individual copies of these environments, i.e. they live and develop in isolation. At the end of their life (2000 actions) they are allowed to reproduce. However, only the 20 individuals which have accumulated the most food during their life reproduce by generating 5 copies of their genotype. These 20x5=100 new Os constitute the next generation (G1). Random mutations are introduced in the copying process resulting in possible changes in the phenotypic network and/or in the time of development of different subparts of the phenotypic network. The organisms of G1 are also allowed to live for 2000 cycles. The process continues for 1000 generations.

The neural development model is inspired by biology but it does not pretend to be a realistic model of neural development. It is simplified in several respects and in particular:

(a) It contemplates only growth and no degeneration or disappearence of parts of the developing neural structure. One could easily include in the model some form of "death" of units or connections, either programmed in the genotype or as a consequence of time past since the appearence of the particular unit or connection or because of experience and (in)activity. However, in order to keep the model as simple as possible, we decided to not allow cellular degeneration and "death".

(b) The developmental process is extremely simplified with respect to what happens in natural organisms. In particular, the cell differentiation and migration process, the effects of the interactions among neighboring cells, and the role of extracellular entities and trophic factors are not reproduced in the model. (For models which are somewhat more biologically plausible, cf. Cangelosi, Parisi, and Nolfi, in press; Dellaert and Beer, 1994).

(c) The time of appeareance of neural material during the ontogeny of the individual is directly encoded in the genotype while in natural organisms it results from an interaction between genetic products and possibly exogenous information. This implies that in our model neurons and groups of

interconneted neurons can be anticipated or postponed in ontogeny independently of each other. This in turn implies that they cannot assume interphene functions. A character has an interphene function when in addition to having an adaptive value with respect to the external environment it assumes an adaptive value as an intermediate stage which is necessary to induce subsequent developmental characters (cf. Mayr, 1994). We choose to use this type of implausible coding both to keep the model simple and to be able to track diacronic changes phylogenetically.

(d) In our model individual fitness, i.e. the individual's reproductive chances, is calculated by summing the number of food elements collected by the individual during each epoch of life. This implies that the individual's ability to find food at different phases of the individual's ontogeny has the same impact on fitness. In natural organisms the situation is more complex and it can be different in different species. In most species the first phases of ontogeny appear to be the most important ones because if individuals are not able to collect enough energy or to escape from predators they can easily die. In other (e.g. mammal) species, with strong parental care behaviors, the survival in the first phases of ontogeny is facilitated and almost independent from the ability of the individual. As a consequence, one can hypotesize that the ability in the first phases of ontogeny weights less on the reproductive chances of the individual.

Simulation results

If we look at how fitness changes phylogenetically and ontogenetically we observe an increase in both cases (Figures 3 and 4). If we count the number of food elements our Os are able to collect during their life across the 1000 generations, we see that Os of successive generations are increasingly able to approach food elements. Figure 3 shows the fitness value of the Os of the winning lineage for each of the 1000 generations. (Average of 20 different simulations.) The winning lineage is the lineage of the best individual of the last generation. The lineage is constituted by this individual, by its (single) parent in the preceding generation, by its grand parent, its grandgrand parent, etc., until the originator of the lineage in the first generation is reached. The evolutionary increase in fitness implies that generation after generation Os are able to adapt their architecture and synaptic weights to the evolutionary task. The number of food elements available in the entire lifetime of an O is 400. On average the best Os of the last generation reach 57% of these food elements, with one of the 20 simulations reaching 95%.

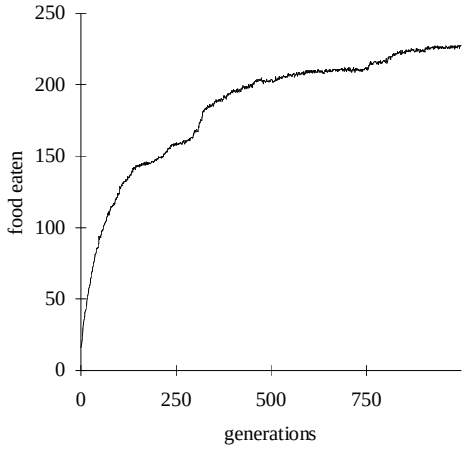

Figure 3: Fitness (= total number of food elements eaten during life) of the Os of the winning lineage. The graph represents the average result of 20 simulations starting from initial populations of different randomly generated genotypes.

