Local Over-connectivity Reduces the Complexity of Neural Activity: Toward a Constructive Understanding of Brain Networks in Patients with Autism Spectrum Disorder

Koki Ichinose, Jihoon Park, Yuji Kawai, Junichi Suzuki and Minoru Asada
Graduate School of Engineering
Osaka University
Osaka, Japan
{koki.ichinose, jihoon.park, kawai, suzuki.junichi, asada}@ams.eng.osaka-u.ac.jp

Hiroki Mori
Graduate School of
Fundamental Science and Engineering
Waseda University
Tokyo, Japan
mori@idr.iassci.waseda.ac.jp

***Abstract*—The human brain has a huge number of neurons connected to each other, forming a multitude of networks; notably, such connectivity typically exhibits a small-world structure. However, the brains of persons with autism spectrum disorder (ASD) reportedly have what has been termed as "local over-connectivity." The neural activity of the ASD brain is also atypical; resting-state EEG signals in the ASD brain have lower complexity and enhanced power at low and high frequency oscillations. In this study, we used a small-world network model based on the model proposed by Watts and Strogatz to investigate the relationship between the degree of local over-connectivity and neural activity. We controlled the degree of local over-connectivity in the model according to the parameters laid out by Watts and Strogatz. We assessed connectivity using graph-theoretical approaches, and analyzed the complexity and frequency spectrum of the activity. We found that an ASD-like network with local over-connectivity (i.e., a high clustering coefficient and a high degree of centrality) would have excessively high power in the high frequency band, and less complexity than that of a network without local over-connectivity. This result supports the idea that local over-connectivity could underlie the characteristic brain electrical activity in persons with ASD.**

I. Introduction

Autism spectrum disorder (ASD) is characterized by difficulties in social interaction and communication. Many researchers have reportdd that the brains of patients with ASD have atypical connectivity and neural activity [1]–[3]. However, the relationship between atypical connectivity and atypical neural activity in ASD is not well understood.

In terms of connectivity, the brains of persons with ASD reportedly have increased short range or local connectivity, particularly in the frontal cortex [1]. From this observation, the so called "local over-connectivity" hypothesis is derived [1]. Given the importance of the frontal cortex in cognitive functions such as language, emotion, and decision-making, local over-connectivity could disturb communication between the frontal cortex and other regions of the brain, thus producing the characteristic disorder of social communication seen in persons with ASD.

In the brain, neural networks have characteristic styles of connectivity, or topologies. One of the most important is the small-world network [4]. Small-world networks have many triplet connections, in which three nodes connect to each other to form a cluster. Note that this graph-theoretical definition of a cluster differs from that used in the term cluster analysis, a common method of data-mining. One feature of small-world networks is that clusters connect to the larger network through only a few connections. The path length, or shortest distance between two nodes, is typically short in small-world networks. The large number of clusters and short path lengths allow small-world networks to store and integrate information. In this study, we constructed a spiking neural network brain model and focused on its topology to better understand the relationship between connectivity and function.

Electroencephalography (EEG) studies report atypical resting-state neural activity in ASD; specifically, one of these atypical aspects is that of lower complexity than that found in EEGs in typical development (TD). One such measure of complexity is that of entropy-based complexity (i.e., multiscale entropy, MSE), in which entropy-based complexity is calculated over multiple temporal scales. Bosl et al. [2] analyzed the resting-state EEG signals of infants aged 6 to 24 months, and determined that MSE can predict ASD risk. Wang et al. [3] reviewed studies of atypical neural activity in individuals with ASD and argued that EEG power in persons with ASD has a U-shaped power spectrum. That is, EEG signals in ASD have excessively high power at low frequencies (delta and theta bands) and high frequencies (beta and gamma bands). Thus, there is evidence of atypical neural activity in individuals with ASD. The mechanism underlying the relationship between atypical structure and atypical function, however, is still unclear.

The aim of this study was to use the framework of small-world networks to understand the relationship between connectivity and neural activity in the normal and locally over-connected ASD brain. We used the method proposed by Watts

and Strogatz to construct a small-world network [5], and to control the extent of local over-connectivity without changing the number of connections. Thus, we could directly evaluate how variations in network structure affect network-level function. We calculated graph-theoretical measures to assess the degree of local over-connectivity in each network, and evaluated neural activity using MSE [6], [7]. We also analyzed the frequency spectrum of the simulated neural activity to elucidate the relationship between the frequency of the neural oscillation, its complexity, and network topology.

