

# 6th MEW at ALife XV

Morphogenetic Engineering Workshop, at the Artificial Life Conference 2016

Monday, July 4, 2016 Cancun International Convention Center, Mexico

Overview - *Program* - Organizers - Past Editions References - Call for Abstracts - Topics of Interest - Registration

# **Overview**

Traditional engineered products are generally made of a number of unique, heterogeneous components assembled in complicated but precise ways, and are intended to work deterministically following specifications given by their designers. By contrast, self-organization in natural complex systems (physical, biological, ecological, social) often emerges from the repetition of agents obeying identical rules under stochastic dynamics. These systems produce relatively regular patterns (spots, stripes, waves, trails, clusters, hubs, etc.) that can be characterized by a small number of statistical variables. They are random and/or shaped by boundary conditions, but do not exhibit an intrinsic architecture like engineered products do.

Two salient exceptions, however, strikingly demonstrate the possibility of combining pure self-organization and elaborate architectures: biological development (the self-assembly of myriads of cells into the body plans and appendages of organisms) and insect constructions (the stigmergic collaboration of colonies of social insects toward large and complicated nests). These structures are composed of segments and parts arranged in very specific ways that resemble the products of human inventiveness. Yet, they entirely self-assemble in a decentralized fashion, under the control of genetic or behavioral rules stored in every agent.

How do these collectives (cells or insects) achieve such impressive morphogenetic tasks so reliably? Can we export their precise self-formation capabilities to engineered systems? What are principles and best practices for the design and engineering of such morphogenetic systems?

# Program

The workshop will take place on **Monday, July 4 morning**, in room **Tulum 2** of the <u>Cancun International Convention</u> <u>Center</u>:

# Part I: Insects, bacteria, chemicals

- 9:00-9:55, Opening keynote: Complex systems engineering: multi-scale collective construction in artificial insects Seth Bullock
- 9:55-10:20
   Emergence of bacteria talk and morphology of group behavior Nesrine Ouannes, N. Djedi, Y. Duthen & H. Luga
- 10:20-10:45
   Coupling self-assembling materials with digital designs to grow adaptive structures Adam Blaney, J. Alexander, N. Dunn, D. Richards, R. Doursat, A. Rennie & J. Anwar
- 10:45-11:15 Coffee break

# Part II: Gene regulatory networks

- 11:15-11:40
   RBN-based morphogenetic systems with spring-mass-damper kinetics Hyobin Kim & H. Sayama
- 11:40-12:05
   Intelligent cell using on-line GRN policy enzyme <u>Rima Hiouani</u>, N. Ouannes, N. Djedi, Y. Duthen & S. Cussat-Blanc
- 12:05-12:30
   Evolving genetic regulatory networks for online neurogenesis Dennis Wilson, S. Cussat-Blanc & H. Luga
- 12:30-13:00, Closing guest talk: Spiral autowaves as minimal, distributed gait controllers for soft-robots Michal Joachimczak, R. Kaur, R. Suzuki & T. Arita
- 13:00 Lunch

# **Organizers**

- René Doursat, Manchester Metropolitan University, UK
- Hiroki Sayama, Binghamton University, NY, US

# **Past Editions**

This workshop is the 6th Morphogenetic Engineering Workshop or Special Session (MEW) of its kind. It follows:

- the 1st MEW (2009), which was held at the Complex Systems Institute, Paris (ISC-PIF) on June 19, 2009
- the 2nd MEW (2010), which was held as a special session of the 7th International Conference on Swarm Intelligence (ANTS 2010) in Brussels, on September 10, 2010
- the 3rd MEW (2011), which was held as a satellite workshop of the 11th European Conference on Artificial Life (ECAL'11) in Paris, on August 12, 2011
- the 4th MEW (2014), which was held as a satellite workshop of the 14th International Conference on Artificial Life (Alife XIV) in New York, on July 31, 2014
- the 5th MEW (2015), which was held as a special session of the 13th European Conference on Artificial Life (ECAL'15) in York, on July 20, 2015

# References

- Doursat, R., Sayama, H. & Michel, O. (2013) A review of morphogenetic engineering. "Frontiers of Natural Computing" (FNC 2012) Special Issue. M. Lones, A. Tyrrell, S. Stepney & L. Caves, eds. Natural Computing 12(2): 517-535 [19 pages]. PAPER
- Doursat, R., Sayama, H. & Michel, O., eds. (2012) Morphogenetic Engineering: Toward Programmable Complex Systems. "Understanding Complex Systems" Series, Springer-Verlag, ISBN 978-3-642-33901-1 [452 pages]. OVERVIEW

# Call for Abstracts (closed)

This workshop aims to promote and expand a recent field of research called "Morphogenetic Engineering", which explores the artificial design and implementation of autonomous systems capable of developing

**complex**, **heterogeneous morphologies**. Particular emphasis is set on the programmability and computing abilities of self-organization, properties that are often underappreciated in complex systems science—while, conversely, the benefits of self-organization are often underappreciated in engineering methodologies.

Authors are invited to submit an abstract (up to 2 pages) on their research, or on a review and discussion about any aspect of Morphogenetic Engineering. It should be prepared following the ALife XV paper format. Contributions may be original or already published (please specify when submitting and add proper bibliographical references, if applicable). Accepted abstracts will be compiled into the Workshop Proceedings and will be published online on the Workshop website for free downloads.

Please submit your abstract in PDF by email to both organizers:

- Rene Doursat: r.doursat mmu.ac.uk
- Hiroki Sayama: sayama binghamton.edu

Important Dates:

- Deadline for abstract submission: April 30, 2016 May 7, 2016
- Notification of acceptance: May 15, 2016
- Camera-ready abstract due: May 31, 2016
- Date of workshop: July 4, 2016

The workshop will last about 3.5 hours and the total number of speakers is limited to 6. Submissions will be reviewed based on their relevance to the workshop, clarity, and overall quality. Whether submitting or simply attending, please register via the online ALife XV conference registration system.

