

Self-organization of connectivity in spiking neural networks with balanced excitation and inhibition

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Atypical neural activity and structural network changes have been detected in the brains of autism spectrum disorder (ASD) [1]. It has been hypothesized that an imbalance in the activity of excitatory and inhibitory neurons causes the pathological changes in autistic brains, denoted by the E/I balance hypothesis [2]. In this study, we investigate the effect of E/I balance on the self-organization of network connectivity and neural activity using a model approach. Our model follows the Izhikevich spiking neuron model [3], and consists of three neuron groups, each composed of 800 excitatory neurons and N_i inhibitory neurons (Fig.1A). Each excitatory neuron had 100 intraconnections with randomly selected neurons in the same neuron group, and 42 inter-connections with randomly selected neurons in its neighboring neuron group. These synaptic weights were modified using the Spike-timing-dependent plasticity rule [3]. Each inhibitory neuron had 100 intraconnections with randomly selected excitatory neurons in the same neuron group, but they did not have any interconnections nor plasticity. We simulated the model with different N_i and inhibitory synaptic weights (W_i) in one neuron group (neuron group 1 in Fig.1A) to change the degree of inhibition in the neuron group. N_i and W_i in the other groups (2 and 3 in Fig.1A) were set to 200 and -5, respectively. The simulation results show greater intraconnections in all neuron groups when N_i and W_i were lower values, i.e., the E/I ratio increased compared to those in the typical E/I ratio (Fig.1B). Moreover, asymmetric interconnections between neuron groups emerged where the synaptic weights from neuron groups 2 to 1 were higher than when the connectivity was in the opposite direction (Fig.1C), where the E/I ratio was found to increase. Furthermore, the phase coherence between the average potentials of neuron groups was found to be weak with an increased E/I ratio (Fig.1D). These results indicate that the disruption of the E/I balance, especially the weak inhibitory, induces excessive local connections and asymmetric intergroup connections. Therefore, the synchronization between neuron groups decreases, i.e., there is a weak long-range functional connectivity. These results suggest that the E/I imbalance might cause strong local anatomical connectivity and weak long-range functional connectivity in the brains of ASD [1].

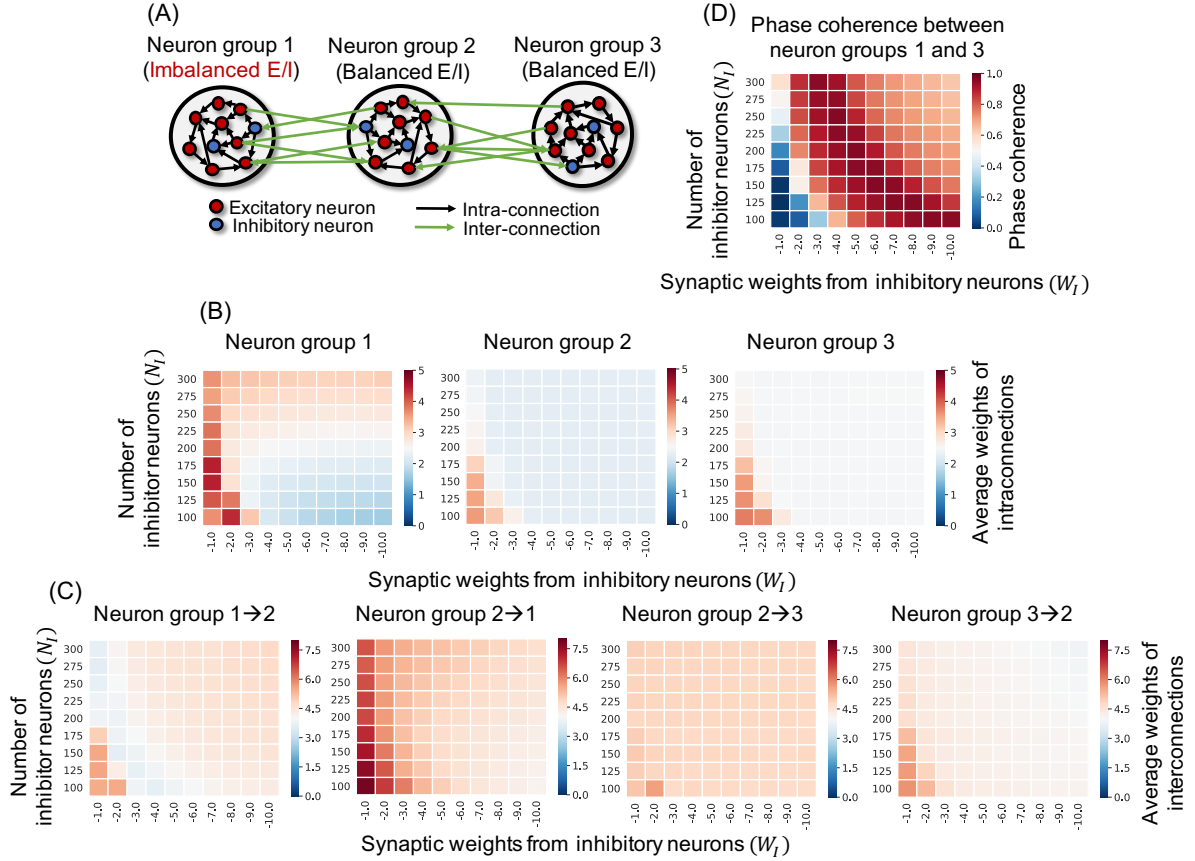


Fig. 1. Model overview and results. (A) A model consisting of neuron groups. Neuron group 1 has controlled inhibitory neurons and does not directly connect with neuron group 3. (B) Average weights of intraconnections in each neuron group after self-organization. (C) Average weights of interconnections among neuron groups after self-organization. (D) Phase coherence between neuron groups 1 and 3.

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