Methods for Open-box Analysis in Artificial Development

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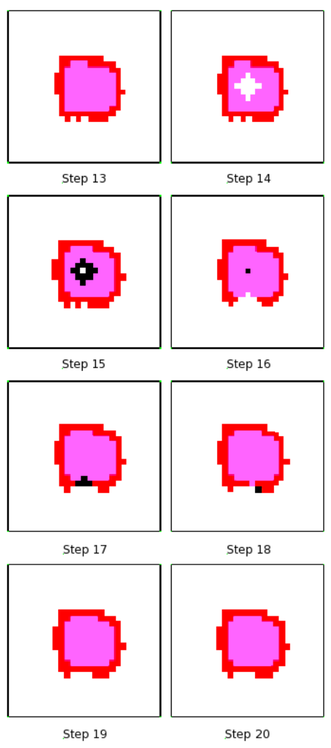
1 An Evolved Self-Repairing Colony



The Task:
- Start with an egg-cell
- Grow a colony of cells to a certain size
- Maintain the size
- Environment randomly kills patches of cells
- Detect damage and self-repair

ADS framework:
- Internal and external protein concentrations
- Genes are *if* <*condition*> *then* <*action*> rules
- Genome is a collection of rules/genes evaluated in sequence
- Variable length representation
- All cells share same genome

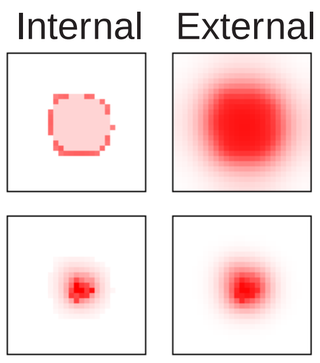
Configuration details:
- 27×27 grid seeded in center with egg
- 4 proteins configured (p0 – p3)
- Evolutionary algorithm: ES(2+16)

2 Investigative Methods

Understanding the evolved self-repair mechanisms requires an open-box analysis in addition to traditional methods.
- Gene activation map
- Environmental features ablation
- Gene suppression
- Chemical concentration monitors
- Modeling the regulatory network
- Subsystem simulation

3 Chemical Concentration Monitors

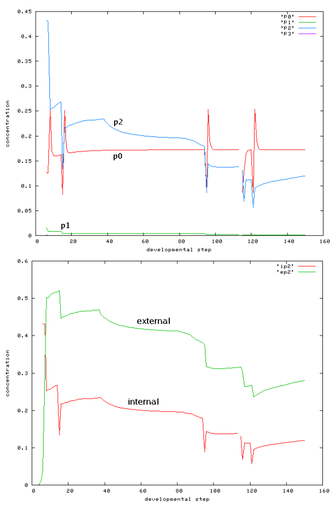
3.1 Overall Protein Concentrations



p0 — Protein p0 establishes a colony border of higher concentration.

p2 — Protein p2 establishes a gradient, both internally and externally.

3.2 Protein Concentrations in a Typical Cell

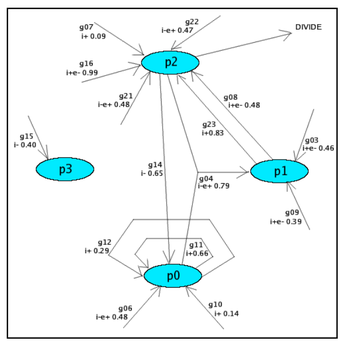


Internally, protein p0 concentration is very highly regulated.

The internal and external protein p2 concentrations move in sync.

4 Modeling the Regulatory Network

Includes genes that fire at least sometimes during a typical cell's stationary regime. Each gene is an arrow. Conditional or unconditional firing is captured by the base of the arrow being tied to proteins or not. The protein affected by a gene designates the head of the arrow.



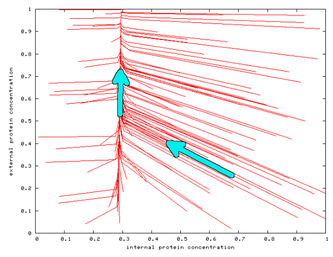
Identifies clusters of genes around each of the proteins.