II. Modeling and Analysis

A. Network model based on the Watts and Strogatz model

We designed the initial network connectivity according to the Watts and Strogazt model [5] to control the degree of local over-connectivity of the network. First, we constructed a lattice network (see graph A in Fig. 1) in which each node connects to six neighboring nodes (three on each side). Second, each connection in the lattice network is rewired with a probability p_{WS}. The larger p_{WS} becomes, the greater the randomness of the connectivity of the network. The lattice network ($p_{\mathrm{WS}} = 0$) has a large number of clusters and long path length. When p_{WS} is slightly larger than zero, a few connections are rewired to create shortcut pathways (the red lines in Fig. 1) between clusters. A network with a limited number of shortcut pathways is a small-world network as shown graph B in Fig. 1. Here, we regarded the "lattice" network, characterized by local over-connectivity, as the ASD-like network, and the small-world network as the TD-like network.

B. Spiking neural network model

We employed the Izhikevich spiking neuron model [8] to construct our network model. This model can represent various firing patterns of biological neurons (e.g., spiking, bursting, and mixed mode firing patterns). Spiking neurons can fire synchronously in various frequency bands, similarly to activity patterns seen in the EEG. Furthermore, these spiking neurons are computationally efficient, so a large-scale model of a neural network is easily constructed. The neuron model is described as:

$$\frac{dv}{dt} = 0.04v^2 + 5v + 140 - u + I, \quad (1)$$

$$\frac{du}{dt} = a(bv - u), \quad (2)$$

where v denotes the membrane potential, u denotes the membrane recovery variable related to the activation of ionic currents, I denotes input current into the neuron and t is the time. I is calculated as the sum of connection weights of the firing pre-synaptic neurons that connect to the neuron. Eq. (3) describes the after-spike resetting:

$$\text{if } v \geq 30\,\text{mv, then } \begin{cases} v \leftarrow c \\ u \leftarrow u + d. \end{cases} \quad (3)$$

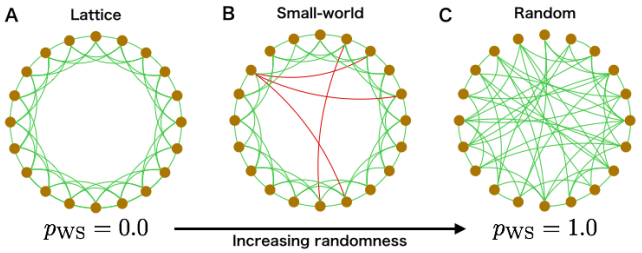

Fig. 1. Initialization of the connectivity structure of 100 neuron groups using the Watts and Strogatz model [5]. The brown nodes and the green edges indicate the neuron groups and connections, respectively. Graph A, a lattice network, is the initial structure that has local over-connectivity, where each node connects to six neighboring nodes, three on each side. All edges are rewired with the rewiring probability p_{WS}. As the probability increases, network structure becomes more random. Graph B is a small-world network that has a large number of clusters and short path lengths between these clusters. Graph C is the random network, where the nodes are randomly connected to others.

Here, a and b in Eq. (2) determine the time scale of u, c is the resetting membrane potential, and d describes the after-spiking reset of the recovery variable. We used excitatory (regular spiking) and inhibitory (fast spiking) neurons which neurons increase and decrease the probabilities of firing in post-synaptic neurons, respectively. These parameters were same as those in the Izhikevich model [8].