# **Topics of Interest**

- New principles of morphogenesis in artificial systems
- Bio-inspiration from plant vs. animal development
- Programmability of self-organizing morphogenetic systems
- Indirect, decentralized control of morphogenetic systems
- Sensitivity to environmental/boundary conditions vs. endogenous drive
- Evolvability, by variations and selection, of morphogenetic systems
- · Links with evolutionary computation, artificial embryogeny, "evo-devo" approaches
- Swarm-based approaches to morphogenetic systems
- Design techniques for morphogenetic engineering
- · Causalities between micro and macro properties of morphogenetic systems
- Physical implementations
- Applications to real-world problems (swarm robots, synthetic biologie, complex networks, etc.)
- Philosophical questions about morphogenetic engineering

# Registration

Registration should be made through the ALife XV website.

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# Complex systems engineering: multi-scale collective construction in artificial insects

Seth Bullock

Bristol Centre for Complexity Sciences (BCCS), Department of Computer Science, University of Bristol, UK

In many insect species, colonies of individuals collaborate to construct impressive homes. These structures can exhibit functional organisation at many scales despite the limited cognitive capacity of the individual builders. Understanding how these feats of collective construction are achieved could unlock a new design paradigm for human architecture. Here, a series of simulation studies of collective construction in populations of idealised artificial insects is presented. Particular consideration is given to the problem of simultaneously achieving both fine-grained spatial structure and large-scale spatial organisation by combining distal and proximal behavioural mechanisms in the form of pheromone-mediated behaviour and stigmergic building rules.

# Emergence of bacteria talk and morphology of group behavior

Nesrine Ouannes<sup>1</sup>, NourEdinne Djedi<sup>1</sup>, Yves Duthen<sup>2</sup> and Hervé Luga<sup>2</sup>

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#### Abstract

In this paper, we present a molecular network that bacteria use to talk to each other (i.e. communicate), this network calculates the dynamics of some primary components of the quorum sensing (QS) system that will develop a model with more working compounds. This QS system is acting when bacteria are growing. This action gives the bacteria new conditions to grow as response to form shapes or collective motions.

#### Introduction

Bacteria are unicellular cells that move by their flagella, their reproduction is very fast compared to other living creatures in the nature. Bacteria grow by a chemotaxis process; one of the first behaviors that existed in the history of life on earth.

One more important behavior that has been recently discovered (1970) [1] is the quorum sensing (QS); mechanism of communication between bacteria cells.

In this paper we are focusing on studying this communication process by molecular signaling. Bacteria use this way by molecular exchange in order to monitor and respond to changes in bacterial density and to regulate their collective behavior. Signals, sensors and signaling pathways in a QS process are simulated in this paper as a simplification of the biological reality.

In this model, the bacteria cells grow exponentially according to our previous model of simulated bacteria chemotaxis [2,3], and communicate together according to the proposed algorithm of quorum sensing [4], with a molecular network. This network will facilitate the estimation of the number of bacteria present in a specific region (exp. nutrient presence, detection problem or geometry follow).

The quorum sensing process plays a major role in: (a) colonial behavior of bacterial populations (or multicellular behavior emergence); (b) the control of cell population density; (c) changes in behavior that are triggered by the molecules when a quorum is present; these molecules migrate from one cell to another cell, that is close in proximity;

### Quorum sensing: the way to talk

Natural and artificial systems use the quorum sensing to perform a lot of tasks such preventing QS by therapies, initiating biofilm formation, as a regulator of bioluminescence [5], or obtaining new mechanisms of antibiotic-resistance or virulence pathway. The QS process is a complex network of protein and genes, that forms the control channels, often controls biological phenomena. Many of these pathways have their advantage of environmental processes and molecular signals to regulate cellular activities. However, it is not always clear to determine how biological systems are able to support the propagation of a specific signal over a sufficiently wide dynamic range within the cell [6].

#### The Quorum Sensing signaling pathway

The QS equips individual bacteria with the ability to detect and measure local cell density, and respond to this signal by coordinating their group behaviors.

The regulation of gene expression depending on the cell density of system involves a signal to be produced, distributed and collected by the bacteria.

In addition to this signal (A), the QS system consists of: an inducer synthase (LuxI); a receptor (R) which can bind the inducer molecules and an R-inducer complex. The concentration of the different components over time will be considered as will their rates of changes, as described in the following equations (ODE-models proposed in [7, 8]).

$d[A]/dt = A_0 + (k_a + [C]/k_A[C]) k_{2A}[A] k_1[R][A] + k_2[RA]$	(1)
$d[R]/dt = R_0 + (k_r + [C]/k_R[C]) k_{2R}[A] - k_1[R][A] + k_2[RA].$	(2)
$d[RA]/dt=k_1[R][A] k_2[RA] 2k4[RA]2 + 2k5[C] \dots$	(3)
$d[C]/dt = k4[RA]^2 k5[C]$	(4)
<b>DA</b> is the <b>D</b> induces complex. C is the dimensional complex. A	

RA is the R-inducer complex, C is the dimerized complex.  $A_0$  and  $R_0$  account for the basal level of A and R, respectively.

The first term of Eq.(1) describes the basal level transcription, the second term captures the positive feedback loop regulated by the dimerized complex C, the third and forth terms describe the inducer (A) concentration changes caused by the binding and unbinding reactions of molecules (A) and receptor (R), respectively. Eq.(2) and Eq.(4) describe the binding reaction of molecules (A) and receptor (R), as well as dimerization process of the binding product [RA]. The principle of the QS system modeled in the above equations is: **1.** First, the community members produce auto-inducers that are the signaling molecules. At a low cell density (LCD), the inducer diffuses throughout the environment at a constant rate.

**2.** At high cell density (HCD), the cumulative production of molecule (A) leads to a high local concentration, allowing detection and response.

**3.** The detection mechanism in the bacteria causes the up regulation of the genes that produce (A) molecules. This

creates a positive feedback loop, which increases the level of inducer (A) in the environment.

