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Bridging Cellular, Circuit, and Network Mechanisms in Consciousness and Its Disorders: A Computational Framework

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In the computational modeling of the neural correlates of consciousness, many efforts have focused on the whole-brain scale [1]. Although this powerful approach successfully captures global dynamics, it may overlook potentially insightful details at the population and microcircuit levels. An alternative strategy is to concentrate on a limited subset of nodes considered most essential and to represent them with greater biological specificity, embedded in a whole brain network –i.e. a multiscale modeling approach [2]. Moreover, growing evidence points to the contribution of single-cell computations, namely in layer 5 cortical pyramidal neurons, to the maintenance of conscious states [3]. Because these dynamics can be integrated into mesoscale and even macroscale models [4], their incorporation represents a natural and important next step in advancing computational approaches to consciousness.

Here, we present a biophysically detailed cortico-subcortical spiking network model that covers cellular, microcircuit, and brain network levels. The model includes a subset of cortical regions selected for their theoretical significance [5], clinical relevance to the disorders of consciousness (DoC) [6, 7], and availability of experimental recordings. These mainly include nodes of the default mode, frontoparietal, and salience networks. Each cortical region of interest is represented as a column, organized into superficial, middle, and deep layers. Thalamus comprises the intralaminar nuclei and the reticular nucleus. The inclusion of basal ganglia (striatum and globus pallidus internal) further enables the model to test predictions of the fronto-striato-thalamic circuit model for recovery of consciousness [8]. Interareal projections are constrained by layer-specific anatomical structural connectivity. This laminar resolution enables examination of two mechanisms thought to be crucial for maintaining conscious states: the cellular mechanism for coupling inputs arriving at different cortical layers [4], and the interaction of feedforward and feedback streams of cortico-cortical communication [9, 10]. The model was implemented in NetPyNE [11], a tool for modeling large neural circuits with the NEURON simulator.

Although the focus of this work is on modeling pathological impairments of consciousness in DoC, we have preliminarily validated the thalamocortical module against empirical markers of medically induced loss and recovery of consciousness [12, 13], which also helped constrain parameter values. The cortical and thalamocortical components were adapted from prior modeling works on anesthesia [14, 15]. The next step is to extend the validation to the full network to assess its capacity to reproduce pathological states of DoC, by manipulating long-range cortico-cortical and thalamo-cortical pathways to mimic the structural disconnections characteristic of these conditions.

The proposed framework aims to provide a unified mechanistic platform for capturing signatures of conscious states, with particular relevance to pathological conditions. Its biophysical plausibility enables testing hypotheses about pharmacological, invasive, and non-invasive interventions. The model offers a means to assess metrics such as fronto-parietal communication breakdown, frequency-band alterations, changes in structure-function correlation, neural complexity and integration and segregation of information. Ultimately, this work seeks to establish a generative platform that bridges theoretical, experimental, and clinical perspectives on the mechanisms of consciousness and its disorders.

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