Emergence of synchronicity in phasic sensitive cellular automata with stochastic topology

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Objective:
phenomenon of synchronicity in a system of oscillating units, understanding of the source of mammalian beating heart

Method:
cellular automata model strongly motivated by physiology of the sinoatrial node – the heart’s first pacemaker

Result:
FRA model in which sustained periodic signal emerges robustly but critically

Conclusion:
Critical properties of FRA system reveal accurately some known physiological facts

Discrete modelling lacks classical mathematics, provides new mathematics
Cellular automata approach to excitable medium

What is the basic idea?

A rule is needed instead of maths

Sinoatrial node

\( A \)

\( \begin{pmatrix} 1 \\ 2 \end{pmatrix} \)

\( F \)

\( \begin{pmatrix} 1 \\ 2 \end{pmatrix} \)

\( F \)

\( \begin{pmatrix} f \\ f \end{pmatrix} \)

\( R \)

\( \begin{pmatrix} 1 \\ 2 \end{pmatrix} \)

\( R \)

\( \begin{pmatrix} r \end{pmatrix} \)

\( \begin{pmatrix} a \end{pmatrix} \)

FRA
Formal definition of a FRA rule:

Let $\varphi(i)$ be an oscillatory phase of a FRA cell at $i$ time step.

Let $\sigma_k \in \{1, 2, \ldots, f, f + 1, \ldots, f + r, f + r + 1, \ldots, f + r + a\}$

$F_{f'}$, $R_{r'}$, $A_{a'}$

$k \in \{1, \ldots, f + r + a\}$

$\varphi(i) \in \{F_{f'}, R_{r'}, A_{a'}\}$ \hspace{1cm} $1 \leq f' \leq f$, $1 \leq r' \leq r$, $1 \leq a' \leq a$

Free evolution of a FRA cell means:

$$\varphi(i + 1) = \begin{cases} 
\sigma_{k+1} & \text{if } k \leq f + r + a \\
1 & \text{if } k > f + r + a 
\end{cases}$$

If a stimulus arrives:

$$\varphi(i + 1) = \begin{cases} 
F_1 & \text{if } \varphi(i) = A_{a'} \\
R_{\text{max}\{1, r'/2\}} & \text{if } \varphi(i) = R_{r'} 
\end{cases}$$
Consider 2 interacting FRA cells:

- \((R_i) \rightarrow (R_{i/2}) \rightarrow \ldots (R_1)\)
- \((F_j) \rightarrow (F_{j+1}) \rightarrow \ldots (F_k) \rightarrow \ldots (R_1)\)
- \((A_i) \rightarrow (F_1) \rightarrow \ldots (F_k) \rightarrow \ldots (R_1)\)

Synchronization as marching cells
Heart tissue in the working heart:

A network of cardiomyocytes interconnected by "gap junctions"
Individual myocytes from the sinoatrial node - the heart natural pacemaker
The nodal myocytes versus the atrial myocyte

Individual myocytes from the sinoatrial node - the heart natural pacemaker

Fig. 3. (A) Immunolabeling of both Cx43 and Cx45 in a large spindle SA node cell (cell double-labeled). Projection image of Cx43 (first image) projection image of Cx45 (second), superimposed projection image of Cx43 and Cx45 (third) and superimposed image of Cx43 and Cx45 at a sin optical plane sectioned at the level close to the top surface (fourth) shown. (B,C) Immunolabeling of Cx40 in spider SA node cells. (D) Immunolabeling Cx40 in a left atrial cell. Projection images shown in B–D. Scale bar, 20 μm.
**FRA network**

- **d:** probability that two neighbouring cells of the Moore neighborhood have established a "gap junction" connection.
- Any free cell is connected to its right neighbor.
- Output cells have extra connections to its horizontal neighbors.
Kuramoto order parameter to measure synchronization between cells:

\[ r(k) = \left| \frac{1}{N} \sum_{n=1}^{N} e^{i\varphi_n(k)} \right| \]

- no synchronization: \( r = 0 \)
- march synchronization: \( r = 1 = e^{i\varphi(k)} \)

Kuramoto order parameter separates order from disorder due to interaction strength

Interaction settings:
- density
- a stimulus:

Experimeent parameters:
- \( d \): probability to establish a connection
- \( F \): how many nghb in Firing to skip Activity
- \( R \): how many nghb in Firing to elongate Refractory

f=10, r=11, a=19
time lengths of automata states
Phase order vs curvature:
- refractory
- firing
- activity

predator-prey idea

reds
eat
greens

reds
stops
blue

Phase order vs curvature:
- activity
- firing
- refractory
low density means high temperature

expanding impulse

collapsing impulse

high density means low temperature
F>2 R>0

Kuramoto order parameter

0.0 0.2 0.4 0.6 0.8 1.0

Hypokalemia:
lack of potassium
1. d = 0.65 - 0.70 density of connections in the canine SAN

2. weak sensitivity of intercallular connections

3. dominance of interactions that lead to shortening of cellular intervals
Thank you