STUDYING COMPLEX ADAPTIVE SYSTEMS USING MOLECULAR CLASSIFIER SYSTEMS

James Decraene, George Mitchell, Barry McMullin
{james.decraene, george.mitchell, mcmullin}@eeng.dcu.ie

ESIGNET — SIXTH FRAMEWORK PROGRAMME — DCU

GOAL OF WORK PRESENTED IN THIS POSTER

A novel approach to study Complex Adaptive System is presented. This work refines John Holland's proposal to allow the study of *Molecular* Complex Adaptive Systems such as Cell Signaling Networks. We present preliminary experiments focusing on the self-replication ability of these complex systems. Counter intuitive results were encountered, which suggest the importance of molecular specificity and necessity of a theoretical framework for the study of Artificial Chemistries.

LIMITATIONS OF HOLLAND'S APPROACH

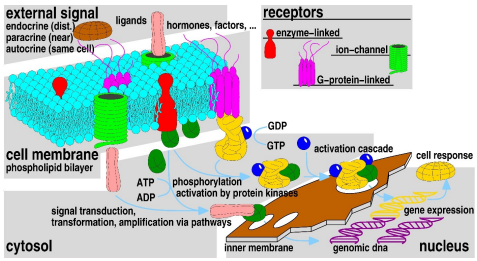
To study **Complex Adaptive Systems**, Holland proposed to employ an agent-based system in which Learning Classifier Systems (LCS) were used to determine the agents behavior and adaptivity. We argue that **LCS are limited for the study of CAS**: the rule-discovery mechanism is pre-specified and may limit the evolvability of CAS. Secondly, LCS distinguish a demarcation between messages and rules, however operations are reflexive in CAS, e.g., in a cell, an agent (a molecule) may both act as a message (substrate) and as a catalyst (rule).

OUR APPROACH

To address these issues, we proposed the **Molecular Classifier Systems** (MCS.b), a string-based Artificial Chemistry based on Holland's broadcast language. In the MCS.b, **no explicit fitness function** or rulediscovery mechanism is specified, moreover **no distinction** is made between *messages* and *rules*.

CELL SIGNALING NETWORKS

In the context of the **ESIGNET** project, we employ the MCS.b to study a subclass of CAS: **Cell Signaling Networks** (CSNs) which are complex biochemical networks responsible for coordinating cellular activities.



J. Decraene, P. Dittrich, T. Hinze, T. Lenser, B. McMullin, G. Mitchell. "A Multidisciplinary Survey of Modeling Techniques for Biochemical Networks". Integrative Post-Genomics Conference, IPG06. 29 November - 1 December 2006, Lyon, France.

MOLECULAR CLASSIFIER SYSTEMS - MCS.b

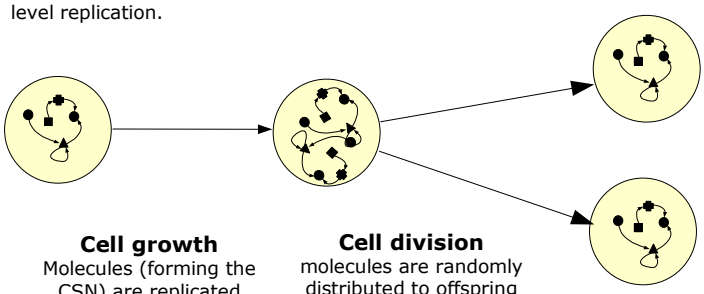
- « Strong Artificial Life » approach
- String based artificial chemistry, loosely based on John Holland

Broadcast Language

- Chemical processes as condition-action rules (IF condition x THEN action y)
- A molecule (string) can function both as substrate (message) and as catalyst (classifier)
- Formally an **Abstract Term Rewriting System**

SELF-REPLICATION IN CELL SIGNALING NETWORKS

As CSNs occur in cells, these networks must **replicate** themselves prior to cell division. So that offspring cells obtain the necessary molecules to be **functional**. Errors may occur during this replication process, e.g., an offspring cell may inherit only a partial CSN. Thus resulting in a potentially defective cell which would lead to a variety of undesired effects (e.g., premature cell death). As a result, the **fitness** of a cell is implicitly represented by the **survival** and **performance** of a cell in achieving cell-level replication.



Cell growth
Molecules (forming the CSN) are replicated

Cell division
molecules are randomly distributed to offspring cells

1 2
3 4

PRELIMINARY EXPERIMENTS ON SELF-REPLICATION

*Closely relates to other studies: Tierra, Alchemy, Alpha Universe, Amoeba, etc, which were implemented **differently** but exhibited **similar** behavior.*

1st experiment: domination of the self-replicators

$A+A+X \rightarrow 3A$

Common intuition: if a self-replicase molecule *A* is present in the reactor (filled with random molecues and no mutation occur), then *A* **should quickly dominate** the reactor.