The other parameters in our model herein are summarized in TABLE I, which were determined based on the previous models [8]–[10]. Our model consisted of 100 neuron groups (N_{group}). Each neuron group has 1,000 spiking neurons (N), where 800 neurons are excitatory (N_{E}) and 200 are inhibitory (N_{I}). Excitatory neurons are connected to 100 neurons ($= N \times C_{\mathrm{intra}}$) belonging to the same neuron group. Each excitatory neuron connects to six neighboring neuron groups, and has three inter-connections ($= N \times C_{\mathrm{inter}}$) to neurons in each neuron group. Inhibitory neurons have connections to 100 excitatory neurons ($= N \times C_{\mathrm{intra}}$) in the same neuron group. These intra-group connections are randomly constructed. We set the numbers of neuron groups and inter-connections high enough to sustain spontaneous activity and to generate differences between network topologies with different p_{WS} values. In contrast, we determined the initial number of inter-connections between neuron groups via the Watts and Strogatz model. The rewiring probability (p_{WS}) regulates the degree of local over-connectivity (see Fig. 1). We tested the set of p_{WS} values: $\{0.0, 0.001, 0.002, 0.005, 0.01, 0.02, 0.05, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, 1.0\}$. The "lattice" network, which includes local over-connectivity, corresponds to the model representing ASD-like connectivity. We considered the small-world network, with a large number of clusters and short path lengths between these clusters, to be the model of TD. We defined the intra- and inter-connections as the intra-regional cortical connections and the white matter tract-based connections in the real brain, respectively.

After initializing the model's network connectivity using the Watts and Strogatz parameters [5], its network connectivity is

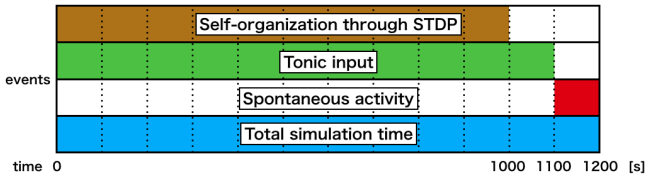

Fig. 2. Simulation timeline after connectivity initialization. We analyzed spontaneous neural activity during the red block.

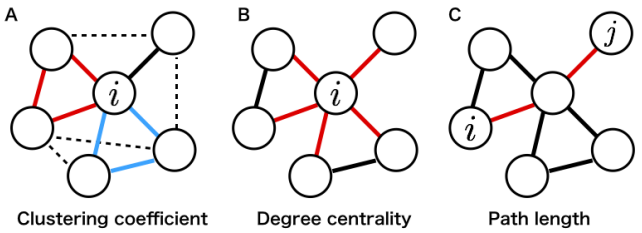

Fig. 3. Concepts of the three graph-theoretical measures. Panel A shows clustering coefficient of the ith node. The clustering coefficient indicates how many closed triangular connections (the red and blue connections in panel A) a node has, and is calculated by Eq. (6) or Eq. (7) (see Section II-C). The connections containing the dashed lines and forming closed triangular connections indicate the possible triangles in Eq. (6). Panel B shows degree centrality of the ith node. The degree centrality indicates the number of connections a node has (the red connections in panel B), and is calculated by Eq. (8). Panel C shows the path length from the ith node to the jth node. The path length (red line) indicates the shortest distance between these nodes.

updated through the spike-timing-dependent plasticity (STDP) rule proposed by Izhikevich [11], which enables the network to be spontaneously active. STDP is a self-organizing process that adjusts the weights of connections between neurons based on their pre- and post-synaptic spike timing. The update rule of the connection weight is given as:

$$\Delta w = \begin{cases} A_+ \exp(-t/\tau_+) & \text{if } t > 0 \\ A_- \exp(t/\tau_-) & \text{if } t < 0, \end{cases} \tag{4}$$

where t denotes the time lag between firings of the pre- and post-synaptic neurons:

$$t = t_{\text{post,fire}} - t_{\text{pre,fire}}. \tag{5}$$

If a neuron fires, the time lag (Eq. (5)) is calculated by the spike time of the nearest pre- or post-synaptic neuron, and the connection between them is updated according to Eq. (4). Here, the cases of $t > 0$ and $t < 0$ represent long term potentiation (LTP) and long term depression (LTD), respectively, and τ_+ and τ_- are constants that denote the durations of the time lags between firing. The constants A_+ and A_- denote the amplitude of the weight change or update.

The timetable of the simulation is shown in Fig. 2 and the parameters are described in TABLE I; they were determined based on the previous models [8]–[10]. The STDP (self-organization process) and tonic random input continued until 1000 and 1100 sec, respectively. We then analyzed spontaneous activity (from 1100 to 1200 sec) to observe the activity in the absence of tonic random input.

C. Graph-theoretical analysis

We evaluated network structures in terms of their graph-theoretical properties, i.e., the clustering coefficient, the degree centrality, and small-worldness [12]. The network graph consisted of 100 nodes that represent neuron groups. The clustering coefficient and degree centrality are indices of local over-connectivity, for where local over-connectivity is defined by increased short-range connectivity (i.e., connections between regions within the same lobe or cerebral hemisphere). Small-worldness is defined by the shortest path length and clustering coefficient. These properties are conceptualized in Fig. 3.