**4.** The inducer (A) binds and actives R, the concentration of the active R (the complex) further dimerizes and actives the downstream gene, resulting in a coordinated behaviors [7, 8].

### **Proposed algorithm**

Two phases are considered in our model to simulate the QS:

```
Step 1: The chemotaxis network, which guarantees the nutrient foraging behavior [2].
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**Step 2**: A Proposed algorithm that ensures communication between these same bacteria that forage nutrients by chemotaxis [3], with a molecular network of QS.

The following algorithm of QS summarizes the application of (A) molecules that are released into the environment, tested and then re-transformed in these bacterial cells that form groups and behave like a multicellular organism.

```
Inputs: Nutrients concentration, cell density;
Outputs: Tumble Frequency, synchronized behavior;
Initialization();
For (each time (t = t + dt)) do
For (each cell) do
If (LCD) then
QS Network();// just A and R are calculated
Chemotaxis Network ();// tumble frequency
```

```
Next cell position(x(t+dt), y(t+dt);
Metabolism ();
Division ();
else // Quorum reached
QS Network();// 4 equations are calculated
Increased production of the (A) signal and R;
Activation of A-R complex;// gene expression
```

Increase in growth rate and metabolic rate;

# **Results: Obtained morphologies**

In this paper we develop a model of bacteria quorum sensing, enabling us to capture population level patterns. This communication process considers two types of dynamics: cell population and diffusible signal (regulated with a positive feedback loop), all the parameters of the simulation are those used in our previous paper [3], except the parameters of the QS network that are adapted from [8]. First, the bacteria growth according to the chemotaxis process, with a constant rate of inducer's production, once the bacteria density threshold is reached, the feedback loop starts working (all the bacteria components are calculated), the concentration of the complex (A-R) will allow gene expression, resulting in a duplication of the activities and properties of a lot of bacteria in a defined region (or geometry). This mechanism obtained here is like a fitness function that leads a population in an area of specific solution, but our QS model got it spontaneously. Figure 1 shows two regions obtained were bacteria detect a quorum and form shapes.

# **Conclusion and future work**

In this paper we propose an algorithm of quorum sensing coupled with an ODE-model, in order to simulate bacteria communication. Experiments of this type of communication between bacteria are intended to understand better the natural phenomena and can provide insights to learn new methods and propose software solutions to resolve some natural problems with different means that we cannot have in nature. The presented results demonstrate that bacteria are still able to evolve through mutation and adapt according to this type of communication and as results the shape and behavior of the total population is changed. We can extend our research on quorum sensing by analyzing some social behaviors.

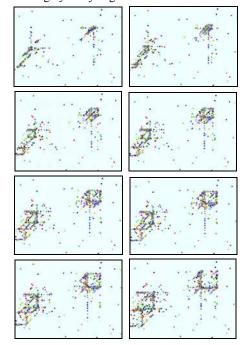


Figure 1: Morphology obtained by bacteria groups.

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# Coupling self-assembling materials with digital designs to grow adaptive structures

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### Abstract

There is a discrepancy between digital design simulations and the physical structures they produce. While current fabrication technologies and materials used to create artefacts lack the flexible and adaptive qualities present within the digital models, this is not the case in biological structures. The latter continually adapt their shape and material compositions to suit imposed environmental demands, maximise available resources and have the ability to self-heal, a process particularly evident in bone remodeling [1]. In order to instill these qualities into manufactured structures we propose a fabrication system that incorporates self-assembling / self-organising materials and design simulations. The resulting objects would have the ability to tune and adapt their material properties (location, type, composition, volume, rate, shape) and offer radically new opportunities for design and manufacturing. Firstly the paper highlights major benefits of fabricating adaptive structures from self-assembling/self-organising materials. Then it describes ongoing research that uses self-assembling materials (crystal growth) to fabricate adaptable structures by inducing turbulence electrically.

### Introduction

Today, in most design and engineering fields, a linear design and fabrication process predominates which produces physical structures that cannot physically adapt, this due to the inert materials that fabricate them [2,3,4]. This means they are unable to deal with changing or unforeseen demands, which in turn leads to significant waste (i.e. financial, material, and pollution) in construction. We propose a fabrication system (Figure 1), which creates adaptive structures out of selfassembling / self-organising materials (e.g. crystal growth). The main benefits of these adaptive self-assembling / selforganising materials / structures are: (i) Longevity as fluctuating and multiple demands can be accounted for; (ii) Reduction of material waste as resources can be redistributed within the structure / system over its lifetime (self-healing [5]): (iii) *Increasing complexity* as material properties can be governed through external stimulus / environmental

manipulations (pH, temperature, voltage). For example, these primitive self-organising materials can be manipulated – or architected – indirectly using electrolysis and digital controllers (Figure 2-4). This in turn may lead to more complex structures being created [6,7], which can physically *respond* and *adapt*. (iv) *Scalable fabrication processes* as the structures are based on the material scale (molecules, chemicals, proteins), this also enables adaption across all scales of the structures (material – global shape changes) leading to emergent results [8]. As the fabrication process is based on its constituent parts which self-assemble it resembles principles of *morphogenetic engineering* [9], which creates highly robust systems/structures due to their decentralized properties/ agency.

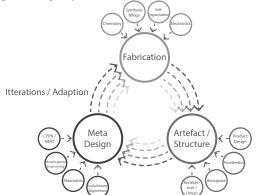


Figure 1. Self-assembling / self-organising materials can enable a discourse between design, fabrication and structures as induced environmental manipulations can govern material properties [10]. Logics from *digital design* simulations [11,12,13,14] can be embedded within the structures as they can govern the environmental manipulations. More complex self-assembling / self-organising *fabrication* platforms highlight the potentials to create radically new opportunities for design and fabrication [15,16,17,18]. *Structures* generated on material properties [19,20,21] and multiple demands have produced highly integrated solutions [22,23] with functionally graded properties [24,25]. The material properties of structures could be continually tuned if self-organising / self-assembling materials were employed to fabricate them.

