Evolution, Morphogenesis, Genes and Automatons: A Contemplation of Artificial Biological Structures cmpe59e Spring 2021 (covid-19 term)

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1 Abstract

Evolving artificial genomes in artificial environments seems like a very promising technique in artificial intelligence research and it may deepen our understanding of the dynamics of evolution and the structure living beings. It has some successes but compared to the Mother Nature, it fails miserably. Many successes and failures in diverse branches from the theory of computation to evolutionary developmental biology are discussed to contemplate about why. Very little novel approach is presented but an extensive research is done.

2 Introduction

Richard Dawkins programmed a computer program to evolve artificial morphologies for teaching purposes. John Von Neumann has proposed a class of complex systems that he has built to study self-replication. Alan Turing came up with a mathematical model for morphogenesis. What all of these have common is that they try to imitate the products of billions of years of evolution. We will discuss them and more by starting from cellular automata.

3 Cellular Automaton

Cellular automatons are a versatile tool for studying many different branches ranging from biology to the theory of computation first proposed by the famous mathematician John Von Neumann[10]. Von Neumann originally introduced the concept to treat self-reproduction with mathematical rigor[10]. One of the most basic and perhaps the famous cellular automaton is known by the name "Game of Life", introduced by John Conway. Game of life happens on a 2 dimensional infinite grid of squares. Every cell is either alive or it is dead and the time is discrete. At each step every cell becomes dead or alive according to the following deterministic rules:

- If the cell is dead and it has exactly 3 alive neighbours, it becomes alive.
- If the cell is dead but it does not have exactly 3 alive neighbours then it remains dead.
- If the cell is alive and it has exactly 0 or exactly 1 alive neighbours, it dies.

- If the cell is alive but it has more than 3 alive neighbours it dies.
- If the cell is alive and it has exactly 2 or 3 alive neighbours then it remains alive.

Neighbour in this sense means the 8 squares in the immediate surroundings. They are also known as Moore neighbourhoods.

It has shown that the game of life is Turing-Complete. This means that one is able to build a digital computer in game of life, capable of universal computation[9]. What this means is that one can build a digital computer that can simulate any other digital computer. If one is able to deduce the behaviour of a system capable of universal computation then they can also find the answers to computational questions. Indeed, they should "give" the question to the digital computer built in the system and then use their tools to find out how the system will evolve. Since computational problems can be very hard, one also expects that finding the behaviour of these systems capable of universal computation to be also hard. Game of life is a mostly unpredictable and a complex system reminiscent of the complexity of the nature.

4 Morphogenesis

Morphogenesis is the genesis of biological form. While a zygote is becoming an organism distinct forms appear. Organs like heart and intestines appear in mammals and wings emerge in insects like drosophila. Some research is done about some chemicals called morphogens which some researchers think is responsible of the communication of differentiating cells[13]. Actually there is a mathematical model of morphogens which we will discuss in the following section.

5 Reaction-Diffusion Models

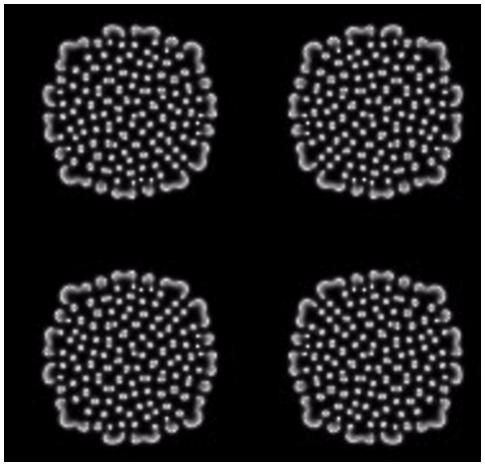
The father of the theory of computation and a mathematician, Alan Turing, has proposed a mathematical model for morphogenesis in 1951[13]. In this model there are some number of morphogens. These molecules diffuse in space and they are chemically active. These dynamics result in very interesting shapes which are reminiscent of biological images.

5.0.1 Gray-Scott Model

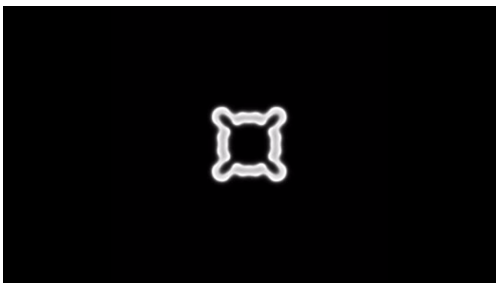
Gray-Scott model[7] is a particular simple reaction diffusion model in which there are only 3 morphogens A, B and C. These morphogens live in a plane and they diffuse according to their diffusibilities. There are only two reactions:

- $A + 2B \rightarrow 3B$
- $B \rightarrow C$

The concentration of C is not of interest. The rate of the first reaction is proportional to $[A][B]^2$ and the second is proportional to $[B]$ where square brackets mean the molar density of the morphogen. Lastly A is added constantly to the system throughout simulation but density of squares of less concentration of A's increase faster than those with high concentration. If the parameters are tuned very complex patterns emerge in this system:

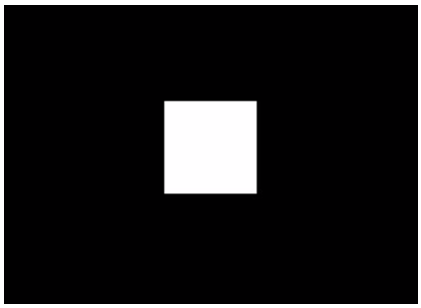


The simulation starts with a square of B's in the middle. The images show only the density of B. White squares are dense with B and black squares do contain B in very small amounts. The insides of this square quickly die out since B's become C and the first reaction cannot replenish the diminishing numbers of B due to absence of A's. However those B's at the boundary keep existing due to the diffusion of A's to the square of B's. Since corners get the highest amount of diffused A they multiply fast and start to diverge:

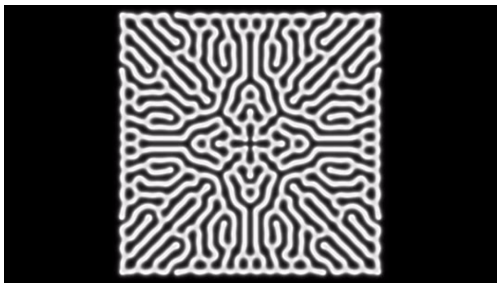


The reader is advised to see the article "Complex patterns in a simple system"[7] if they are interested in other emergent structures of different parameters. The author has tried a slightly different model and have observed the following:

As the simulation continues sharp boundaries tend to "poke out". Pockets of B do not merge since they share the diffusion of the surrounding A's and they cannot sustain the bridge. The gap is their "food source":



Since there is a constant supply of $A's$ coming from outside, zones with no B supply A's to their surroundings. B's follow these "feed zones" until they reach the border. The simulation does not halt completely and one is able to observe small movements after the emergence of the global pattern:



It is important to note that these structures change drastically as one plays with the parameters or the initial densities of molecules.

5.0.2 Relation to Biology and Morphogenesis

These patterns are similar to some biological patterns like the spots on hyenas. In fact, there is strong (according to the author) evidence for the applicability of reaction-diffusion model in biological patterns that occur in nature[4].

6 The Function of The Genes

Kumar and Bentley describe the cells as follows:

"... , cells are fundamentally protein-processing machines, sensing protein signals, being controlled by proteins and outputting new proteins for other cells to sense."[5]

Many critical functions in the cells are carried out by proteins. Proteins seems to be the by far the most important group of molecules for the cell function. Genes code for proteins. Some molecules bind to the gene to read the blueprint of the protein and they produce molecules called mRNA's which makes ribosomes synthesize the protein. Other regions on the same gene are for the regulation of that reaction. Some proteins called transcription factors bind to these regions and adjust the rate of protein synthesis of that gene. This transcription mechanisms allow cells to behave very differently for different levels of proteins. It seems like the belief that genes code for static descriptions of organisms (like the eye colour) is wrong[11] and the genes are pleiotropic. Genes code for dynamical recipes that give rise to the organism. The genes of organisms of different castes in eusocial insects are the same but they obviously gave rise to different phenotypes. Smith et. al. argue that very small changes in nutrition or hormones may be responsible for the expression of a different set of genes that give rise to a very different developmental process of the young[12].

7 Evolution of Evolvability

Even though the computational power available to the mankind has increased to an unimaginable extent, we still cannot replicate the evolution seen in the nature properly. Simply implementing random mutation and selection is not enough. Wagner and Altenberg provide a simple example to answer why. They say that Friedberg(1959) has attempted to evolve programs by random mutation to the letters and selection[14]. It doesn't work (no sur-

prise) because random change to some letters generally yields a code which cannot be even compiled! Wagner et. al. argue that the problem with this method is a bad genotype to phenotype map. Small mutations to the genome yield radical and deleterious changes to the phenotype.

We know that evolution works in the inhabitants of The Earth. Therefore a question arises: How did the genes around us has gained their good genotype to phenotype maps? Is it also an evolvable feature or is it intrinsic to the way biology works? Many argue that it is indeed evolvable and this concept is known as the evolution of evolvability[1][14][2]. Some argue that it is a frail concept[6] but there are some strong evidence for the existence of such dynamics even in very basic models[2].

8 Conclusion

The attempts at imitating biology in computer simulations is discussed. The reasons for the failure of artificial evolution to create the diversity that has been observed in nature has been discussed. The role of genes in cells seems to differ much from the role of the genes in genetic programs[11]. This difference is best explained by the concepts Simon has proposed: State descriptions and process descriptions[11]. The genetic programs usually use state descriptions: The full grown individual is fully determined by it's genes. However, in biology, genes only determine the cell behaviour and the full grown individual is determined both by its genes but by also the environment during its development[11]. Artificial genes coding for the dynamical processes may be the key to evolvable artificial organisms.

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