The clustering coefficient indicates how many closed triangular connections (the red and blue connections in Fig.3A) each node has, and is calculated as:

$$C_i = \frac{\text{number of closed triangles}}{\text{number of possible triangles}}. \tag{6}$$

Since our model is a directed weighted network, we used an extended clustering coefficient [13]:

$$C_i(W) = \frac{[W^{1/3} + (W^\top)^{1/3}]^3_{ii}}{2[d_i^{\text{tot}}(d_i^{\text{tot}} - 1) - 2d_i^{\leftrightarrow}]}, \tag{7}$$

where $W = \{w_{ij}\}$ is a weighted matrix of connectivity of 100 nodes, w_{ij} denotes the connection weight from the ith node to the jth node, $d_i^{\text{tot}} = d_i^{\text{in}} + d_i^{\text{out}}$ denotes the total number of connections into and out of the ith node, and $d_i^{\leftrightarrow}$ denotes the number of bilateral connections between the ith node and its neighbors.

The degree centrality indicates the number of connections. The value of the ith node is given as:

$$deg_i = \sum_{i \neq j}^{N} (w_{ij} + w_{ji}) \tag{8}$$

where N is the number of nodes.

The path length is the shortest distance between two nodes in a network. If there exists a connection between the ith and jth node ($w_{ij} > 0$), then its distance is defined as:

$$d_{ij} = \frac{1}{w_{ij}}. \tag{9}$$

The path length between any two nodes is given as the total distance along the paths.

Small-worldness [12] is an index that quantifies the small-world status of a network. The small-worldness for a network G is defined as:

$$S_G = \frac{C_G/C_0}{L_G/L_0}, \tag{10}$$

where C_G and L_G denote the averaged clustering coefficient and the averaged path length of G, respectively. L_G is the

TABLE I
PARAMETERS OF THE SIMULATION MODEL USED IN THIS STUDY.

parameters	values	descriptions	notes
$D_{\mathrm{intra,exc}}$	[0,20]	Transfer delay of excitatory synapse in neuron group	(uniform dist., msec)
$D_{\mathrm{intra,inh}}$	1	Transfer delay of inhibitory synapse in neuron group	(msec)
I_{tonic}	20	Tonic input	-
τ_+	20	Time constant of LTP	(msec)
τ_-	20	Time constant of LTD	(msec)
A_+	0.1	Amplitude of update weight (LTP)	-
A_-	-0.12	Amplitude of update weight (LTD)	-
$w_{\mathrm{init,exc}}$	6.0	Initial weight of excitatory synapse	-
$w_{\mathrm{init,inh}}$	-5.0	Initial weight of inhibitory synapse	-
w_{upper}	10.0	Maximum value of weight	-
N_{E}	800	The number of excitatory neurons in a neuron group	-
N_{I}	200	The number of inhibitory neurons in a neuron group	-
N	1000	The number of neurons in a neuron group	$= N_E + N_I$
C_{intra}	0.1	The percentage of connections in a neuron group	-
$D_{\mathrm{inter,exc}}$	[10,30]	Transfer delay of excitatory synapse between neuron groups	(uniform dist., msec)
N_{group}	100	The number of neuron groups	-
C_{inter}	0.003	The percentage of connections between neuron groups	-
p_{WS}	[0.0,1.0]	Rewiring probability	-
t_{step}	1	Time step	(msec)
T_{total}	1200	Total simulation time	(sec)
T_{tonic}	1100	Time length of tonic input	(sec)
T_{STDP}	1000	Time length of self-organization through STDP	(sec)
N_{sim}	10	The number of independent simulations	-

average of the shortest paths across all pairs of nodes in G. C_G and L_G are scaled by their counterparts in the lattice network ($p_{\mathrm{WS}} = 0.0$), C_0 and L_0. The small-world network has higher small-worldness than that of the lattice and the random networks.