### **Proof of concept**

In order to develop adaptive tunable materials / structures we use the mineral accretion [26,27] process (electrolysis of seawater) to demonstrate a proof-of-concept. The mineral accretion process is used because it is: (i) robust, low cost (in the laboratory) and initially easy to setup (ii): it enables the self-assembly of composite / multi-materials (limestone and/or brucite) [28] on cathode scaffolds: (iii) material properties (type/composition, location, volume, porosity) can be tuned based on environmental manipulations (voltage, pH) [29] and cathode properties. Material properties of grown structures deposited on various cathode typologies have been analysed [10] (Figure 2) and illustrate that different materials and properties (composition, growth rate, surface textures) can be created from an abundant base material/solution (natural seawater) by manipulating the environmental conditions (i.e. electrical current) [10]. The composition of the base solution may also be altered to produce varying material property build-up. Manipulating environmental conditions produces turbulence, which can be used to tune and adapt material properties. The resultant structures have the capacity to adapt across scales (material to global structural shape) as imposed demands fluctuate (live loading, solar shading).

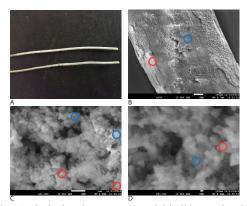


Figure 2 – A. Cathode wire to test material build-up using SEM. B - SEM revealed effects of the cathodes purity as varying amounts of brucite and limestone crystals depending on analysis locations. C, D - Brucite highlighted with blue circles compared to the limestone highlighted with red circles. The rosette shaped crystals are Magnesium Hydroxide, the needle like crystals are calcium carbonate [29]. 100g of marine salts were dissolved in 3L of water, the cathodes were 100 x 1mm diameter steel wire and a 50 x 5mm steel rod was used for an anode.

The cathode type shown in figure 2 had very limited control over what material properties were deposited over the whole of the cathode, this is due to the fact voltage could not be supplied to or varied at a specific location. In order to govern material properties when using the mineral accretion process a distributed network of cathode elements was created, this allows control over voltage location, voltage amount and time each element is supplied with a set or varying voltage (Figure 3 - 4). As this fabrication process is basically material aggregation, which can occur in a 3D volume, this enables structures to be created as a 3D volume.



Figure 3 – A basic heart shape was draw on a distributed cathode network to determine the correseponding wires to be connected and disconnected after various time intervals, this was done manually. The right hand side of the heart was connected and supplied with 3 volts first, once 27 hours had passed all the wires were disconnected and the process repeated for the left hand side at 4.7 volts.



Figure 4 – Resultant heart shape growth establishes that a distributed cathode network can be used to control material properties (volume, location, composition, surface texture). 300g of marine salts were dissolved in 6.5L of water, the cathodes are 1mm copper wire, the anodes are two carbon rods of radius 6.3mm and length 75.0mm.

### **Discussion and Future work**

Future work will combine the distributed network cathode typologies with digital design simulations. The design simulations will be used to govern self-assembling material properties by inducing environmental manipulations through hardware. The adaptive material properties can also be incorporated back into the design simulations resulting in them becoming "an active generator of design and an adaptive agent [30]," which may further enhance adaptive and emergent properties. The materials of the mineral accretion process (limestone and brucite) do not hold any information; they are a mass of distributed molecules/particles within a solution. By incorporating design simulations into the system to induce environmental manipulations, design logics and information can be instilled within the materials and resultant structures, this coupling may give the material agency and develop a fabrication system that resembles principles of morphogenetic engineering.

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# **RBN-based Morphogenetic Systems with Spring-Mass-Damper Kinetics**

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#### Abstract

Non-trivial morphologies of gene regulatory network (GRN)based morphogenetic systems have been actively studied in artificial life and morphogenetic engineering research. Here we present a morphogenetic system that uses Kauffman's NK random Boolean network (RBN) as a GRN and spring-massdamper kinetics for cellular movement. This system can show diverse spatial patterns in 2D space. We show that a spring constant plays an important role in cellular clustering, and biases in cellular movement influences overall morphology.

In the fields of artificial life and morphogenetic engineering, the emergence of non-trivial cellular patterns of GRN-based morphogenetic systems has been an interesting topic [1,2,3]. Here, we add spring-mass-damper kinetics to GRN-based morphogenetic systems to show self-organization of spatial patterns during the developmental process.

We use our NK random Boolean network (RBN) model [4] as a control basis of this kinetic morphogenetic system. The model represents an aggregation of cells, where each cell has an identical RBN as a GRN which consists of 10 nodes. A cell has four key cellular functions: proliferation, apoptosis, differentiation, and quiescence. The performance of cellular functions depends on the number of attractors of a GRN. This is because the cell fates are assigned to attractors. If there is only a single attractor, the cellular function of proliferation is assigned to the attractor. If there are two attractors, proliferation and apoptosis are randomly assigned to those attractors. Similarly, if there are three, proliferation, apoptosis, and differentiation are randomly assigned to those attractors. If there are four or more attractors, proliferation, apoptosis, and differentiation are randomly assigned to three attractors and quiescence is assigned to the rest of the attractors. Fig. 1(a) is a schematic diagram that shows four cell fates randomly assigned in a GRN which has five attractors.

That the cell fate of each cell continues to change means that jumping from one attractor to another may occur in every time step. Perturbations in internal gene expression caused by cell-cell interactions can cause switching of cell fates. Specifically, cells interact with one another through the transport of signal molecules between the environment and cells. The transport occurs through diffusion by the concentration difference of signal molecules. If the concentration of a signal molecule is beyond a certain threshold, it controls the expression of assigned genes. After that, its concentration decreases below the threshold.