5 Subsystem Simulation

5.1 Stabilizing Protein p0

Isolating the effects of genes g10, g11, g12 and g14 in order, leads to the following recurrent system:

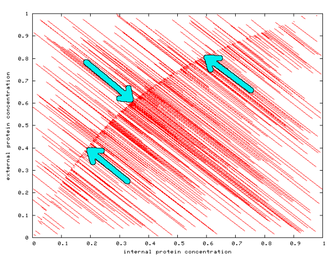
$$\begin{cases} int_{t+1} = 0.274607 + 0.0367324 \times int_t + 0.032411 \times ext_t \times int_t \\ ext_{t+1} = ext_t + 0.46875 \times int_t - 0.46875 \times ext_t \times int_t \end{cases}$$



Iterating the system from 100 random starting points identifies an attractor point. Internal concentration quickly approaches a stable value, followed by a steady increase in external concentration.

5.2 Regulating Protein p2

Isolating the effects of genes g07, g16, g21 and g22 in order, leads to a recurrent system that is a polynomial of degree $int_t^8 \times ext_t^8$.

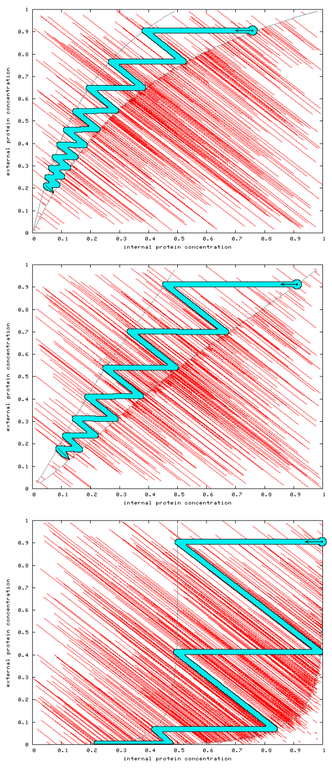


An infinity of attractor points are revealed, laid along a concave curve. Individual trajectories are parallel to the second diagonal, revealing a total conservation of protein concentration.

6 Gene Suppression Pathologies

6.1 Perturbing the Self-Repair Mechanism

When a cell dies it is respawned by one of its neighbors with half the internal concentration of p2. The concavity of the equilibrium curve in the p2 regulation cycle boosts the number of possible death/rebirth cycles.

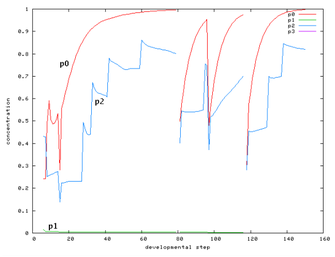


This is what confers the colony the ability to self-repair damage throughout the 150 developmental steps. Beyond that, the ability to self-repair is eventually lost, in a gradual process akin to *natural aging*.

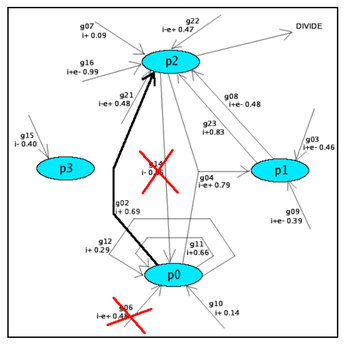
When either g21 or g22 are suppressed, the equilibrium curve loses the concavity; this leads to a reduced number of possible death/rebirth cycles, translating into a *premature aging* pathology.

When both g21 and g22 are suppressed, the equilibrium curve is sharply convex; the number of possible death/rebirth cycles is severely diminished, translating into an *accelerated aging* pathology.

6.2 Explaining Cancer-like Growth



Suppressing g06 or g14 (or both) breaks the stabilizing cycle of p0. Its internal concentration increases and triggers gene g02 firing. This leads to the concentration of p2 to increase too, followed by uncontrolled growth.



A potential remedy seems to be to also suppress any of g10, g11, or g12. Experiments reveal that knocking out g10 is not enough to block the cancerous growth, but blocking either g11 or g12 does.