Figure 4 shows the number of food elements Os are able to eat in different epochs of their life. The amount of food eaten increases during life, especially in the early epochs. This implies that genetically controlled developmental changes in neural structure induce behavioral changes that lead to

progressively more effective behaviors with increasing age. In other words, selective reproduction and mutations in successive generations of individuals results in the evolutionary emergence of developmentally adapted genotypes.
This might in part be explained as a necessary by-product of our model of development. Our model contemplates neural growth but no neural degeneration or programmed cell death. Hence, development tends to be a one-way progressive process with addition but no subtraction of neural structure. However, even the addition of newly developed neural structure could in principle have negative effects on fitness. Apparently, the evolutionary process in our simulations is able to rule out this possibility.

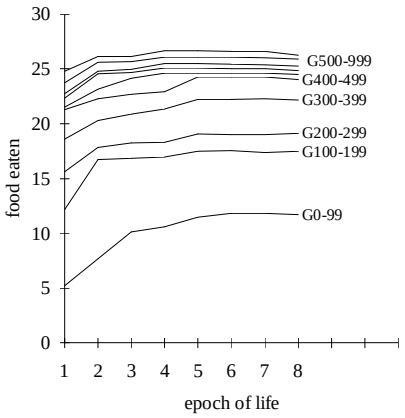

Figure 4: Number of food elements eaten in successive epochs of life by Os of the winning lineage. The graph shows the average result of 20 different simulations. Each curve represents the average performance of 100 successive generations.

But the role of evolution in selecting an adaptive course of development becomes evident if we analyze more closely what happens during development in our organisms. Our model implies that the time of development of both functional and nonfunctional neural structure is under genetic control. As will be recalled, the functional structure is that part of the neural structure generated during development which maps input into output and therefore controls the individual's behavior. The nonfunctional structure (units and connections) is also generated during development but is not part of the input/output pathways and therefore has no role in controlling behavior. We can ask how evolution shapes the temporal dimension of development both generally and with respect to these two components of neural structure. In other words, is there a general tendency to evolutionarily anticipate or posticipate the generation of neural structure during development and are there differences in this tendency between functional and nonfunctional structure?

An answer to this questions is in Figure 5 which shows how the average epoch of life in which functional and nonfunctional units are generated changes evolutionarily. (Functional units are units that are part of the functional network while nonfunctional units are part of the nonfunctional neural structure.) Functional units tend to develop earlier in life than nonfunctional units from the very first generations on and the effect of evolution is to increase the average difference in the epoch of development for the two types of units. More specifically, there is an evolutionary tendency for units that are part of the functional network to be anticipated in their epoch of development across generations.

A similar conclusion is reached by counting the number of neutral and non-neutral mutations in the genotype that cause posticipation or anticipation in the time of development of individual neurons (cf. Figure 6). Neutral mutations are those mutations which do not translate into a change (increase or decrease) in fitness. Non-neutral mutations are those that cause either an increase (adaptive mutations) or a decrease (maladaptive mutations) in fitness. Non-neutral anticipations (i.e. mutations affecting the 'maturation time genes' that determine a modification in the individual's fitness and cause an anticipation in the time of development of neurons during ontogeny) largely outnumber posticipations. On the contrary for neutral mutations, posticipations and anticipations do not differ significantly. (Notice that in the winning lineage non-neutral mutations are likely to be adaptive rather than maladaptive.)

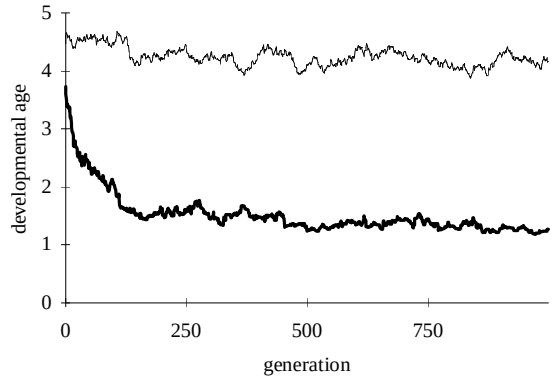

Figure 5: Average epoch of development of functional and nonfunctional units in the individuals of the winning lineage across 1000 generations. Average results of 20 different simulations.

