INFERRING LARGE-SCALE STRUCTURAL CONNECTIVITY FROM LOCAL ACTIVITY IN A MODEL OF THE MOUSE LARGE-SCALE CORTICAL NETWORK

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ABSTRACT

Inferring the structural connectivity from electrophysiological measurements is a fundamental challenge in systems neuroscience. Directed functional connectivity measures, such as the Generalized Partial Directed Coherence (GPDC), provide estimates of the causal influence between areas. However, the relation between causality estimates and structural connectivity is still not clear. We analyzed this problem by evaluating the effectiveness of GPDC to estimate the connectivity of a ground-truth, data-constrained computational model of a large-scale network model of the mouse cortex. The model contains 19 cortical areas comprised of spiking neurons, with areas connected by long-range projections with weights obtained from a tract-tracing cortical connectome. We show that GPDC values provide a reasonable estimate of structural connectivity, with a Pearson correlation of 0.73. Moreover,

even in a typical electrophysiological recording scenario containing five areas, the mean correlation was above 0.6. These results indicate that GPDC estimates provide statistical information on structural connections, but individual connection estimates are not reliable.

1 Introduction

The communication between brain regions is often analyzed through structural and functional connectivity [1]. The former refers to anatomical connections between brain regions generally quantified using tracer injections or diffusion magnetic resonance imaging [2]. The map of these connections is called "connectome" [3], whose analysis includes methods to evaluate the networks defined by nodes (brain regions) and edges (synapses) [4, 5]. Functional connectivity evaluates brain communication from statistical relations between recorded brain signals [6, 1]. Particularly, directed functional connectivity methods use the concept of causality to infer both the intensity and the direction of the connections between brain regions [7]. Even though there is some association between structural and functional connectivity, the relationship between them is not straightforward [1]. While the former is practically static and compose the map of possible pathways for information flow between brain regions, the latter changes continuously and depends, for example, on the dynamical states of brain regions, noise, and strength of structural connections [8].

During electrophysiological procedures, researchers typically record brain signals using electrodes positioned in different depths of brain regions. Even with the improvement in technologies for recording signals, it is usually possible to record signals only from a few areas compared to the number of sources of activity in the brain [9, 10, 11]. Thus, the functional connectivity analysis presents a problem because many unrecorded regions may indirectly influence other regions as common inputs [7, 12, 6]. Therefore, the comparison between structural and functional connectivity becomes more complicated since spurious inferred causality relations can lead to misinterpretations of electrophysiological data.

Some simulation studies evaluated the relation between directed functional connectivity and structural connections [8, 13, 14, 15]. However, most of these studies used either autoregressive models for the dynamics of each node [16] or rate-based models [13]. These studies provided important steps towards evaluating the reliability of causality measures, but they are still distant from laboratory experimental conditions.

In this work, we investigate the relationship between directed functional connectivity and structural connectivity in a large-scale network model of the cortex, derived from a cortical connectome of the mouse obtained using tracer injections [17]. Causality measures obtained using GPDC explained most of the variance of structural connection strengths. The mean correlation remained high ($r > 0.6$) even when only a few cortical areas were considered in the GPDC calculation, indicating that this causality measure provides reliable results in typical experimental conditions in which only recordings from a subset of areas are available.

2 Methods

2.1 Neuron model

We modeled the neurons using a single-compartment Hodgkin–Huxley-type model, where the membrane potential of the i-th neuron described by,

$$C_m \frac{dV_i}{dt} = -g_{Na} m_i^3 h_i (V - E_{Na}) - g_K n_i^4 (V - E_k) - g_L (V - E_L) + I_{\text{ext},i} + I_{\text{syn},i}, \qquad (1)$$

the membrane capacitance C_m is 0.50 nF (0.125 nF) for excitatory (inhibitory) neurons. The maximal conductances values were $g_{Na} = 12.5$ μS, $g_K = 4.74$ μS and $g_L = 0.025$ μS. The reversal potentials $E_{Na} = 40$ mV, $E_K = -80$ mV, and $E_L = -65$ mV correspond to the sodium, potassium and leakage channel, respectively [18]. The dynamics of the voltage-gated ion channels are described by activation and inactivation variables m, n, and h, where m and n accounts for the

dynamics of Na channels and h for K channels. The probability that an ion channel is open evolves according to a set of ordinary differential equations [19],

$$\frac{dm}{dt} = \alpha_m(V)(1-m) - \beta_m(V)m, \tag{2}$$
$$\frac{dh}{dt} = \alpha_h(V)(1-h) - \beta_h(V)h, \tag{3}$$
$$\frac{dn}{dt} = \alpha_n(V)(1-m) - \beta_n(V)n, \tag{4}$$

where,

$$\alpha_m(V) = 0.1\frac{(V+16)}{1-\exp(-(V+16)/10)}, \tag{5}$$
$$\beta_m(V) = 4\exp(-(V+41)/18), \tag{6}$$
$$\alpha_h(V) = 0.07\exp(-(V+30)/20), \tag{7}$$
$$\beta_h(V) = [1+\exp(-V/10)]^{-1}, \tag{8}$$
$$\alpha_n(V) = 0.01\frac{(V+20)}{1-\exp(-(V+20)/10)}, \tag{9}$$
$$\beta_h(V) = 0.125\exp(-(V+30)/80), \tag{10}$$