Our morphogenetic model starts with one seed cell. The change of concentrations of signal molecules by diffusion leads to the change of gene expression, which eventually converges to one attractor. If the converged attractor is proliferation, the cell is divided into two at the next time step. Each of the two cells inherits half of signal molecules of the mother cell. If the converged attractor is apoptosis, the cell dies. Once the cell becomes dead, it remains in the space as an inactive (empty) object for the rest of the simulation, so that we can keep track of how apoptosis influences morphological development. If the converged attractor is differentiation, the cell is simply regarded as differentiated. Lastly, if the converged attractor is quiescence, the cell does not perform any cellular function of proliferation, apoptosis, or differentiation, but is put on the inactive status (but not dead). In this model, cells staying in the cellular states except for apoptosis can remain capable of switching cell fates as long as the values of gene expression are changed by perturbations due to cell-cell interactions.

To produce non-trivial morphological patterns in a 2D space, we introduced spring-mass-damper kinetics following Doursat's approach [5]. Each cell with radius r has a position P=(x,y) in Cartesian coordinate system. Edges connecting cell centers are modeled as springs with spring constant k and equilibrium length l. For viscous resistance, dampers with damping coefficient c are included. Thus, the equation of movements of a cell is as follows:

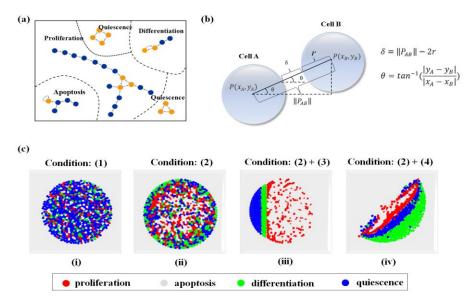
$$m\dot{P_{AB}} = -k\left(1 - \frac{l}{\|P_{AB}\|}\right)P_{AB} - c\dot{P_{AB}}$$

Here, we neglect the effect of inertia. Then, the equation for a position update is the following at each time step  $\Delta t = 1$ :

$$\Delta P_A = -\Delta P_B = \frac{\Delta P_{AB}}{2} = \frac{-k}{2c} (1 - \frac{l}{\|P_{AB}\|}) P_{AB}$$
  
where,

 $||P_{AB}|| = ||P_A - P_B|| = \sqrt{(x_A - x_B)^2 + (y_A - y_B)^2}$ 

 $P_{AB} = P_A - P_B = (\delta cos\theta, \delta sin\theta)$ 



**Figure 1:** (a) Schematic diagram of randomly assigned three cell fates in a GRN which has five attractors. Each node represents a cell's dynamical state. Orange nodes are attractors. (b) Schematic diagram to represent distance and angle between two cells. (c) Spatial patterns of GRN-based morphogenetic systems with various parameter conditions.

Fig. 1(b) visually shows these mathematical quantities.

Using the above position updating rule, we simulated our RBN-based morphogenetic systems. We set the upper limit of the number of proliferation to 1,000 in order to prevent overgrowth. We also assumed that, when proliferation occurs, a daughter cell is produced at distance R from its mother cell (before spring-mass-damper kinetics are applied). As a result, physical interactions such as pushing, adhesion, and movements among cells happen in this system.

We investigated spatial patterns in 2D space by varying parameters for the spring-mass-damper kinetics. Specifically, the following four parameter conditions are tested: (1) Cells of all cell fates have the same spring constant. (2) Different spring constants are applied to cells with different cell fates. (3) Biases are applied to the final cell positions. (4) Flexible biases are applied to the cell positions only when a certain condition is satisfied. Fig. 1(c) shows various morphologies generated under the four parameter conditions. Pattern (i) is generated in parameter condition (1). Because the same spring constant is applied, cells with different cellular states are well mixed. Pattern (ii) is obtained in parameter condition (2). Because the spring constants are heterogeneous and specific to cell fates, cells with the same cellular functions tend to cluster, simply due to their kinetic compatibility (similar to [6]). We set the values of spring constants in the following order: apoptosis < proliferation < quiescence < differentiation. Thus, cells are clustered roughly along this order from inside to outside. Pattern (iii) is obtained under parameter condition (2) + (3). Because different spring constants are applied, cells are segmented according to their cell fates. In addition, by giving biases to the final cell positions of x, we obtain the effect of self-organizing cell sorting. Finally, pattern (iv) is formed under parameter condition (2) + (4). We applied flexible biases to the cell positions only under a certain condition. Specifically, when the position of a cell is smaller than those of its neighbor and adhesion between them occurs,

then small random values are added to the cell position (in both x and y). Patterns (iii) and (iv) show completely different shapes with patterns (i) and (ii) due to the effect of biases in cellular movement.

Based on these results, we found that a spring constant has an important role in clustering of cells, while biases in cellular movement makes a contribution to overall morphology. This work is still at its preliminary stage, and we plan to conduct more systematic simulations to explore more diverse, more non-trivial morphogenetic dynamics. Furthermore, we plan to design and evaluate RBN-based mechanisms to achieve specific morphogenetic patterns in a more controlled manner.

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# Intelligent Cell using On-Line GRN Policy Enzyme

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#### Abstract

Nowadays, morphogenetic engineering (ME) [1] is inspired by biological systems (embryogenesis) to export their self-formation capabilities to engineered autonomous systems. As cells are intelligent by nature, researchers of ME are trying to recreate this intelligence in artificial systems, so that these cells know how and when to act in order to accomplish a specific function (e.g. Build an organism).

# Introduction

Morphogenesis in computer science has become a topic of interest in natural systems. In their attempts to understand embryogenesis, biologists realized that the morphogens, the genes [2], chemical gradients [3], and mechanical forces between cells [4], affect the cell development, without knowing how these mechanisms work exactly.

Thus, since we still ignore the exact mechanisms of this process, that leads us to ask whether there are other mechanisms or not. Especially because scientists have not discovered everything about a cell due to its complexity.

The automatic design of complex systems, such as artificial multicellular organisms, is a considerable challenge; especially if this organism exhibits the same 'strong' characteristics of living organisms. The self-organization process results in architecture without any central planning or external drive. To achieve these properties of embryogenesis, many developmental models have been designed to simulate the growth of virtual multicellular organisms, with different levels of biological realism. In [5], the author argues that the functional requirements impose the shape development. Aligned with this idea, we propose a model of cell development that uses the function requirement at hierarchical levels to develop an organism, without using morphogens as an external driver in the environment [6] or a cell driver [7].

