A Traffic Analysis Perspective on Communication in the Brain

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Abstract— **In this short paper, we report on an approach to datamine the brain from a novel perspective, namely traffic analysis. Our datamining approach considers the brain regions and the tracts that connect them as a road network, and the signals traveling between them as vehicles. We analyze travel patterns by a process called traffic assignment. The results are unexpected in the sense that the movement of signals in the brain seems to follow some global optimization patterns as opposed to the anarchical system that would be favored by evolution.**

Keywords—Data mining; Connectomics; Transportation; Mobile Data Analytics; Brain Applications of Mobile Data

I. INTRODUCTION

Brain research has received a boost recently from the BRAIN initiative of the Obama administration ([1]). The data management community has also become interested in the subject, emphasizing graph mining (e.g. [2, 3]), and the performance improvement of database operations (e.g. [4]).

In this paper we propose a novel approach that combines datamining and transportation research. More specifically, we examine the brain in terms of demand for communication of signals[1] which move between neurons and brain regions, and the supply of fibers that enable, and serve the conduit for such movements. The objective of our demand/supply examination is to determine whether or not signal movement in the brain obeys some form of global design for optimum efficiency. Or, as evolution would suggest, movement patterns are emergent thus more anarchical, with various neurons and regions acting selfishly for the purpose of local optimization, regardless of the global perspective. The former would suggest a System Optimum (SO) signal movement, whereas the latter would suggest a User Equilibrium (UE) type of movement.

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[1]   Also called firings

Surprisingly, we have found that signal movement in the brain is closer to SO than to UE. This holds for healthy as well as depressed subjects. Nevertheless, the difference between the two is smaller in depressed subjects than in healthy ones. In other words, our analysis has two important implications: 1) it indicates some form of global control of communication traffic that is unexpected, and raises the question of how and by whom this control is exercised, and 2) it shows a distinction between healthy and depressed subjects that may be used as a diagnostic tool. Analysis of more subjects and finer granularities is desirable to confirm these results.

The data used in this paper is obtained by neuroimaging technology, and it represents the movement and traffic of signals in the brain. Although analogous to vehicular traffic, limitations of the technology do not allow capturing the movement of individual signals yet. Instead, aggregate movement is inferred from the level of activity of distinct brain regions. This is in contrast to common practice in the mobile data community, where mobile units (vehicles or pedestrians) are tracked by, for example, GPS receivers. Nevertheless, as we demonstrate in this paper, questions in an important area such as brain research can be addressed even when the available data is at a coarse level of granularity. Furthermore, human brain mapping is advancing at a rapid pace, and animal studies produce brain data at a finer level of granularity ([5]). In one such study, researchers performed discrete-time simulations on structural connectomes of macaque monkeys [6]. The authors showed that information flow in that particular connectome had "higher loss rates, faster transit times, and lower throughput, suggesting that neural connectivity may be optimized for speed rather than fidelity". These simulations relied on a random walk model of communication, in which the source and destination nodes of the signals were randomly selected. Then signals were propagated from source to destination by traveling to a neighboring node with equal probability. As such, the study did not incorporate information from a functional connectome, as is done in this paper.

In other relevant work, analysis that combines structural and functional connectomes has been described in [7]. Authors simulated dynamics within the brain using a susceptible–infected–susceptible model. They showed that the functional activity is linked to the underlying structure. Other works that

describe a link between the structural and functional connectomes include [8] and [9]. In [10], Daniel Graham discusses attempts at modeling communication in the brain, but references a fact that "very little work has been devoted to possible routing schemes in the brain". In particular, the question of whether communication in the brain follows optimum or equilibrium patterns has never before been addressed.

The data used in our experiments (see sec. IV) was produced at the University of Illinois at Chicago hospital (see [11] and [12] for details). However, data and software tools that are necessary to produce the necessary data structures for many more subjects have recently become available in the public domain as part of the Human Connectome Project (HCP) ([13]). According to [14]: "The goal of the Human Connectome Project is to build a "network map" (connectome) that will shed light on the anatomical and functional connectivity within the healthy human brain, as well as to produce a body of data that will facilitate research into brain disorders such as dyslexia, autism, Alzheimer's disease, and schizophrenia".

