Beta- and gamma-band oscillatory dynamics in the cortico-basal ganglia and thalamocortical circuits during and after repeated periodic movements.

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Introduction

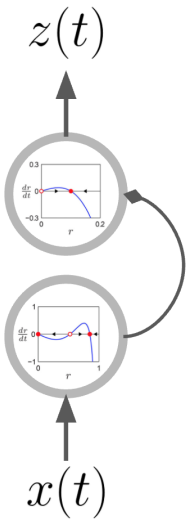
Coordinated neural computations underlie voluntary movements. Patterns of activity at the basal ganglia (BG) are modulated by dopaminergic neurons upon the initiation of movement at the motor cortex (MC). In Parkinson's disease (PD) dopaminergic activity at the BG is low, impairing voluntary movements. Brain oscillations arise from the coordinated computations in BG and MC, showing that excessive activity in the beta-band ($\sim$ 20Hz) is associated with anti-kinesis in PD. Contrastingly, high-gamma ($\sim$ 80Hz) bursts are associated with the initiation of movement and subsequent desynchronization of beta-band. In PD, dopamine replacement therapy and deep brain stimulation with high-gamma restore normal oscillatory dynamics. Recent computational work focuses on understanding directional connectivity between nuclei within the BG. However, these models have not yet explained gamma- and beta-band dynamics in relation to periodic movements. We implement a model using non-linear oscillatory neural networks (ONNs) to explain the coupled activity between MC BG during periodic periodic movement. The building block of our ONNs is a canonical neural oscillator with dynamics at a Hopf Bifurcation, allowing for an abrupt and qualitative change in behavior upon stimulation. Our model consists of two neural oscillators, one at the high-gamma-band in a double-limit cycle, and one at the beta-band in a limit cycle. An MC impulse drives the high-gamma oscillator, whose activity is fed as inhibitory input to the beta-band oscillator. An emergent memory state in our model allows neural substrates to continue carrying out periodic computations after prolonged periodic initiation of movement. Our model reproduces oscillatory dynamics in the beta- and high-gamma bands observed when healthy individuals carry out periodic motion, such as finger tapping. If the coupling between oscillators in our model is reduced, PD activity emerges, capturing dopaminergic malfunction in which weakened inhibitory connections give rise to excessive beta-band activity in the BG. The significance of our model relies on its ability to capture oscillatory brain activity triggered by periodic stimulation as observed in both healthy individuals and patients affected by Parkinsonism.

Methods

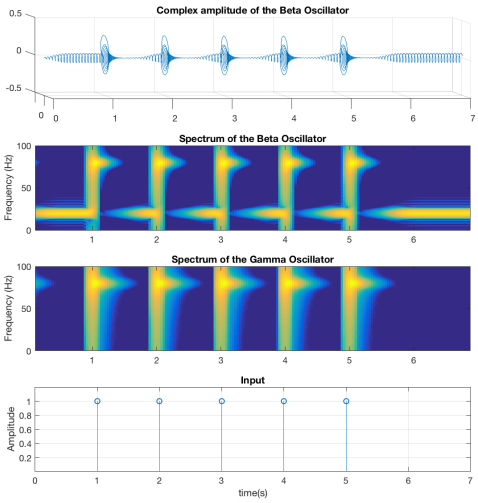
We build a model using nonlinear neural oscillators described by the ordinary differential equation:

$$\dot{z} = z(\alpha + i\omega + \beta_1|z_i|^2 + \frac{\epsilon\beta_2|z_i|^4}{1-\epsilon|z_i|^2}) + x(t) \tag{1}$$

where $x(t)$ is the input to the oscillator, $z(t)$ is a complex state variable containing amplitude and phase information ($z = re^{i\phi}$), ω is the natural frequency of oscillation, and the parameters that determine the dynamical properties of the oscillator are α, β_1, β_2, and ϵ [1][2]. In our model, the high-gamma oscillator receives MC impulses as input. The parameters of the high-gamma



(a) Architecture of the ONN



(b) Summary of Results

oscillator are $\alpha = -15$, $\beta_1 = 1$, $\beta_2 = -1$, and $\epsilon = 1$, which make it be a double-limit cycle oscillator capable of learning upon stimulation. The beta oscillator receives inhibitory input from the gamma oscillator and has parameters $\alpha = 8$, $\beta_1 = -2000$, $\beta_2 = 0$, and $\epsilon = 0$, which pose it at a limit cycle that spontaneously oscillates. Figure 1a The architecture of our model.

Results

Figure 1b shows the behavior of our model through a brief period of isochronous MC impulses. The top two rows show the complex amplitude response and the spectogram, respectively, of the beta oscillator. The oscillator changes in amplitude upon receiving inhibitory input from the high-gamma oscillator, whose spectrogram is shown in the third row of the figure. The MC impulses, input to the high-gamma oscillator, are shown in the bottom row of the figure.

Discussion

Our results are consistent with evidence from electrophysiology literature indicating that isochronous motion, as well as isochronous tactile and auditory stimulation, trigger a high-frequency burst, and a subsequent event related desynchronization of beta-band oscillation. Our model is also able to memorize the period of isochronous MC impulses after an extended interval of stimulation. Consistent with electrophysiological data, this memory is observed in the activity of the gamma-band. This feature of our model is attainable due to the gamma oscillator locking at the higher-state of a double limit cycle.

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