Due to the embryogenesis complexity, it would be very difficult for the cell to undertake the whole process of creating an organism, where some specific cells use a function to create a specific tissue not the whole organism. subsequently, we propose an "Autopoietic Multilevel System" to subdivide the complex shape (by function requirement).

The individuals in our model are different from a level to another. For example, cells in the  $1^{st}$  level, tissues in the  $2^{nd}$ , and so forth, until arriving at the final shape which is the organism. In this paper, we demonstrate how our GRN works in one of the levels, starting by stimulating cells that are controlled by an "on-line GRN Policy Enzyme".

# **On-line GRN Policy Enzyme**

To achieve an embryogenesis process, we must know "how the tissues and organs of the developing embryo take their miraculous forms?". We use Artificial GRN with new additions in order to obtain an on-line learning process with memory concept. Our proposed model is defined as follows:

- 1. A reinforcement learning realized by an Artificial GRN;
- 2. A policy enzyme that accelerates the activation of one of the potential actions using the memory system;
- 3. A memory system that is represented by two matrices: "best action" and "worst action";
- 4. An evaluation module with two evaluation functions; the first one against local interest, and the second against the global interest, initialized at the birth of the individual.

Morphogens in our model are obtained from neighbors as a signal of the individual, the energy wich is fundamental to achieve an action, and proteins from the upper GRN that determine the function requirements. These morphogens can activate more than one action at the same time in the first layer. With a GRN, an individual can activate the right action in the right place using reinforcement learning. Two regulation layers are used here; the first one assures that all the necessary morphogens of the environment are present for each action. If the gene action of the first layer is active, they pass to the second layer (Policy enzyme), where it chooses between the activated actions to select the right action according to the memory. These two layers regulate the output of the GRN.

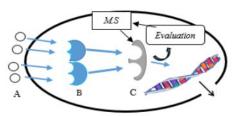


Figure 1. GRN Policy Enzyme Architecture. (A) Represents morphogens in the environment, (B) the first layer or regulation (gene action). (C) The second layer of regulation (Policy enzyme), M.S the Memory system.

# **Primary Results**

Embryogenesis is a complex process, in which achieving some degree of morphology's complexity by a selforganization system is complicated. This task can be accomplished by using the performance of functional requirements. This function is used here to regulate the response of the GRN. Initially, just one cell is present with an unregulated *GRN Policy Enzyme*, and a function that conducts the morphogenesis process to a target shape.

Generally, a function requirement is subdivided, so each level will have a specific sub-function (eg. Obtaining a tissue shape). To achieve this function, we will demonstrate the capability of:

- Each cell to know which action to execute, where and when.
- The auto-organization process to generate a corresponding shape without external control.

To demonstrate how our GRN works, we put a stem cell in our simulation grid and we give it the task of creating an organ. Without limit geographies in the environment, morphogens in the environment have a partial effect in the cell development, but they are not defined as rules in the cell's genome. In the first generation, stem cell has no information about its memory system. Policy enzyme chooses randomly between actions that have the possibility to be executed. Each cell has its own function that it should accomplish. For example, the cell level tries to respond to the tissue function.

The figure 2 presents the obtained results of the GRN learning process with the Policy Enzyme. In the first generation (figure 2.a) just the first tissue (yellow) of the development process appears, this latter uses all the energy present in the environment. If its own function is achieved, then the cell level responds to a tissue level, which in turn will create a second sub function to cell level as creating another tissue.

Here, we observe that the second tissue doesn't appear because the energy was all used by the first tissue.

In (figure 2.b), cells in the first tissue try to use their memory differently from the previous generation; the GRN\_Policy enzyme allows them to use less energy (actions chosen from the memory matrices). This will generate a competition between tissues; each tissue works on its own interest to create its shape. So, other tissues can also appear and the function to obtain an upper level (an organ) will have enough individuals (tissues) to be achieved. (Figure 2.c). When the development process generates the specific organ, each cell generates its right path, according to its function. *Policy enzyme* is regulated for each cell, in time and space. Cell modifies its *Policy enzyme* whenever the morphogens or the functions to be achieved are modified.

# **Conclusion and future work**

In this paper, we propose a new model of an organism's development, where we try to inspire from biological systems by using a *self-organized* system that uses a new kind of

GRN: "On-line GRN Policy Enzyme", coupled with a hierarchical level of development using the concept of autopoietic system.

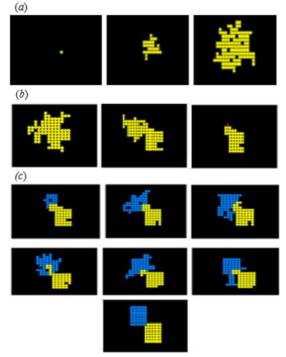


Figure 2. Development of a target shape from a single cell.

In our next work, we will detail this hierarchical development, and this by presenting how individual (cells, tissues, organs) can create itself basing on the autopoiesis concept.

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# Evolving Genetic Regulatory Networks for Online Neurogenesis

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#### Abstract

We evolve a Genetic Regulatory Network (GRN) in a three dimensional morphogen gradient environment to determine the topology of the neurons in a Spiking Neural Network (SNN). A genetic algorithm is used to optimize the GRN, selecting individuals based on the performance of the SNN grown by the GRN. Performance is measured on two tasks: visual discrimination and robotic foraging. Early results show potential for this method as both an indirect encoding and online regulator of neural networks.

# Introduction

Artificial neurogenesis has been a fascination of the artificial life community long before the advent of modern neural networks. Gruau (1994) evolved grammars which encoded ANNs capable of controlling six-legged robots. Fleischer and Barr (1994) used controlled morphogen emissions to guide the growth of complex neural network morphologies. More recently, Kowaliw et al. (2014) covered a number of approaches, such as Wróbel et al. (2012), which showed that GRNs can encode SNNs that exhibit desirable and realistic spiking patterns.