The rest of the paper is organized as follows. In section II, we provide the connectome model, and we explain how data about the connectome is gathered. In section III, we describe the transportation model. In section IV, we make the connection between the connectome and transportation models, describe our experiments, and present the results. In section V we conclude and discuss future work.

II. CONNECTOME MODEL AND DATA

In this section we first present an abstraction of the brain as a graph, and then we discuss the data structures that we use to estimate the graph, and the activity within it.

A *brain* is a graph $G = (V, E)$ where V is a finite set of nodes representing neurons, and E is a set of ordered pairs of nodes. A neuron communicates with other neurons by sending signals along the directed edges which represent synapses. So a signal that arrives at a neuron excites it, causing it to fire; which in turn excites a neighbor, causing it to fire, etc. In this sense, the signals travel along paths in the brain.

Time is divided into slots. Intuitively, the slot represents the resting, excited (i.e. sending a signal), refractory (i.e. recovering, during which it does not respond to signals) biological cycle. Let us assume for simplicity that the system is *synchronous* in the sense that the time periods are common to all the nodes[2]. A *Signal* (or a firing, which in this paper is analogous to a vehicle or a message) is an *(origin, destination, time-slot)* triple. The *origin* is a node, so is the *destination*, and the time slot is a natural number. Intuitively, it means that a signal arrives at its origin from outside the brain, in the given time-slot, and travels from its origin to its destination.

At the neuron-level granularity the human brain has on the order of 100 billion nodes, and 100 trillion edges [15]. Unfortunately, brain imaging technology cannot map individual neurons and monitor their signals even in animals (see [5]). Thus we use an approximation called the connectome in which each node is a contiguous 3-dimensional brain region, or volume, rather than an individual neuron. Then using neural imaging techniques we produced three data structures: 1) The structural connectome matrix, 2) the functional connectome matrix, and 3) the ALFF vector. Intuitively, these represent: 1) the brain regions and the signal-carrying capacity between each pair of regions, 2) the signal traffic between brain regions, and 3) the level of activity of each region in terms of signals that are generated in, or passing through, the region.

For the rest of this section we elaborate of these data structures, and how they are produced. Brain connectivity information is typically acquired via Diffusion Tensor Imaging (DTI) [16]. DTI is a magnetic resonance imaging technique that enables the measurement of the restricted diffusion of water in tissue. This technique produces neural tract images that are analyzed to calculate the number of fiber tracts that connect regions in the brain (see [17]).

Using the DTI images, a weighted undirected graph called the *structural connectome* is produced. In this graph the nodes are brain regions, and the weighted edges specify the strength of a connection between two brain regions; the higher the weight, the more signals can travel simultaneously between the two regions connected by the edge. The graph is incomplete in the sense that not every pair of regions is connected. In the structural connectomes used in this paper the human brain is parcellated into 87 regions (the nodes).

In neuroscience the structural connectome is usually represented by a square matrix, M (instead of sets of nodes and edges). Each element M_{ij} denotes the weight of the edge between the two regions i and j.

In neuroscience, signal activity information is gathered using *resting state fMRI* (rsfMRI or R-fMRI), which is a method of functional brain imaging. It is used to evaluate interactions between brain regions that occur when a subject is not performing an explicit task [16, 18]. This resting brain activity is observed through changes in blood flow in the brain. These changes generate a Blood-Oxygen-Level Dependent (BOLD) time series, that is measured using functional MRI.

We measured the BOLD time-series of the 87 regions of the structural connectome. From this time-series, we compute the activity level of the region using the Amplitude of Low-Frequency Fluctuation (ALFF) measure (see [11]). We use ALFF as a representation of the ground truth level of activity (i.e. number of signals produced) in each region.