The parameters used in this neuron model was previously reported and applied in some studies that modeled cortical neuronal populations. [20, 19, 21]

2.2 Spiking neuronal population model

Each spiking neuronal population was composed of 2000 neurons, 1600 excitatory and 400 inhibitory. Neurons within each spiking neuronal population were randomly connected with probability $p_{\text{intra}} = 10\%$. The synaptic current I_{syn} that arrives to postsynaptic neuron i is modeled by,

$$I_{\text{syn},i}(t) = \sum_{j \in \text{presyn}} g_{\text{syn},i,j}(t) \left[E_{\text{syn}} - V_i(t) \right], \tag{11}$$

where the index j represent a presynaptic neuron connected to neuron i and the sum over j accounts for all the synapses that impinge on neuron i. E_{syn} is the synaptic reversal potential which is 0 mV for excitatory and -70 mV for inhibitory synapses. The dynamics of synaptic conductance $g_{\text{syn},i,j}$ is described by an exponential function as follows [22],

$$\frac{dg_{\text{syn},i,j}}{dt} = -\frac{g_{\text{syn},i,j}}{\tau}. \tag{12}$$

When a presynaptic neuron j fires a spike, a synaptic weight w is added on the synaptic conductance $g_{\text{syn},i,j}$ after the axonal delay, which was set as 1 ms [19]. The value of w depends on the excitatory/inhibitory nature of the presynaptic and postsynaptic neurons. The synaptic time constant τ is 2 ms and 8 ms for excitatory and inhibitory synapses, respectively. Furthermore, all neurons receive a background input given by a heterogeneous Poisson-process spiking activity with a rate of 7.3 kHz [19]. The effect of the background input in each neuron is an excitatory synaptic current. To add heterogeneity in our model, all synaptic weights w for recurrent connections and background input were taken from a Gaussian distribution (Table 1).

Table 1: **Synaptic weights for intra-areal connections**. Mean synaptic weight $\overline{w}$ and standard deviation σ_w for all possible synapses. E, I, and Input represent excitatory neurons, inhibitory neurons, and external input, respectively. The arrow indicates the direction of the connection.

Synapses	$\overline{w}$ (nS)	σ_w (nS)
$E \to E$	2.5	1.0
$E \to I$	2.5	1.0
$I \to E$	240	10
$I \to I$	240	10
$Input \to E$	3.2	1.0
$Input \to I$	3.2	1.0

2.3 Mouse large-scale cortical network

The mouse cortex's large-scale network model comprises 19 cortical areas where a spiking neuronal population models each area with long-range and recurrent synapses. Parameters related to recurrent synapses were described in the previous session. Neurons from different areas are randomly connected with probability $p_{\text{inter}} = 5\%$. The synaptic weights between cortical areas are based on the recently published anatomical connectivity dataset for the mouse cortex [17] obtained by retrograde tracer injections [23]. This technique consists in injecting a tracer that flows from the target area to the cell bodies, allowing to identify neurons projecting to the target area. The Fraction of Labeled Neurons (FLN) was measured as the ratio of the number of labeled neurons in a source area to the total quantity of labeled neurons in all source areas [24, 25]. The synaptic weights for directed long-range connections are the FLNs scaled by the global scaling parameters $\mu_{\text{E}} = 50$ and $\mu_{\text{I}} = 25$,

$$w^i_{\text{lr,E}} = \mu_{\text{E}} \sum_j^N \text{FLN}_{i,j} \tag{13}$$

$$w^i_{\text{lr,I}} = \mu_{\text{I}} \sum_j^N \text{FLN}_{i,j}. \tag{14}$$

Long-range connections as excitatory, targeting either excitatory or inhibitory neurons with synaptic weight, $w^i_{\text{lr,E}}$, and $w^i_{\text{lr,I}}$, respectively. The index j represents the source area, i the target area, and N is the total number of simulated cortical areas. The axonal delay for long-range connections is given by the ratio between the anatomical distance estimates between cortical areas and the conduction speed set as 3.5 m/s [26].