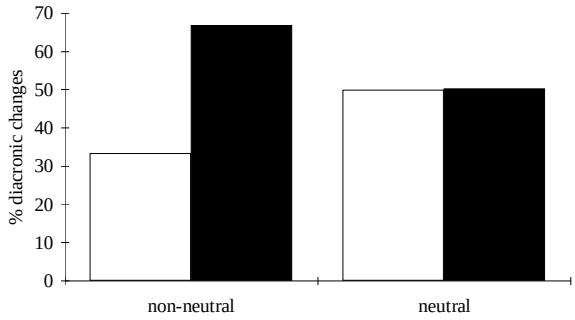

Figure 6: Percentage of posticipations (empty bars) and anticipations (full bars) for non-neutral and neutral mutations in the 1000 individuals of the winning lineage. Average results of 20 different simulations.

These results show that evolution selects genotypes which dictate early maturation of functional neural structure but not of nonfunctional structure. Selective reproduction based on fitness appears to be the force that causes this developmental anticipation of newly evolved structures by favouring the reproduction of Os which are efficient at all epochs of their life and, therefore, as early as possible during lifetime. It is clear that anticipation pressures only apply to functional neurons (i.e. to neurons that contribute to determining the individual's behavior) and, among them, only to neurons that produce an increase in performance. Functional neurons that decrease performance are likely postponed. Thus, in simulations with artificial organisms that develop in time evolution can follow two adaptive routes: (a) it can select individuals with adaptive characters overall (cf. Figure 4), or (b) it can anticipate adaptive characters and posticipate maladaptive ones. The fact that retained adaptive anticipations outnumber posticipations can be interpreted as a consequence of (a) which ensures that among non-neutral characters the adaptive outnumber the maladaptive ones.

Other interesting questions that can be posed are the following: Are evolutionary novelties retained independently from the time they appear during ontogeny or are novelties which appear at a certain phase of development more like to be retained? And have mutations the same probability to be retained independently from the time in which they affect development?

In order to answer these questions we measured the average epoch of appearence in the individuals of the winning lineage of neurons that become functional for the first time during the evolutionary process. What we found is that novelties may appear both in the very first (1) epoch of life and in the very last epoch (8) with an average value of 4.4 which is very close to the value that would be obtained with a

uniform distribution across the 8 different epochs (i.e. 4.5). So it appears that novelties may be retained whatever the epoch in development in which they emerge.

Although evolutionary novelties can be retained independently from their epoch of appearence during development, the probability that adaptive mutations are retained depends on the time of appearence (cf. Figure 7). In particular, neurons that appear in the later epochs of life are more likely to receive mutations than neurons that appear in the first epochs of life. Mutations that have their effects early in life are more likely to affect greatly the fitness of the individual than mutations that have their effects later in life. But most mutations have negative rather than beneficial effects. As a consequence, it is very unlikely that individuals that have received mutations with effects early in life will leave offspring.

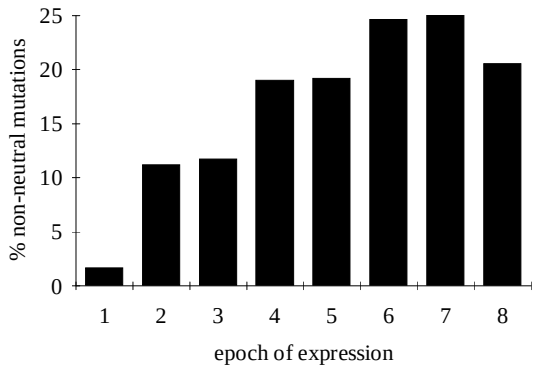

Figure 7: Probability that a neuron of an individual of the winning lineage was subject to a non-neutral mutation for different epochs of appearence of the neuron. Average results of 20 different simulations.

The neurons that mature in the early phases of development in addition to being the most important in determining a fit behavior and the less mutated are also the oldest phylogenetically. As Figure 8 shows, neurons that appear in the first epochs of life are significantly older on average (i.e. have become part of the functional network a greater number of generations before) than neurons that mature in the last part of an individual's lifetime.