In this abstract, we provide an overview of an artificial neurogenesis model designed to both growth and continuously modify an online SNN. The design and evaluation of this model is undergoing; we present a broad overview in 2 and preliminary results in 3.

### Neural model

Neurons are modeled as single points p in a three dimensional cube with an orientation o. They emit morphogens and can move in the space following mophogen gradients, aligning both their orientation and movement with the chosen gradient and bound in all dimensions. Morphogens are distributed radially from each neuron's emission,  $e_m$  and are normalized globally, the concentration of morphogen m at neuron i computed as

$$c_{m,i} = \frac{1}{\max(c_i)} \sum_j \frac{e_{m,j}}{\|p_j - p_i\|}$$
(1)

At each time step, morphogen concentrations are recalculated based on the emissions from each neuron, which in turn are determined by the neural controller. At each interval of time steps  $t_{action}$ , neurons take one of the following actions: movement, division, quiescence, and, for hidden neurons only, apoptosis. The neuron's  $\beta$  parameter, used to determine its firing influence, is also updated at each  $t_{action}$ .

#### **Neuron controller: GRN**

The neuron controller in this model is a genetic regulatory network. In nature, a GRN is a network of proteins that controls the behavior of cell. An explanation of GRNs, and specifically the model used in this work, can be found in Cussat-Blanc and Banzhaf (2015).

The inputs to the GRN are an important consideration in the design of the model. The following inputs have been chosen not only to enable the growth of interdependent neurons, but also to give each individual neuron information about its contribution to the performance of the network. The inputs are the neuron position p, the morphogen concentrations at p, neurotransmitter concentration, firing decay, problem reward, and the neural influence coefficient  $\beta$ .

The reward is specific to the problem domain, as explained in 3. Firing decay is an exponential timer reset every time a spike is fired in the neuron, to give the controller input as to how recently the neuron fired. There are three position inputs, one for each dimension, and five morphogen inputs, one for each morphogen emitted by other hidden neurons, and two for the distinct input and output morphogens.

The controller's outputs then determine the state and actions of each neuron. The outputs consist of actions: movement along a morphogen gradient, division, apoptosis, and quiescence. Also output are the morphogen emissions  $e_m$ ,  $\theta_e$ ,  $\delta_\beta$ , and  $\theta_{\delta_\beta}$ . Morphogens are emitted from the neuron and  $\beta$  is updating according to

$$e_m = \frac{e_m - \theta_e}{e_m + \theta_e}$$
 and  $\beta_{t+1} = \beta_t + \frac{\delta_\beta - \theta_{\delta_\beta}}{\delta_\beta + \theta_{\delta_\beta}}$  (2)

An action is chosen as the maximum output from the action outputs. If one of the 5 movement actions are chosen, the

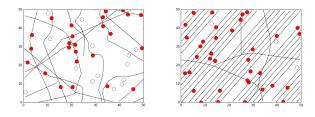


Figure 1: The path and food consumption of the robot. Consumed food is filled; ignored food is empty. The first path displays a use of sensors, the other displays an ignorance of them.

neuron orients based on the chosen morphogen gradient and moves along it.

### **Firing model**

The neurons are then translated into a leaky integrate and fire spiking neural network, with position in the cube determining neural connectivity. The weight from a neuron i to another neuron j is

$$w_{i,j} = \frac{1}{\exp\left(\beta_i \frac{o_i \cdot (p_j - p_i)}{\|o_i\|}\right) - 1}$$
(3)

where the distance between the neurons is the projection of their distance vector onto the orientation of i,  $o_i$ . Input neurons are oriented along the z axis, facing the output neurons directly. Neurons fire if the neurotransmitter concentration reaches the threshold vt, at which point the neurotransmitter concentration is set to resting potential, vr; otherwise they leak a percentage  $\alpha$  of their neurotransmitter at each timestep.

# **Evaluation**

GRNs were evolved in a genetic algorithm based on Cussat-Blanc et al. (2015), using a robotic foraging task for fitness. In this task, a two wheeled robot is placed in a torus environment populated by food particles. The robot has 8 sensors on its front half. The robot turns and moves by firing neurons on the left and right side of the output plane, accelerating the left and right wheel, respectively. Eating prolongs the robot's life, which is decreased at each time step. The problem ends when the robot runs out of life, and fitness is awarded based on the amount of food consumed. The reward input provided to the GRN is the current life of the robot. A growing ANN forages over five instances of a map generated at the beginning of each evolutionary generation and the worst fitness is chosen, which motivates improvement and stability of the ANN.

Using an evolved GRN and the resultant ANN, the robot displays a tendency to move towards food detected by select sensors; the topology may underutilize some inputs, evident as food not being approached from certain angles. Another undesirable evolutionary trait is the efficient blind search methods that attempt to cover the entire map, ignoring food placement. As seen above, a persistent movement strategy though the map can result in food consumption competitive with the strategy of following sensory input.

The model acheieved near perfect fitness on the foraging problem using both sensor following and map coverage strategies. The stability of the network is still under consideration; while the model grows and modifies the network according to the reinforcement problem, as desired, the modifications can also destabalize the network. We are focused on addressing this issue in the continuing development of the model.

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# Spiral autowaves as minimal, distributed gait controllers for soft-robots

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#### Abstract

Inspired by the self-organization of growing embryos and coordinated movement of multicellular assemblies such as the slime mold Dictyostelium, where each cell is controlled by the same controller (a DNA-encoded gene regulatory network), we evolve distributed gait control mechanisms for soft-bodied animats. The animats are made of compressible material, with each body region capable of independent actuation, controlled by a cell at its center. Each animat consists of hundreds of cells uniformly distributed throughout the body, each sharing the same artificial gene regulatory network and aware of the state of their local neighborhood. We found that one of the most common actuation patterns that emerged relied on cells synchronizing their oscillations in order to produce a rotating, spiral wave spanning throughout the body. We found this type of mechanism to emerge for a wide range of animat morphologies as well as in very different types of initial conditions. We investigate how the evolved controllers produce the pattern through local feedbacks and evaluate spiral stability when imperfect, noisy cells are used.