2.4 LFP signal

We computed the local field potential (LFP) signal as a sum of the currents' absolute values acting upon excitatory neurons in a spiking neuronal population [27, 28]. Thus, for a cortical area in our

model, the LFP signal will be given by,

$$\text{LFP} = R\frac{\sum_i^{N_\text{E}}(|I_{\text{E},i}| + |I_{\text{I},i}| + |I_{\text{bkg},i}|)}{N_\text{E}}, \tag{15}$$

$I_{\text{E},i}$ accounts for both the local (within population) and global (inter-areal projections) excitatory synaptic currents, while $I_{\text{I},i}$ corresponds to the local inhibitory current. $I_{\text{bkg},i}$ is the synaptic current related to the background Poisson input. R represents the resistance of a typical electrode used for extracellular measurements, here chosen to be 1 MΩ [19]. N_E is the number of excitatory neurons in each neuronal population.

The mean was subtracted from the simulated LFP signal. The resultant signal was filtered using a low-pass filter in 1 kHz to avoid aliasing and downsampled to 1 kHz.

2.5 Generalized partial directed coherence

Generalized partial directed coherence (GPDC) is a frequency-domain method of directed functional connectivity established on multivariate vector autoregressive (MVAR) model [29]. Considering that LFP signals from all areas in the model are represented by a set $\mathbf{x}(t) = [x_1(t) \cdots x_N(t)]^T$ of simultaneously observed time series. The MVAR model for $\mathbf{x}(t)$ is defined as:

$$\mathbf{x}(t) = \sum_{k=1}^{p} \mathbf{A}_k \mathbf{x}(t-k) + \epsilon(t) \tag{16}$$

where p is the MVAR model order. $\mathbf{A}_k$ are coefficient matrices in which the element $a_{ij}^{(k)}$ define the effect of $x_j(t-k)$ on $x_i(t)$. The term $\epsilon(t)$ is a vector of N white noises with covariance matrix $\mathbf{\Sigma}$. The GPDC from the time series x_j to the time series x_i at frequency λ is defined as,

$$\text{GPDC}_{ij}(\lambda) = \left| \frac{\frac{1}{\sigma_i}\overline{A}_{ij}(\lambda)}{\sqrt{\sum_{k=1}^{N} \frac{1}{\sigma_k^2}\overline{A}_{kj}(\lambda)\overline{A}_{kj}^*(\lambda)}} \right|^2, \tag{17}$$

where

$$\overline{A}_{ij}(\lambda) = \begin{cases} 1 - \sum_{k=1}^{p} A_{ij,k} e^{-2\pi\sqrt{-1}\lambda k}, & \text{if } i = j \\ -\sum_{k=1}^{p} A_{ij,k} e^{-2\pi\sqrt{-1}\lambda k}, & \text{if } i \neq j, \end{cases} \tag{18}$$

and σ_i^2 refers to the variance of white noise $\epsilon_i(t)$ [29]. λ is a normalized frequency where $\lambda = 0.5$ means one half of the sampling rate fs [30]. The MVAR model was estimated by the method of ordinary least squares (OLS) [31]. Akaike's information criterion gave the model order indicated that the best model order p that was lesser than or equal to 50 [30]. For all analysis it was evaluated the peak of GPDC.

2.6 Weighted FLN and weighted GPDC

In order to investigate the propagation of neuronal activity through the links defined by structural and directed functional connectivity, we defined the weighted FLN (wFLN) and the weighted GPDC (wGPDC) [32],

$$\text{wFLN}_{ij} = \frac{1}{n} \sum_{j=1}^{n} \left(\frac{\text{FLN}_{ij} r_j}{\sum_{j \in S} r_j} \right) \tag{19}$$

$$\text{wGPDC}_{ij} = \frac{1}{n} \sum_{j=1}^{n} \left(\frac{\text{GPDC}_{ij} r_j}{\sum_{j \in S} r_j} \right) \tag{20}$$

where FLN_{ij} is the FLN from area j to area i, r_j is the firing rate for area j, GPDC_{ij} is the peak of GPDC from area j to area i, S is the set of source areas for target area i and finally and n is the cardinatility of set S.

2.7 Numerical simulations

All simulations were performed using the simulator Brian2 [33] applying the exponential Euler method [34] to integrate the differential equations with an integration step of 0.1 ms. Each simulating was 30 s long, generating sufficient data points to apply GPDC on the simulated LFP signals [35].

3 Results

The large-scale network model of the mouse cortex contains 19 spiking neural populations with recurrent connections and excitatory long-range connections between populations, constrained by the directed and weighted structural connectome (Figure 1A and Figure 1B). The dynamical behavior of each simulated cortical area is predominantly asynchronous with transient spike synchronization [36, 37] (Figure 1C), with the typical power spectral density (PSD) of LFP signals displaying a peak in the gamma band (Figure 1D and 1E) [38]. The firing rate of inhibitory neurons is 4.74 ± 0.11, higher than excitatory neurons rate of 3.64 ± 0.42 (Figure 1F). Differences in population behavior are mostly due to inputs from other areas since we sample their parameters from the same distributions (Section 2.2).