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[2]   This is an abstraction commonly used in distributed computing systems, but is not strictly necessary for the purpose of this paper.
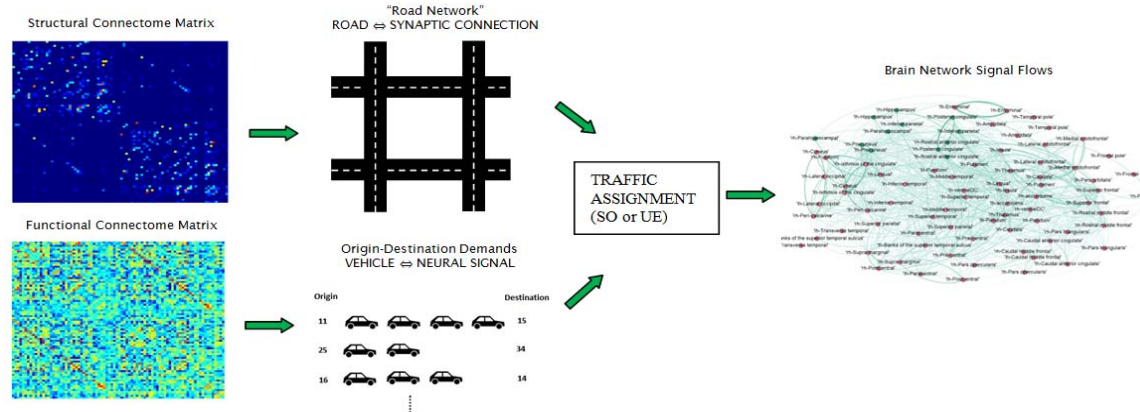
Fig. 1. Process of mapping brain data to a transportation network. First, structural and functional connectome matrices are mapped as roads and origin-destination travel demands, respectively. Then the resulting traffic is assigned to the road network using either System Optimal (SO) or User Equilibrium (UE) assignments. The result is a set of flow values for each edge in the connectome graph that connects brain regions.

The BOLD signal time-series data is also used to produce the *functional connectome*, F, which is a square matrix of the same order as M. Each element F_{ij} denotes the cross correlation between the time-series of region i and region j. We interpret the correlation as a proxy metric for the magnitude of communication (i.e., signals traveling)[3] between regions i and j (see fig. 1), in a way that is described in sec. IV.A. This concludes the description of the three data structures.

III. TRANSPORTATION PLANNING: UE AND SO ASSIGNMENTS

In order to achieve our objective of determining whether communication of brain signals follows a SO or a UE pattern, we employ approaches and tools from transportation planning [18][19]. Transportation planning manages travel supply-demand.

The supply is the road network, formally, a graph in which the nodes are the intersections and the edges are the road segments between two consecutive intersections. Each edge e is labeled by its *free-flow travel time*, t_e^{ff}, which is the time it takes for a vehicle to traverse the edge assuming no traffic and a specified maximum speed; and *capacity* c_e, which is the maximum number of vehicles per time unit that can exit this edge. Observe that c_e may be obtained at a speed v_e which is lower than the free-flow travel speed. For example, for an edge the max speed may be 60mi/hr, and at that speed at most 50 vehicles per minute may exit the edge; however, if the speed decreases due to an increased density of vehicles, the flow may increase to 100 vehicles per minute.

The demand for travel is usually given in the form of an Origin-Destination (OD) matrix. An OD matrix divides a metropolitan region into contiguous zones, and gives the travel demand between each pair of zones. For example, if on an average day 20,000 vehicles travel from region 20 to region 75,

then OD(20,75) = 20,000. *Traffic assignment* is the allocation of a route to each vehicle from its origin to its destination.

For a given traffic assignment, as the vehicles traverse the network on their way from origin to destination, for each edge at any time-unit there is a *density*, which is the number of vehicles per unit length of the edge. The *travel time* of the edge is the time it takes to traverse the edge and it depends on the density; the travel time is monotonically increasing with density. Also, for each edge e in the network at any time-unit there is a *flow* f_e, which is defined as the number of vehicles exiting the edge during the time unit. The flow is monotonically increasing with density up to a point, after which it decreases.