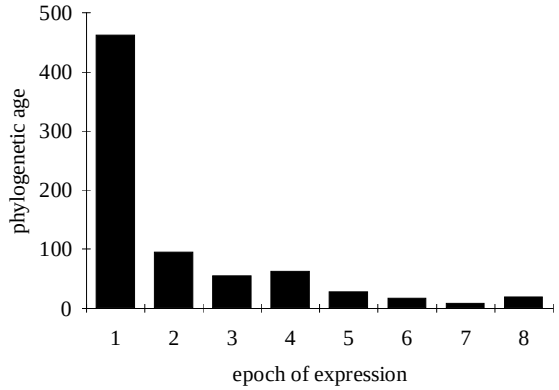

Figure 8: Average number of generations since when neurons that appear at different epochs of life have become functional. Average result of 20 different simulations.

The fact that the anticipation process applies only to functional neural structure combined with the fact that only neurons that are preserved for many generations have the possibility to be anticipated (the

probability for an individual to receive a mutation that anticipates a particolar neuron being very small) ensures that the earlier a neuron or a neural module appears in development the higher the probability that such a neuron or neural model is useful and important in determining a fit behavior and, as a consequence, the higher the probability that it will be preserved in successive generations.

Discussion

We have shown that to develop in time has important consequences. In particular, our results support the claim we made in the introduction that evolution shapes development and development constrains evolution.

Evolution shapes development by anticipating the maturation of adaptive characters but not of neutral ones (and of maladaptive ones). Selective reproduction by favoring individuals that become efficient as soon as possible appears to be the force that causes this developmental anticipation (for a similar explanation see Muller, 1864). This implies that anticipations in non-neutral characters will outnumber posticipations at least if, as seems to be the case in our simulations, evolution is able to select individuals in which adaptive characters outnumber maladaptive ones. This also implies that the fact that individuals develop in time allows evolution to follow two adaptive routes: (a) to select individuals with adaptive characters overall; (b) to anticipate adaptive characters and posticipate maladaptive one.

Development constrains evolution by allowing an easy retention of mutations affecting characters that mature in later stages of development while it remains more difficult to retain mutations with earlier effects. Therefore, although we did not find any tendency for novelties to appear in late developmental stages, it appears that characters that mature late are more likely to be subject to changes and innovations than characters that mature early in development. In other words, development in time forces evolution to be conservative with characters that mature in the first phases of development while it allows evolution to play more freely with characters that mature later in development.

The combination of these two facts: the anticipation of adaptive characters and the tendency to preserve characters that mature in early phases of development, probably explain the other result we found, that is the fact that characters that mature in the first epoch of development are phylogenetically older than characters that mature later in development. This bring us to the final question we discussed in the introduction: Does development (ontogeny) recapitulate evolution (phylogeny)?

The recapitulation theory as was proposed by Haeckel (1966), i.e. the idea that characters that develop in an individual from conception to maturity repeat the evolutionary history of the individual's species, implies two fundamental assumption: terminal addition and anticipation. Terminal addition means that novelties can only be introduced at the end of the developmental process. Anticipation means that the length of an ancestral ontogeny is continuously shortened during the subsequent evolution of the lineage. This assumption means that phylogenetically older characters which first appeared at a certain stage of development often develop at an earlier stage during the ontogeny of individuals of successive generations while more recent phylogenetic characters develop later. This theory is false because it has been empirically shown that acceleration is not general or equal for all characters, that new characters can be introduced at any stage of ontogeny (hence, even earlier than older characters), and that development can be retarded as well as accelerated (cf. Gould, 1977).

However, if one considers, as Gould does, recapitulation not as an absolute law but as a simple tendency that is the result of a more general process, the evolutionary alteration of developmental times and rates to produce acceleration and retardation in the ontogenetic development of specific characters, one can conclude that recapitulation phenomena can be observed if there is some tendency to accelerate development and to preserve characters that have been accelerated. Our results suggest that in individuals that develop in time both requirements may be satisfied to a certain extent and therefore recapitulations phenomena may be observed as we actually do in our simulations. This does not imply that development in time is the only cause of recapitulation. There are in fact at least two other causes that have to do with development per se and that do not concern its temporal character. The first cause has to do with the recursive nature of the cellular duplication process (Belew, 1993; Cangelosi, Parisi, and Nolfi, 1994). Mutations that affect the first cellular duplication phases have in general a huge impact on the resulting phenotype and therefore can be retained very rarely. The second possible cause of recapitulation concerns the fact that characters that mature in early phases of development even if they loose their adaptive function can have an interphene functions, i.e. they can assumes an adaptive

value as an intermediate stage which is necessary to reach subsequent developmental stages (Mayr, 1994). Both facts may force evolution to be conservative with characters that mature in the early phases of development. This may explain why Dellaer and Beer (1994) also observe some recapitulation phenomena with their model in which individuals develop istantaneously.