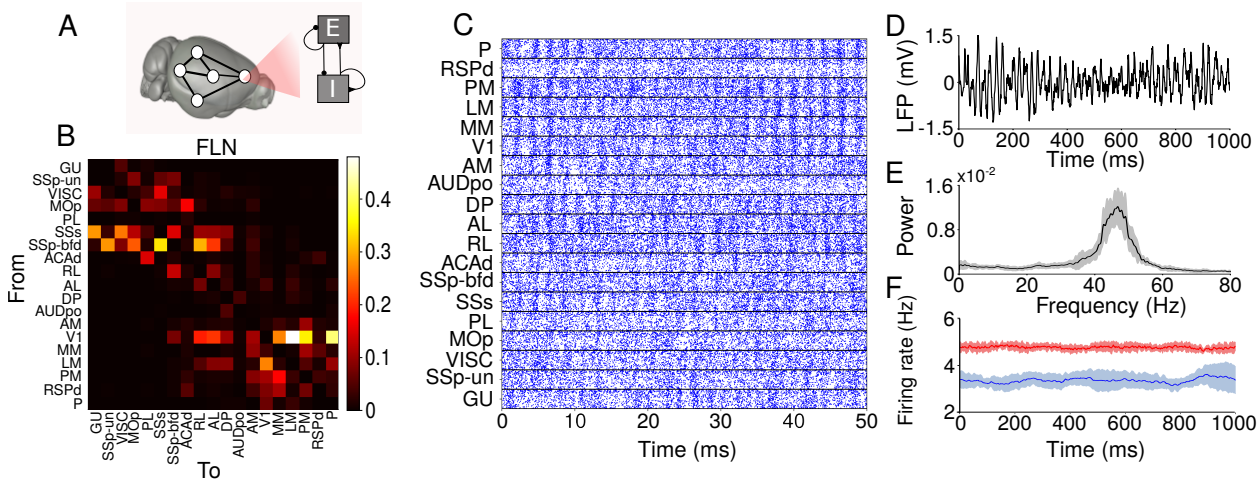

Figure 1: **Large-scale cortical network.** (A) Local neuronal population where E and I are populations of spiking neurons. (B) Map of structural connectivity given by the FLNs. These values define the strength of long-range projections in the large-scale network model. (C) Raster plot of $50ms$ of activity for each cortical area. (D) Simulated LFP signal for an area in the large-scale network model. (E) Power spectral density for simulated LFP signals from one area. Continuous black line corresponds to the average over ten simulations, and the gray shaded area delimits its standard deviation. (F) Firing-rate for excitatory (blue) and inhibitory (red) populations computed using a sliding window of $100ms$. The continuous line corresponds to an average firing-rate over ten simulations, and the shaded area is the standard deviation. To exemplify, we used data from area MOp in (D), (E), and (F).

We first compared the FLN values to the average GPDC over ten simulations of the model. Most medium to strong connections from the structural connectome were also captured by the directed

functional connectivity (Figure 2A and Figure 2B). We used the GPDC largest value (peak), but other approaches such as the average of GPDC over frequencies and area under the GPDC curve (Supplementary Figure S1) produced similar results.

Although the graph density of the structural connectome is 97% [17], most structural connections are weak, which leads to a prevalence of weak average GPDC values. Weak structural connections are characteristics shared by connectomes from different mammals, with FLNs varying by several orders of magnitude, log-normally distributed [23, 39, 17, 40]. To evaluate the relation between structural and directed functional connectivity, we plotted GPDC values from ten simulations against FLNs and fitted a linear model, obtaining the Pearson correlation r (Figure 2C). The scatter plot presents most points close to the origin due to the predominance of small values for the GPDC and FLN. Interestingly, for some FLNs, there is a high variation of GPDC values over different simulations. The Pearson correlation between FLN and GPDC is 0.73, and GPDC explains approximately 54% of the variance in FLN values. This explainability level similar to those obtained by other works that analyzed different structural connectomes using functional connectivity applied to empirical data [41, 42] or firing-rate models [43].