D. Analysis of network activity

We employed the local averaged potential (LAP) [14] as a measure of network activity. The LAP represents synchronous activity of excitatory neurons in a neuron group. The LAP signal in the ith neuron group is shown in Eq. (11):

$$LAP_i(t) = \frac{1}{N_E}\sum_{j=1}^{N_E} v_{i,j}(t), \tag{11}$$

where $v_{i,j}$ denotes the membrane potential of the jth excitatory neuron in the ith neuron group. We analyzed the complexity and frequency spectrum of the LAP signals.

The MSE was proposed by Costa et al. [6], [7] to define the complexity of biological time-series signals. First, an original time-series signal $x(t)$ is downsampled by multiple temporal scales to produce so called coarse-grained signals $y(t)$:

$$y(t) = \frac{1}{\theta}\sum_{i=(t-1)\theta+1}^{i=t\theta} x(i) \quad (1 \le t \le N/\theta), \tag{12}$$

where θ denotes the scale factor. Next, the sample entropy of each coarse-grained signal is described as:

$$SampEn(r, m, N) = -\ln[C_{m+1}(r)/C_m(r)], \tag{13}$$

where

$$C_m(r) = \frac{\text{number of pairs}(i,j)\,(|z_i^m - z_j^m| < r,\, i \neq j)}{(N-m+1)(N-m)}. \tag{14}$$

Here, $z_i^m = \{y_i, y_{i+1}, \cdots, y_{i+m-1}\}$ denotes a subsequence of the coarse-grained signals from the ith to the $(i+m-1)$th, m denotes the length of the subsequence, $Y = \{y_1, \cdots, y_i, \cdots, y_N\}$ denotes the coarse-grained signal and N denotes the length of Y. For this study, we set $m = 2$ and $r = 0.15$. In Eq. (14), $C_m(r)$ represents the probability that similar pairs (z_i^m and z_j^m) exist in the m-dimensional space. The sample entropy evaluates unpredictability of time-series signals as the logarithmic ratio of the probabilities, $C_{m+1}(r)$ and $C_m(r)$.

Neural activity oscillates over a wide range of frequencies. To identify the predominant frequency characteristic of the neural activity, we analyzed its frequency spectrum using the fast Fourier transform.

III. RESULTS

Fig. 5 shows the LAP signals and frequency spectrum in the neuron group in a lattice and a small-world network. The power of high-frequency bands (around 50 Hz) in a lattice network was greater than that in a small-world network.

We calculated the MSE of the LAP signal of each neuron group in each network. Fig. 4 shows the MSE curve of each neuron group in a lattice network ($p_{\mathrm{WS}} = 0.0$), a small-world network ($p_{\mathrm{WS}} = 0.2$) and a random network ($p_{\mathrm{WS}} = 1.0$). These figures demonstrate that the MSE curves tended to become lower with coarse-graining, thus indicating that coarse-graining acts as a low-pass filter. Therefore, the neural oscillation with a lower frequency had lower complexity than the one with a higher frequency.

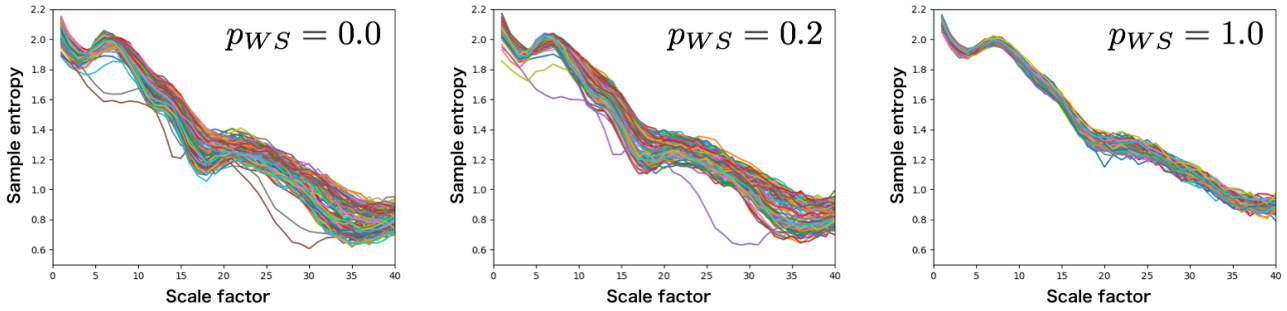

Fig. 4. The multiscale entropy-based complexity (MSE) curves of each neuron group in a lattice network ($p_{\mathrm{WS}} = 0.0$), a small-world network ($p_{\mathrm{WS}} = 0.2$) and a random network ($p_{\mathrm{WS}} = 1.0$). The y-axis indicates the sample entropy, and the x-axis indicates the scale factor θ (see Eq. (12), Section II-D).