However our results differ: Fig 1

Why is this happenning?

- In this experiment, replicase *A* was in fact not only a self-replicase but a **universal replicase.**
- A universal replicator is said to have « zero specificity » and would replicate any molecules.
- Therefore a universal replicator does **not possess any advantages** over the other molecules
- So, to dominate the reaction space, a replicator needs to be specific enough to prevent parasitic effects, Fig 2.

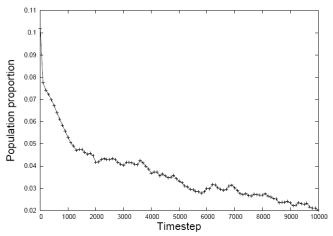


Fig. 1. Relative population growth of replicators SR_0 averaged over 30 simulation runs.

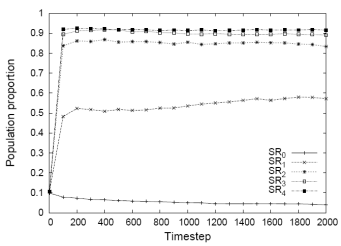


Fig. 2. Population growth of replicators SR_0, SR_1, SR_2, SR_3 and SR_4. Each series is averaged over 30 simulation runs.

So « specificity » matters!

2nd experiment: rise and fall of the fittest

- An ancestor molecule with high specificity was inserted « a la tierra ».
- Mutation may now occur.

Expectetion: Domination of this ancestor over the population and emergence of organizations, such as collectively replicating sets of molecules, as observed in Tierra systems

However, results are again counter intuitive, see Fig 3.

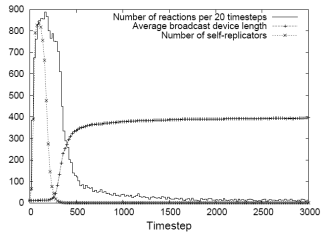


Fig. 3. Effects of molecules length growth upon overall system reactions rates. In this experiment, an ancestor (SR_4 = '#0101 : #0101) is inserted (with initial relative concentration $[SR_4] = 0.1$) in addition to randomly generated molecules. Moreover mutation per molecule and per symbol is turned on.

Why is this happenning?

- Fitter mutants emerged.
- Leading to an increase in molecule length.
- This molecular length growth leads to an **elongation catastrophy**, i.e., system extinction.

CONCLUSION

These simple experiments exhibited **unexpected** results as opposed to those inferred from the literature. We demonstrated that **molecular specificity** plays an important role and may significantly influence the system dynamics. This work highlights the **current deficit** of a theoretical framework for the study of Artificial Chemistries.

AKNOWLEGDMENTS

This work was supported by the **ESIGNET** project (Evolving Cell Signaling Networks in Silico). ESIGNET is a Specific Targeted Research Project funded by the European Commission under the Sixth Framework Programme (contract no. 12789).

Bibliography: **J. Holland**, "Exploring the evolution of complexity in signaling networks," Complexity, vol. 7, no. 2, pp. 34–45, 2002. **J. Holland**, "Studying Complex Adaptive Systems," Journal of Systems Science and Complexity, vol. 19, no. 1, pp. 1–8, 2006. **J.Decraene, G.G. Mitchell, B. McMullin**, "Evolving Artificial Cell Signaling Networks: Perspective and Methods," Advances in Biologically Inspired Information Systems: Models, Methods, and Tools, vol. 69, 2007. **J. Decraene, G. G. Mitchell, B. McMullin, and C. Kelly**, "The holland broadcast language and the modeling of biochemical networks," EuroGP'07. **J. Decraene**, "The Holland Broadcast Language," Artificial Life Lab, RINCE, School of Electronic Engineering, Dublin City University, Tech. Rep. ALL-06-01, 2006. **T. Ray**, "An approach to the synthesis of life," Artificial Life II, vol. 10, pp. 371–408, 1991. **W. Fontana and L. Buss**, "What Would be Conserved if" the Tape were Played Twice"?" Proceedings of the National Academy of Sciences, vol. 91, no. 2, pp. 757–761, 1994. **P. Dittrich, J. Ziegler, and W. Banzhaf**, "Artificial Chemistries-A Review," Artificial Life, vol. 7, no. 3, pp. 225–275, 2001. **B. McMullin, C. Kelly, and D. O'Brien**, "Multi-level selectional stalemate in a simple artificial chemistry." ECAL 2007.