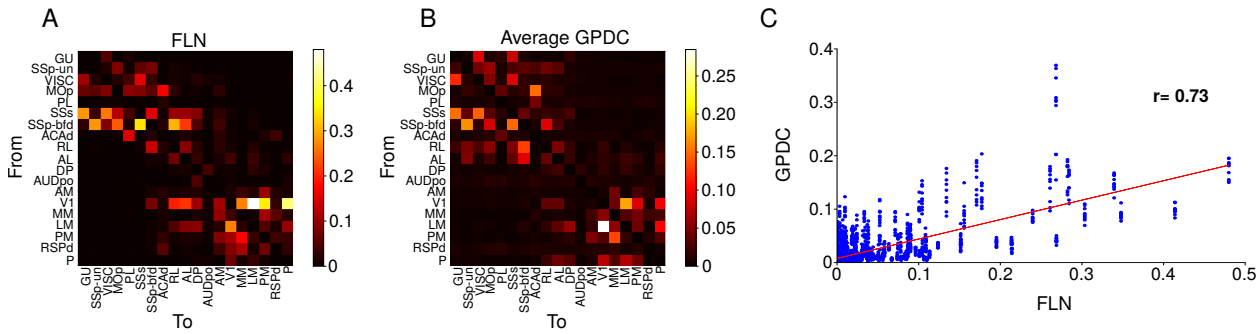

Figure 2: **Relation between structural and directed functional connectivity.** (A) Map of structural connectivity given by the FLNs. (B) Map of directed functional connectivity given by the average of GPDC peaks over ten simulations. (C) Scatter plot of FLNs *versus* GPDC peak. The red line corresponds to the linear fit. The Pearson correlation between FLNs and GPDC is 0.73.

We also investigated the propagation of activity between cortical areas through the pathways defined by structural connectivity. The propagation of activity in the cortex is constrained by direct anatomical connections between areas and indirect paths [44], with the propagation of activity

occurring mainly through the strongest long-range projections [24]. The weighted FLNs are strongly correlated to the target areas' firing rate (Figure 3A), while the correlation of weighted GPDCs and firing rates was 0.54 (Figure 3B). This indicates that neural activity propagation is directly dependent on the strength of structural connections and that GPDC can capture part of this activity propagation.

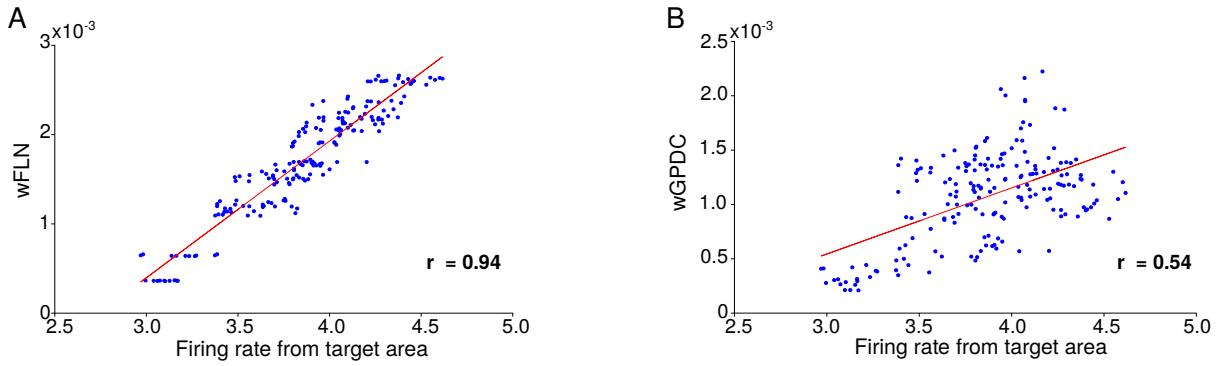

Figure 3: **Propagation of activity through structural and directed functional pathways**. (A) Weighted FLN $versus$ firing-rate for target areas ($r = 0.94$). (B) Weighted GPDC $versus$ firing-rate for target areas ($r = 0.54$). Red lines are linear fits.

We analyzed the behavior of GPDC estimates when considering a reduced number of areas, reproducing typical experimental setups. We considered a visual and a frontoparietal cluster, each containing 7 cortical areas [17]. We evaluated the GPDC estimates between all areas of each cluster (Figures 4C and 4F), conditioned on the whole connectome (Figures 4B and 4E), conditioned on the areas in each cluster, and using only pairwise (bivariate) estimates (Figures 4D and 4G). This analysis simulates the situations where an electrophysiologist only has information from a single cluster of cortical areas or a pair of areas. The highest correlations between the GPDC and FLN occurred when we conditioned GPDC to the whole connectome, followed by GPDC conditioned to the cluster area, and pairwise GPDC. Also, the correlation for the frontoparietal cluster was higher in all scenarios.

We extended the analysis to evaluate the effect of cluster size on GPDC correlation to FLN. We used cluster sizes ranging from 2 to 15 areas. We created 150 random clusters sampled from all areas in the connectome for each cluster size and computed the Pearson correlation for the GPDC