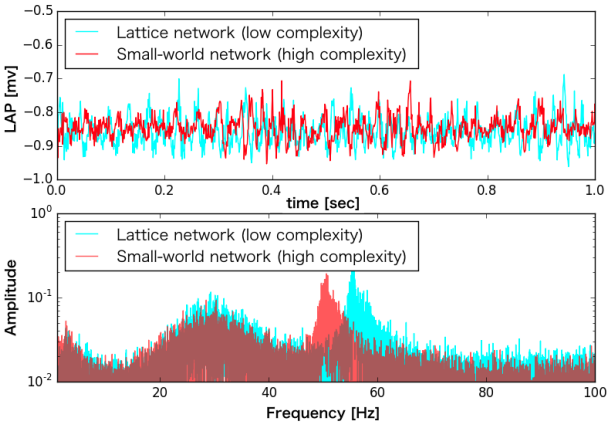

Fig. 5. Representative spontaneous activity (LAP) signals of a neuron group in the lattice network (red, $p_{\mathrm{WS}} = 0.0$) and that in the small-world network (blue, $p_{\mathrm{WS}} = 0.2$). The upper figure shows the LAP signals of neuron groups from 1100 to 1101 sec, and the lower figure shows the amplitude of each frequency spectrum sampled from the LAP signals from 1100 to 1200 sec.

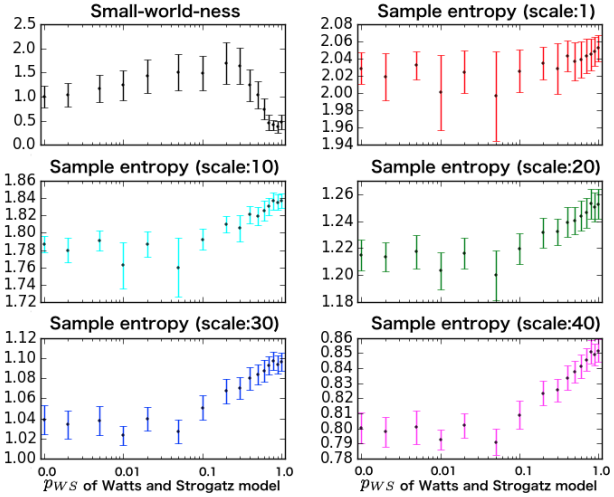

Fig. 6. The relationship between sample entropy and small-worldness. The x-axis indicates p_{WS} (the rewiring probability of the Watts and Strogatz model [5]). The upper left panel shows the small-worldness, the other panels show the sample entropy of scale 1, 10, 20, 30 and 40, respectively. The error bars indicate standard error across 10 independent simulations.

Fig. 6 shows the relationship between the sample entropy and the small-worldness of each network. The sample entropy tended to decrease with a lower p_{WS} at each scale. The small-worldness value was greatest at $p_{\mathrm{WS}} = 0.2$, and both the lattice and random networks had less small-worldness. Notably, the network with the highest small-worldness (the TD-ike network, $p_{\mathrm{WS}} = 0.2$) has higher complexity than the lattice (ASD-like) network.

Fig. 7 shows the relationship between the graph-theoretical properties and the complexity of neural activity. The neuron groups in the lattice network ($p_{\mathrm{WS}} = 0.0$) had higher clustering coefficients and degree centrality than those in the small-world network ($p_{\mathrm{WS}} = 0.2$) and a random network ($p_{\mathrm{WS}} = 1.0$). Notably, the neural activity in the lattice network had lower complexity.

Fig. 8 shows the relationship between peak frequency and complexity of neural activity. Neural activity dominated by a higher frequency had lower complexity. These results suggested that the neuron groups in the lattice network had higher clustering coefficients and degree centrality and that such connectivity enhances the high-frequency components of the neural oscillation, thus decreasing its complexity.