### Introduction

Taking inspiration from distributed control mechanisms observed in nature, such as self-organization of a growing multicellular embryos and movement of multicellular assemblies of certain amoeba known as slime molds (e.g., Dictyostelium), we investigated the possibility of evolving distributed controllers for prespecified morphologies of softbodied robots that would produce gaits in a truly decentralized manner. By dividing animat bodies into hundreds of cells capable of communicating with their neighbors, we were expecting to observe the evolution of some form of autowaves organizing the gaits. Autowaves are a special type of nonlinear waves that are known to occur in active mediums and the main difference between autowaves and classical waves is that propagation of the former occurs at the expense of energy stored in the medium. The energy is used to trigger process into adjacent regions (Roska et al., 1995; Manganaro et al., 1999). Autowaves occur in many biological phenomena, in particular they are essential to multicellular development, but are also central to processes such as propagation in nerve fibers or heart excitation.

The unexpected result of our evolutionary experiments was the predominant type of control mechanism that emerged was based on producing a very specific type of autowave: a rotating spiral known as a spiral autowave. Spiral autowaves are frequently observed in excitable mediums (Ma et al., 2010), and have been observed to emerge in mediums as different as chemical solution of Belousov-Zabotinski reaction, cardiac tissue or neurons of neocortex, though they usually emerge in a chaotic, unpredictable form. In this work, however, we were able to observe how evolution creates controllers that self-organize into spiral autowaves anchored at a specific location of animat's body and produce cyclic, sustainable gaits.

### Methods

We have employed the same approach to simulate softbodied animat locomotion as in our previous studies (Joachimczak et al., 2015), that is animats are two dimensional and are represented as a set of point masses (corresponding to cells) connected with springs. Unlike our earlier work, where we investigated co-evolution of bodies and brains, here we focused solely on the design of distributed controllers only. Hence, we assumed that morphology of an animat is specified at the beginning of an evolutionary run and does not change (other than the elastic changes during locomotion). Animat shapes were specified either as a drawing or a clip-art and then algorithmically triangulated to produce a mesh with a desired number of nodes. Locomotion was possible owing to the local, elastic changes to the body controlled by each cell.

During the evaluation, each cell of an animat is controlled by a copy of the same evolved artificial gene regulatory network (GRN) encoded in the genome. Despite the same controller, cells can differ in their behaviors, due to differences in environmental (input) signals ultimately producing different internal states of the cells. We abstracted the concept of a GRN in the form of a continuous time recurrent neural network (CTRNN), a type of a neural network in which nodes have an activation level that changes in a continuous manner. The GRN topology is then evolved using the NEAT (Stanley and Miikkulainen, 2002) algorithm with fitness function promoting distance achieved by a simulated animat.

We tested a number of minimalistic setups in order to see what kind of distributed gait control mechanisms we can evolve with it. Ultimately, we evolved networks with only a single output determining the current level of contraction or expansion of a body region around a given cell. The main input then represented averaged output state of the cell's neighbors. To facilitate evolution of controllers relying on local communication we disabled the typically used

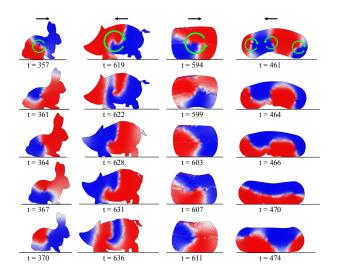


Figure 1: Examples of spiral autowave driven actuation evolved for four different morphologies. Color shows current local actuation signal (red - expansion, blue - contraction). Circular arrow indicates the direction of spiral rotation. Videos of each animat's gait available at: https://goo.gl/ j10UnZ (additional links in the description of the video)

bias signal. This ensured that oscillatory activity cannot start and sustain itself in a cell without receiving signal from the neighbors. We then experimented with different methods of seeding the initial activity by stimulating a few cells or providing maternal gradients. In each case, however, we would remove the seeding signals after a short period of time, so that the actuation of the body had to sustain itself through the propagating autowaves. Finally, to identify the evolved mechanisms of emerging autowave patterns, we compared experiments in which networks are allowed to have recurrent connections with the case where recurrent connections are disallowed.

### **Results**

Having performed multiple evolutionary experiments, we found that evolution, tasked with a problem of evolving distributed, local communication-driven controllers for softbodied animats repeatedly converged on a very simple and creative solution that relies on producing a rotating spiral autowave anchored in the center of the body or even multiple synchronized spirals in case of elongated individuals (Fig. 1). We also found that this simple control mechanism evolves for a wide range of tested animat morphologies and emerges both if a highly localized (two cells) or a global (gradient) seed stimuli are used to initialize the waves of cellular activity. In each case, what starts as seemingly chaotic waves propagating through the body, in a few hundreds of network updates forms a rotating spiral that sustains itself, often indefinitely. As the rotating arm of a spiral sweeps through the bottom of animat's body, it makes the contracting part of the spiral lift part of the body, producing a gait that works both for morphologies that have a flat bottom as

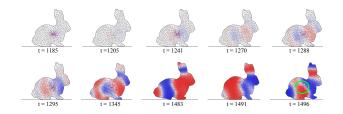


Figure 2: A rotating spiral autowave emerges from cellular activity seeded in two cells at the center of the body. Video available at: https://goo.gl/0klqno

well as for morphologies standing on appendages.

Finally, we have investigated scenarios in which cells are allowed to rely on recurrent feedbacks or forced to rely only on the state of their neighbors and found that while the spiral autowaves emerged in each of the cases, different types of designs had very different robustness to noise. In particular, if we assumed that cells' internal clocks are imperfect, only the experiments in which cells communicate with neighbors were able to produce sustainable spirals.

# Conclusions

While the spiral autowaves are a common phenomenon in many physical and biological systems, we see their unexpected emergence in the context of evolving distributed gait controllers for soft-robots as an example of how artificial evolution can surprise us and suggest entirely new type of design, one that would be otherwise unlikely to be proposed by a human designer.

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