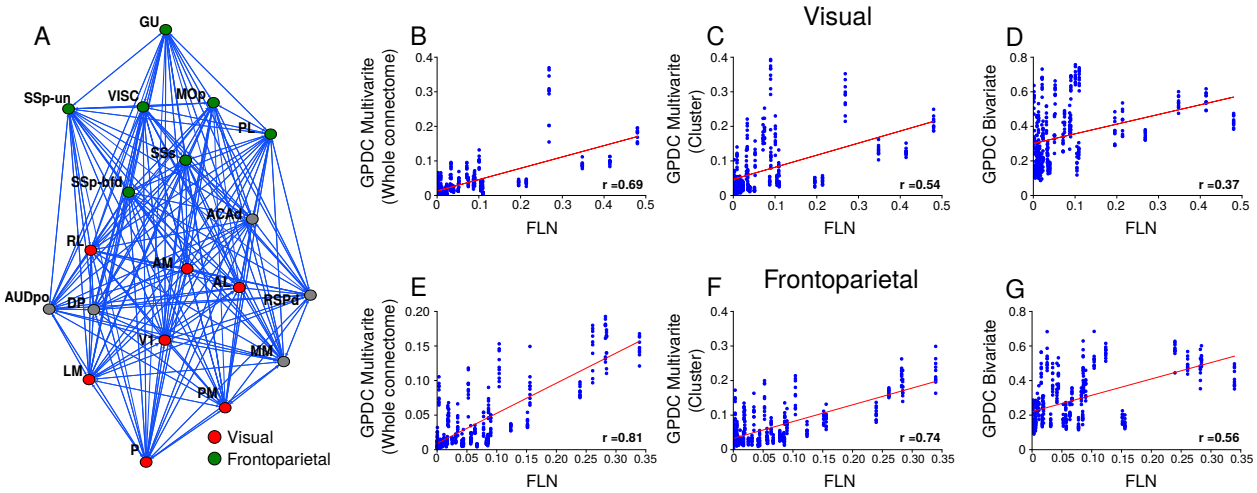

Figure 4: **Correlation between FLN and GPDC for the visual and frontoparietal clusters.** (A) Graph representing the mouse cortical connectome. Nodes represents cortical areas and edges, directed long-range projections between them. Green nodes are cortical areas belonging to the frontoparietal cluster. Red nodes are cortical areas belonging to the visual cluster. Each cluster contains 7 cortical areas. (B-D) FLN *versus* GPDC for the visual cluster, when (B) conditioned to the whole connectome, (C) conditioned to the areas in the cluster, and (D) using bivariate data. (E-G) FLN *versus* GPDC for the frontoparietal cluster when (E) conditioned to the whole connectome, (F) conditioned to the areas in the cluster, and (G) using bivariate data. Red lines are linear fits and r is the Pearson correlation.

(A) conditioned on the whole connectome, (B) conditioned on the cluster areas, and (C) evaluated using pairwise data. For cases (A) and (B), the Pearson correlation increases, and the standard variation decreases as we increase the cluster size (Figure 5), showing that it is advantageous to include more areas in the GPDC calculation. Surprisingly, the correlation between structural and directed functional connectivity when using signals measured in a few cortex areas (blue dots) is similar to using signals from the whole cortex (black dots), with few points showing statistically different results. The correlation between whole connectome GPDC and cluster GPDC estimates increases for large clusters, as expected (Supplementary Figure S2). The bivariate GPDC (Figure 5) had a statistically significant lower average Pearson correlation for all cluster sizes with four or more areas, indicating that these measures are affected by interference from ignored signals.

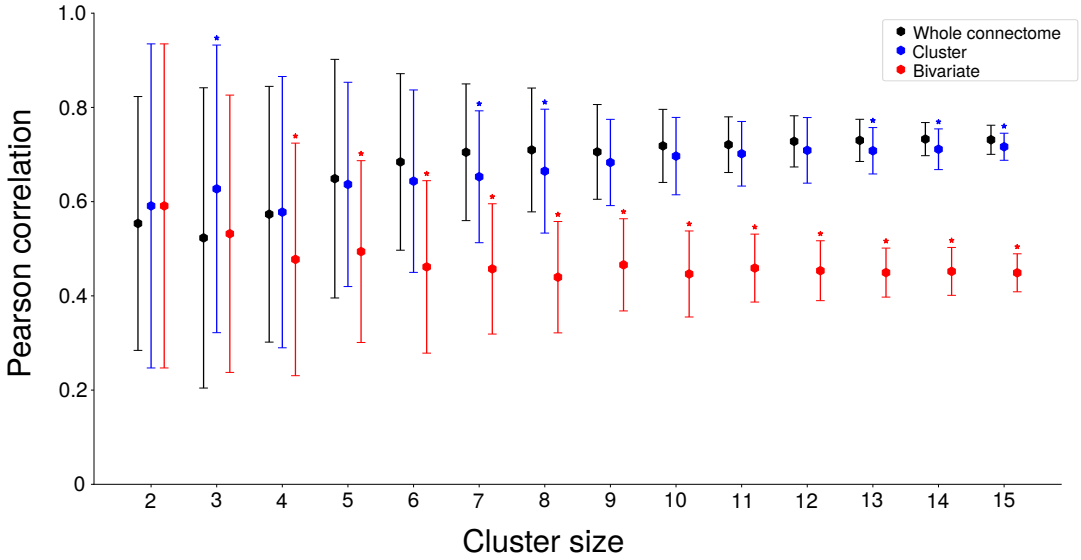

Figure 5: **Pearson correlations between FLN and GPDC for the different cluster sizes with randomly chosen areas.**. The graph shows the average (dots) and error bars of the GPDC conditioned to the whole connectome (in black), to areas in the cluster (in blue), and to pairwise signals (in red). Stars represent statistically different averages when compared to the whole connectome (Holm-Bonferroni corrected Welch t-test).

