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**Pattern formation and criticality in the developing retina**

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**ABSTRACT**

In the early retina, spontaneous collective network activity emerges as propagating waves, playing a central role in shaping the visual system. Elucidating how the characteristics of such waves depend on biophysical parameters would help us understand the mechanisms that underlie the patterns of spatiotemporal waves formation in the developing retina and their role in shaping the visual system. We have elaborated a set of detailed biophysical equations for a network of retinal cells coupled with excitatory lateral cholinergic connections, close enough to reality to reproduce and predict experimental results. From bifurcation theory, we predict that there exists a regime of parameters for which the network of cells in the developing retina is a critical system. This property is manifested via power law distributions for the waves characteristics (i.e. waves size), meaning that waves statistics could exhibit maximal variability. This critical regime is analytically characterized, predicting the exact form of the critical coupling strength of cells. Away from this regime of parameters, no power-law like distributions are observed. This theoretical result is in agreement with our experimental recordings in perinatal mice, revealing power-laws as well, suggesting that there exists a mechanism setting the retinal cells close to this critical regime.

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**Context & Motivation**

Retinal waves characteristics exhibit a vast variability:

- **i)** Across species
- **ii)** Development
- **iii)** Pharmacology
- **iv)** Spatial Variability

**Patterns vary upon parameters variation**

Having proposed a biophysical model for retinal waves [1], we use our equations to understand the underlying mechanisms of waves apparition and propagation:

**Analytic condition for wave propagation**

Waves propagation analytic condition for a critical threshold of cholinergic coupling

\[
\frac{\partial^2 u}{\partial x^2} + \frac{1}{\tau} \frac{\partial u}{\partial t} = - \frac{1}{\tau} \frac{\partial u}{\partial t} = g_A \left( V_C - V_A \right)
\]

Based on bifurcation theory:

1. We derive analytic forms for a critical waves propagation threshold of coupling strength among cells \(g_A\), and for the waves speed (not shown).
2. We propose a possible mechanism of how power-law distributions could appear near this propagation threshold, where the cell is in fact close to a (saddle-node) bifurcation point. At this point, dynamics are driven mainly by noise fluctuations, leading to maximum variance in the patterns characteristics (e.g. waves size), manifesting power-law like distributions, and therefore indicating possible links to criticality.

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**Conclusions and Perspectives**

- Our model allows us to anticipate how biophysical parameters variations (e.g. conductance) may impact the characteristics of waves.
- We predict that SACs are close to a bifurcation point, leading to explaining the different types of variability of retinal waves as well as proposing encouraging, although still primary links to criticality.
- Further analysis is needed to characterize critical systems, such as studying in detail possible phase transitions, and computing critical exponents on the theoretical side.
- On the experimental side, new and more precise methods should be proposed for the exact characterization of power-law distributions in experimental recordings.
- Extend our phenomenological model in order to identify the possible homostatic mechanism that drives the network to a critical state.
- Explore the role of the indicated criticality in the early retina, possibly related to an optimizing the response sensitivity to multi-scale stimuli upon maturation, enhancing the dynamical range of the early network [Steven’s law].

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**Finding power-laws in experiments**

We performed MEA (256 electrodes) experiments on P5 mice (stage II retinal waves) at Vision Institute, Paris.

- A power-law distribution for the waves size is computed at the regime where the transition occurs in our model (B), matching our experimental data on P5 mice.
- This indicates that maybe the network of SACs is naturally set close to a critical state by a possible homeostatic mechanism, yet to be identified.

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**References**


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