IV. Discussion and Conclusions

To understand the mechanism underlying the lower complexity of neural activity in the ASD brain, we investigated the relationship between structural connectivity and neural activity. We focused on the atypical connectivity seen in ASD called local over-connectivity and constructed a spiking neural network model to emulate it. In this model, the degree of local over-connectivity was modified according to the Watts and Strogatz model [5]. We analyzed its connectivity structure using graph theory and measured the frequency spectrum and complexity of the neural activity. Our simulation showed that the neural activity of a lattice network (ASD-like) had excessively high power in high frequency bands and lower complexity than the other tested networks. This result agrees with previous studies analyzing resting-state EEG signals in ASD [2], [3].

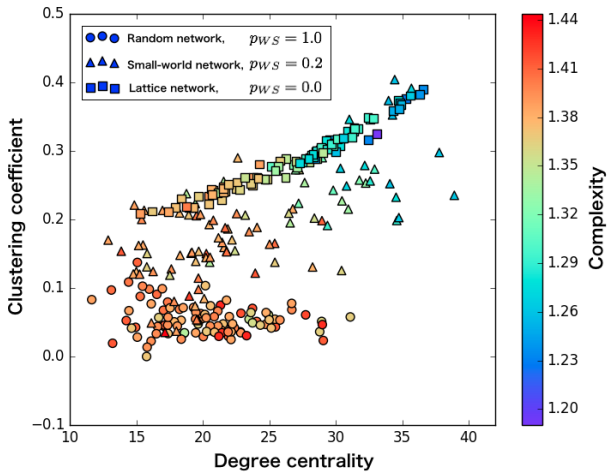

Fig. 7. The relationship between connectivity structure and the complexity of neural activity. Each marker corresponds to a neuron group in the network and its color indicates the sample entropy averaged over all 40 scales. The x-axis indicates the degree centrality and the y-axis indicates the clustering coefficient.

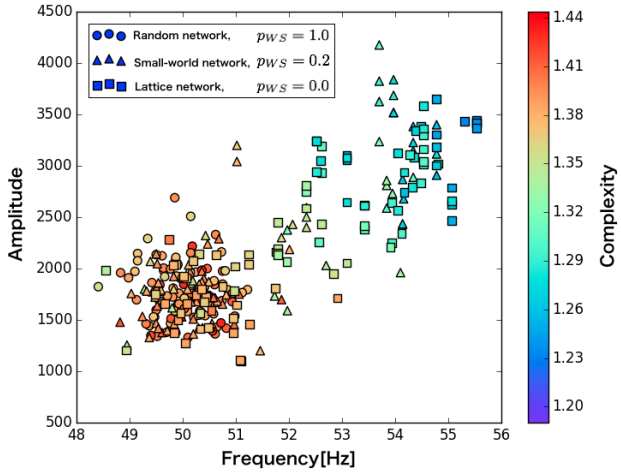

Fig. 8. The relationship between peak frequency and complexity of neural activity. Each marker corresponds to a neuron group in the network and its color indicates the sample entropy averaged over all 40 scales. The x-axis indicates the peak frequency of neural activity and the y-axis indicates the amplitude.

Based on our results, we propose the following hypothetical mechanism:

1) Due to the local over-connectivity, interactions between neighboring neuron groups or in a local cluster predominate (see Fig. 7);
2) Excessive local interaction synchronizes local neural activities, and enhances high-frequency oscillations;
3) The activity dominated by high-frequency oscillations leads to its lower complexity.

Several previous studies of the human brain reported similar phenomena. Ghanbari et al. [15] investigated the resting-state EEG of persons with ASD and TD, and proposed that enhanced connectivity would lead to better regulation of neural activity, resulting in lower complexity. Courchesne and Pierce [1] hypothesized that the brains in persons with ASD have enhanced local synchronization that would disturb inputs from afferent sources, such as sensory areas, leading to de-synchronization between distant regions.

As shown in Fig. 5, although high-frequency oscillations in a lattice network, i.e., the ASD-like network, were greater than that in the small-world network, i.e., the TD-like network, there were no significant differences in the lower frequency bands between the lattice and the small-world networks. This is in contrast to the prediction of a U-shaped spectrum proposed by Wang et al. [3]. Further studies are needed to understand the origin of the higher power in the low frequency band.

In this study, we analyzed spontaneous activity of neural networks. In order to better understand why persons with ASD have difficulty in social interaction, show repetitive behaviors, and dysesthesia, future studies will investigate how external inputs to typical and atypical networks affect neural activity.

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