4 Discussion

Our results shed light on the relationship between structural and directed functional connectivity in circumstances similar to those faced by electrophysiologists. They indicate that the reliability of directed functional connectivity estimates and their relationship with structural connectivity depends on the number of areas considered. Nevertheless, the GPDC conditioned on few cortical areas had similar results to the GPDC conditioned on all areas, providing evidence that it is possible to obtain a reasonable relationship between structural and directed functional connectivity in electrophysiological experiments even with signals from few areas.

Previous studies evaluated the relationship between structural and functional network connectivity strength on electrophysiological data [45], with some using undirected functional connectivity measures [46, 47]. But in electrophysiology studies researchers do not have access to signals from unrecorded areas and have only estimates of structural strengths from tracers. Using large-scale

network models solves this problem, as the researcher has access to all variables in the system, allowing a better understanding of the obtained functional connectivity results.

The relationships between structural and functional connectivity has been largely unexplored through large-scale network models [48], and the existing models use neural mass descriptions (rate models) to describe each area's activity [49, 50]. However, information propagated between brain regions can be characterized not only by the rate code but also by the temporal code [51, 52, 53, 54, 55], and hypotheses are pointing to spike-timing and spike coherence as essential components of cortical communication [56, 36, 57]. Spiking neuronal populations have richer dynamical behaviors than rate models and better resemble cortical activity, including the effects of neuronal synchronization and its effects on information propagation. Moreover, our obtained correlations are in the same range of the studies using more complex electrophysiological data [45], indicating that our simulations did not produce artificially high correlations due to model simplifications.

The propagation of activity between simulated cortical areas is strongly correlated with weighted FLNs and moderately correlated with weighted GPDC estimates, when using the concept of activity flow proposed by Cole *et al.* [32]. This result is interesting since it indicates that structural connectivity is more relevant to activity propagation than direct functional connectivity extracted from GPDC peak values. In other words, GPDC do not directly correspond to activity flow between areas and should be used with caution in this case.

The relationship between structural and directed functional connectivity is the largest when GPDC is conditioned to all areas in the connectome and and decreases as we reduce the number of areas. Gămănut et al. identified 6 clusters in the mouse connectome (prefrontal, frontal, parietal, cingulate, temporal and visual) [17] based on the same approach used to investigate the macaque cortex [58]. We evaluated the relationship between GPDC and FLNs in the visual cluster and in a combination of the prefrontal, frontal and parietal clusters, which we called frontoparietal. We did not use the other clusters, which had a small a small number of regions. The obtained correlation was in the range of random clusters, with $r = 0.74$ for the frontoparietal and $r = 0.54$ for the visual cortex.

This indicates that within anatomical clusters the relationship between GPDC and FLNs does not change in relation to randomly selected areas. These results also show that GPDC estimates provide statistical information on structural connections even when considering a few areas, but individual connection estimates are not reliable.

It is well known that indirect connections and common input from areas not included in GPDC influence the estimates [7, 14, 6, 49], but the magnitude of this influence had not been quantified. We verified that the correlation between whole connectome GPDC and cluster GPDC increases with the number of areas considered in the measurement (Supplementary Figure S2A). For seven areas, the Pearson correlation between the whole connectome GPDC and cluster GPDC was 0.65, resulting in a explained variance of 42%. This shows that areas not considered in the GPDC calculations can have an important impact in the GPDC estimates. We also showed that the difference between the whole connectome GPDC and cluster GPDC values decreases with larger cluster sizes (Supplementary Figure S2B) as expected.

Our large-scale network model has some limitations. First, modeled neuronal population parameters are drawn from the same distributions with activity in the gamma band range (Figure 1). The activity of cortical areas in mice occurs in multiple frequency ranges [59, 60] and the relationship between structural and functional connectivity depends on the frequency [61]. A second limitation is that we do not model changes in network states, which are known to influence functional connectivity [62]. Some studies in computational neuroscience have already explored multistability and temporal patterns of functional connectivity [63, 64, 65]. Finally, we considered only cortical areas in our large-scale network model, excluding subcortical areas, which have a more complex dynamic [66, 67]. Future studies can overcome these limitations by creating richer spiking network models, with different operating frequencies and evolving neuronal dynamics. These models are difficult to create, but would allow one to compare functional connectivity values to structural connection strength in more dynamical settings.