Given a road network and an OD matrix, each vehicle may have many route choices of going from origin to destination, and the shortest-distance path may not have the shortest travel time if it is heavily traveled[4].

Clearly, the higher the number of vehicles that are allocated to the same road link, the slower the travel time of each vehicle on that link. An *SO traffic assignment* allocates routes to vehicles such that the total travel time of all the vehicles is minimized. A *UE traffic assignment* allocates routes to vehicles such that no vehicle can unilaterally (i.e. if no other vehicle v changes v's route) improve its travel time by changing its route from its origin to its destination. It is assumed that normally traffic conforms to a UE assignment. Often mechanisms such as tolls and restrictions are used by transportation authorities to move traffic towards an SO assignment.

Given a road network and an OD matrix (i.e. given an input), it can be shown that UE and SO traffic assignments are often different. For example, [20], [21], and [22] provide examples of cases that result in different UE and SO

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[3]   If the firing of neuron i induces the firing of neuron j, which in turn induces the firing of neuron k, we say that a signal travels from i to k through j. And similarly if $i, j,$ and k, are brain regions rather than individual neurons.

[4]   We postulate that the same phenomenon occurs in the brain due to the fact that immediately after firing, a neuron enters a refractory state during which it cannot fire; thus signals may be delayed if many of them travel through the same region.

assignments. Intuitively, this means that if each traveler behaves selfishly by trying to optimize its own time, then the total travel time is suboptimal; and vice versa, if the total travel time is minimum, then some travelers can improve their individual travel time.

UE and SO traffic assignments are performed by formulating these as optimization problems [23]. These problem take as input: (1) the specifications of the road network, (2) the OD matrix, and (3) the *travel time function*, which maps the flow on each road network edge to a travel time. The common choice for the travel time function is a formula devised by the Bureau of Public Roads (BPR) [24]. The BPR formula is defined as follows:

$$t_e = t_e^{ff}\left[1 + \alpha\left(\frac{f_e}{c_e}\right)^{\beta}\right]$$

where for edge e, t_e is its travel time, t_e^{ff} is its free flow travel time, f_e is its flow, and c_e is its capacity. The α and β are parameters of the BPR formula, usually set to 0.15 and 4, respectively.

The standard algorithm for solving the UE and SO traffic assignment problems is the Frank-Wolfe method [25]. The method works by iteratively solving a linear approximation to an optimization problem until it converges [26, 27].

IV. Traffic Assignment Experiments

In our experiments, we used pairs of structural and functional connectome matrices[5] for 19 normal subjects and 19 subjects suffering from depression, and modeled these as transportation networks (see sec. IV.A.). We then performed UE and SO traffic assignments for each subject for different parameter settings, using an implementation of the Franke-Wolfe method. The assignment procedure produced flow values for each edge in the network for both UE and SO[6]. We then compared the UE and SO flow values, and selected parameter settings for which the overall difference between UE and SO was significant[7] (see sec. IV.B.). Lastly, we analyzed whether the BOLD signals flow in the actual brain network is closer to a UE flow or to an SO flow. This analysis is described in section IV.C.

A. Transformation of Connectome Matrices to a Transportation Network Instance

We scaled the normalized structural matrix M_{norm} by a scaling factor s_m, to get M':

$$M' = M_{norm} \cdot s_m$$

We used this scaled matrix to derive the road network as follows. Each positive matrix value M'_{ij} represents a road network edge going from node i to node j, with a capacity of

M'_{ij}. Zero values of M' were not used. The free-flow travel time of each edge was set to one[8].

