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Role of myelin plasticity in oscillations and synchrony of neuronal activity

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Abstract:
The precise timing of the arrival of neural spikes and signals that propagate along the axons is of fundamental importance for the functioning of neural systems. It was argued that achieving such millisecond timing requires an adaptive regulation of the conduction velocity. Myelination potentially provides one such mechanism of nervous system plasticity, since the changes in myelin thickness or nodal structure can effectively modulate the conduction velocity. It has already been shown that myelination plays an important role in the adjustment of timing during the nervous system development, but here we emphasize its importance for the information processing tasks that involve coupling and synchrony among different brain rhythms. In particular, we focus on the activity-dependent myelin plasticity and demonstrate the effects of such adaptive conduction delays on the stability and synchrony of neural oscillators using some known models of coupled oscillators. We introduce an adaptive time delay parameter to a generalized diffusively and linearly coupled oscillator model and a phase-coupled Kuramoto model. Such systems of delay differential equations (DDE) with history-dependent delays can be very difficult to solve even numerically, and we show how solutions can be obtained using existing DDE solvers with state-dependent delays. We demonstrate how a simple local activity-dependent myelination plasticity rule stabilizes a system of coupled oscillators against hypo- and hyper-activity, and also how the adaptive time delays can provide an alternative mechanism for the entrained frequency modulation. These findings suggest that the impairment of activity-dependent myelination and the adjustable time delays may relate to a number of neuropsychiatric disorders (e.g., dyslexia, schizophrenia, epilepsy, obsessive-compulsive disorder, depressive disorder, etc.). For example, schizophrenia is associated with abnormal neural oscillation patterns, slower natural frequency of oscillations as well as hypo-activity in the pre-frontal cortex, thalamo-cortical dysrhythmia, and abnormalities in the white matter, but all these indicators could be a result of a failed mechanism of myelin plasticity. Our results indicate that the myelin plasticity is not only a possible form of nervous system plasticity, but may be necessary to maintain normal and stable oscillatory activity.
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