Finally, we have shown that the anticipation tendency only applies to the functional characters of individuals, i.e. to characters that imply a direct advantage in the reproductive chances of the individual. In fact, one can expect that, as already observed by Mehnert and Massart at the end of the last century (Menhert, 1898; Massart, 1894), characters which must function first develop first.

However, the fact that there is no evolutionary anticipation of neutral characters during development should not be taken as implying that the nonfunctional neural structure - which is generated under genetic control exactly like the functional structure - is irrelevant from the point of view of the development of the individual. Miglino, Nolfi and Parisi (in press) have argued that although the nonfunctional neural structure has no role in determining the behavior of the individual, it can have an important role in the evolution of the population of which the individual is a member. Nonfunctional neural structure which has accumulated evolutionarily because of neutral mutations can become suddenly functional because of a further mutation and perhaps cause a change in fitness (Loomis, 1988). Without the already existing nonfunctional structure the mutation might be unable to cause the change in fitness.

The same reasoning applies at the individual level if the individual develops in the course of its life. At any given epoch (age) the nonfunctional structure which has developed in the previous epochs of the individual's life has no role in determining the behavior of the individual at that epoch. But a further step in development can make (part of) this nonfunctional structure functional and cause a change in the fitness collected by the individual in the next epochs of life. The new developmentally emerging functional structure and the resulting change in fitness might never be realized unless the previous stages in development had not caused the emergence of the critical nonfunctional structure.

In both cases, a static and a dynamic view of the phenomena concerned offer different perspectives on the role of nonfunctional stucture. If an organism is viewed statically as a nonchanging entity, the nonfunctional structure which is generated inside the organism under genetic control appears to be useless. Only at the dynamic level of evolutionary change in the population, the nonfunctional structure can have a role and a meaning, if not for the individual, for the population to which the individual belongs. Furthermore, if the individual itself is viewed dynamically as a developing entity, then the nonfunctional structure has a role and a meaning also for the individual. The behavior that the individual exhibits in a particular epoch of its life can be dependent on the nonfunctional structure which has developed in previous epochs.

In fact, the various results we have described concerning the evolutionary and developmental changes in the functional and in the nonfunctional neural structure allow us to draw a picture in which development tends to have two opposite functions from an evolutionary point of view. On one side, development tends to be conservative and to guarantee a certain amount of fitness to an individual. This is obtained by anticipating the development of functional neural structure and by sheltering this structure from the effects of disrupting mutations. Mutations that negatively affect the initial functional neural structure tend to be quickly eliminated so that there is no trace of these mutations in the best individual of the next generation. A few mutations can affect the changes in functional neural structure that occur during development but the individual is relatively sheltered from the possible negative effects of these mutations because there is little change in functional neural structure after birth and because most of the individual's fitness is collected using the stable functional structure already present at birth. Those mutations that turn out to have a positive effect on fitness are retained and tend to be anticipated in development in the successive generations.

However, after the initial generations most retained mutations are those that affect the nonfunctional neural structure. These mutations may happen to be retained because they are neutral. By affecting the nonfunctional neural structure, they do not change the behavior of the individual and, therefore, its fitness. This aspect of development may represent an important tool to explore novelty at disposal of evolution. Many evolutionary changes in nonfunctional neural structure can be retained because they do not affect fitness. At a certain point in evolution, due to some further mutation, one of these changes is carried over from the nonfunctional to the functional neural structure. If the change is maladaptive it does not affect fitness too much because it happens later in life. If it turns out to be favourable from the

point of view of fitness, the change is retained because it is adaptive and it may be anticipated in development later in evolution. Hence, development appears to be a flexible tool in the hands of evolution with both a conservative role and a role to explore evolutionary innovation.

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