

## Kuramoto model for populations of quadratic integrate-and-fire neurons with chemical and electrical coupling

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Abstract:

The Kuramoto model (KM) is a minimal mathematical model for investigating the emergence of collective oscillations in populations of heterogeneous, self-sustained oscillators, including large-scale neuronal oscillations. Yet, it remains unclear how the parameters of the KM relate to parameters—such as chemical or electrical synaptic strengths—critical for setting up synchronization in biophysically realistic neuronal models.

Here, we derive the Kuramoto model (KM) corresponding to a population of weakly coupled, nearly identical quadratic integrate-and-fire (QIF) neurons with both electrical and chemical coupling. The ratio of chemical to electrical coupling determines the phase lag of the characteristic sine coupling function of the KM and critically determines the synchronization properties of the network. We apply our results to uncover the presence of chimera states in two coupled populations of identical QIF neurons. We find that the presence of both electrical and chemical coupling is a necessary condition for chimera states to exist. Finally, we numerically demonstrate that chimera states gradually disappear as coupling strengths cease to be weak.

Altogether, these results support the use of the KM for modeling studies in computational neuroscience and introduces the powerful mathematical framework of the KM for the analysis of the dynamics of QIF networks.



We used linear models to assess the serial and history dependence of the monkeys' saccadic responses, and their dependence on ketamine. Surprisingly, monkeys did not exhibit attractive, but mostly repulsive serial and history biases when these were combined. Ketamine reduced repulsive serial dependence but increased repulsive history bias. Moreover, we used neural population decoders to predict the stimulus location from prefrontal neural activity and analyzed the relationship between the decoded locations and the responses of the monkeys. Our analyses suggest that different mechanisms underlie serial dependence and history biases in the prefrontal cortex, based on their inverse modulation by systemic NMDAR disruption, and the close correspondence between decoding errors from prefrontal populations and behavioral errors in the task. These results have strong implications for attractor model simulations that implement serial dependence[1] and history effects[4] based on biophysically plausible NMDAR-dependent mechanisms.

## Refs:

1. Stein, H. et al. Reduced serial dependence suggests deficits in synaptic potentiation in anti-NMDAR encephalitis and schizophrenia. Nat. Commun. 11, 4250 (2020).

2. Bansal, S. et al. Qualitatively Different Delay-Dependent Working Memory Distortions in People With Schizophrenia and Healthy Control Participants. Biol. Psychiatry Cogn. Neurosci. Neuroimaging (2023)

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