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Supplementary Material

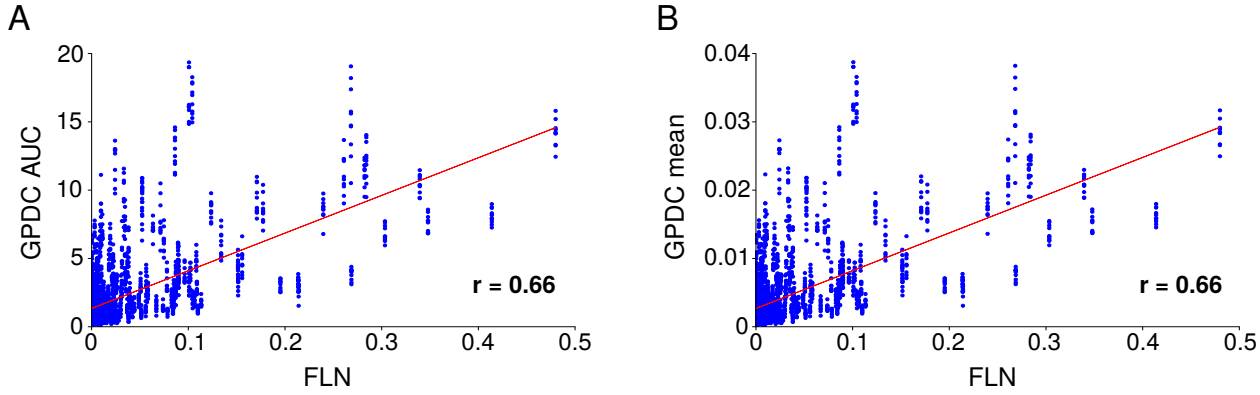

Figure S1: **Area Under Curve (AUC) and mean of GPDC estimates** . (A) GPDC AUC $versus$ FLN. (B) GPDC mean $versus$ FLN. In both cases, the Pearson correlation between GPDC mean or AUC and FLN is 0.66. Red lines are linear fit.

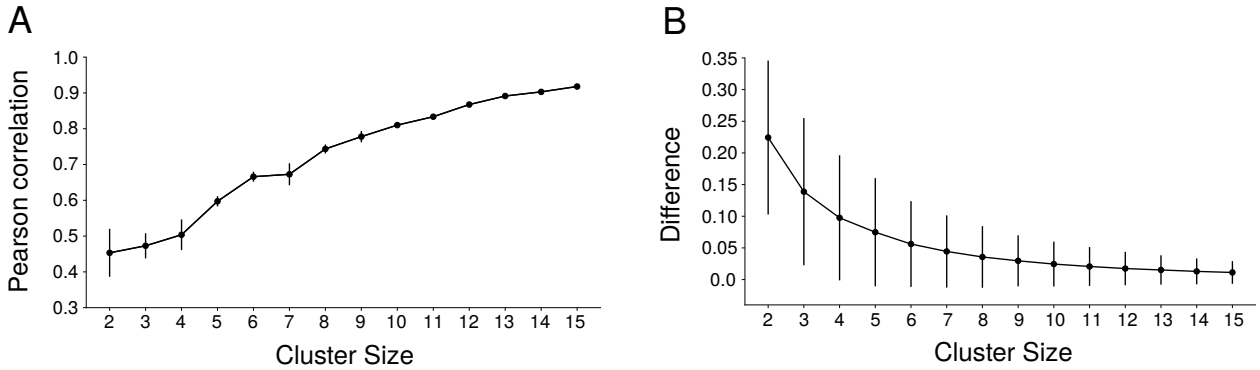

Figure S2: **Correlation and difference between GPDC etimates conditioned to the whole connectome and GPDC estimates conditioned to the cluster areas**. A)Pearson correlation increases with the cluster size. Each bullet represents the average pearson correlation over 150 clusters composed of randomly chosen areas. Comparing all clusters size, the highest value for standard deviation of pearson correlation 0.07. B) Average difference between GPDC estimates conditoned to the whole connnectome and GPDC estimates conditioned to the cluster areas. Average and standard deviation decrease with the cluster size.

Table S1: **Name of areas in mouse cortical connectome.** Adapted from Gămănut et. al., 2018.

Abbreviations	Areas
ACAd	Anterior cingulate area dorsal part
AL	Anterolateral area
AM	Anteromedial area
AUDpo	Auditory cortex posterior area
DP	Dorsal posterior area
GU	Gustatory area
LM	Lateromedial area
MM	Mediomedial area
MOp	Motor cortex primary
P	Posterior area
PL	Prelimbic area
PM	Posteromedial area
RL	Rostrolateral area
RSPd	Rostroplenial area dorsal part
SSp-bfd	Somatosensory cortex primary barrel field
SSp-un	Somatosensory cortex primary unassigned
SSs	Somatosensory cortex secondary
V1	Primary visual area
VISC	Visceral area