The F matrix was then used as the OD matrix for the UE and SO traffic assignment computation[9]. We used the BPR formula as the travel time function. Since we set the free-flow travel time to one for all edges, and since we scaled the structural matrix, the travel time function became:

$$t_e = 1 + \alpha\left(\frac{f_e}{s_m c_e}\right)^{\beta} = 1 + \alpha s_m^{-\beta}\left(\frac{f_e}{c_e}\right)^{\beta}$$

In our experiments, we set $\alpha = 0.15$ as in [13], and we used different values of the scaling factor s_m (0.000001, 0.00001, 0.0001 and 0.001). We also used different values of β (1 through 4). We discuss the effects of varying these parameters in the next section.

B. Comparison of SO and UE Traffic Assignments for Different Parameter Values

We ran the SO and UE traffic assignment procedure for all subjects for different values of the structural matrix scaling factor s_m and different β values (see previous section). We then compared the results of UE to SO by computing the absolute value of the difference between UE and SO flow values, relative to SO flow, averaged over all edges and subjects:

$$\Delta UESO = \frac{1}{K}\sum_{i=1..K}\frac{1}{N_i}\sum_{j=1..N_i}\frac{|UE_{ij} - SO_{ij}|}{SO_{ij}}$$

where K is the number of subjects, N_i is the number of edges in the road network (i.e. structural connectome) of subject i, and UE_{ij} and SO_{ij} are the User Equilibrium and System Optimum traffic assignment flow values for edge j and subject i. The results are shown in fig. 2.
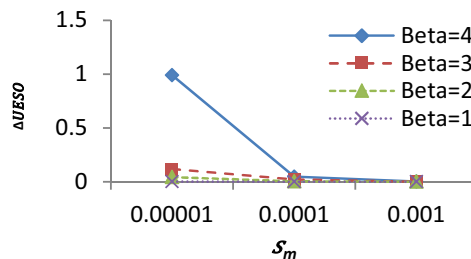

Fig. 2. Comparison of SO and UE traffic assignments using $\Delta UESO$, for different values of s_m and β.

In fig. 2 we see that the difference is maximum for smaller values of s_m and the difference approaches zero as s_m increases. This is intuitive because increasing the s_m factor increases the capacity of the entire network. In the extreme,

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[5]   The functional and structural matrices are 87 x 87 in size, with 87 being the number of brain regions.

[6]   The structural connectome represents an undirected graph, but the traffic assignment interprets each undirected edge as two opposing arcs, and assigns traffic to each one.

[7]   If the overall difference between UE and SO is small, then the question whether the actual flow is closer to UE or closer to SO is not very meaningful because the two ends of the spectrum are close to each other.

[8]   This is based on the assumption that if two nodes a and b are connected by an edge, then when direct signals are transmitted along the connection from a to b, their travel time is independent of the distance in the brain between nodes a and b.

[9]   The F matrix values that were negative were set to zero. These indicated functional anticorrelations and are usually removed (set to zero) in most studies [16].

there will be enough capacity to accommodate all the signals to travel along the shortest-distance paths, and the UE and SO assignments will be the same. Thus, in the experiments we used the 0.000001 scaling factor [10]. In other words, our objective is to find whether the traffic pattern in the brain is closer to UE or SO, assuming that there is a difference between the two. Therefore we are examining calibrations that produce a difference between the two types of traffic assignment, and this is the reason for the choice of scaling factor.

C. Comparison of SO and UE Assignments to Actual Activity

To compare the traffic assignments to actual brain activity, we measured the activity of each node using ALFF. We compare this measure with the computed UE and SO flows for each node, and find the diversion of both UE and SO flows from the ALFF values. Since the ALFF values are computed for each brain region (node), while UE and SO flow values are computed for each edge, we defined the notions of flow per region. This was done by summing up, for each region r, all the flow that originates at region r. Let UE_{rd} and SO_{rd} be the flow values for UE and SO, respectively, for the edge from region r to region d[11]. Then the UE and SO flows for each region r, is defined as:

$$UE_r = \sum_d UE_{rd} \qquad SO_r = \sum_d SO_{rd}$$

In other words, the UE flow for region r is the sum of the flows on the edges that are exiting from r. After computing the UE and SO flows per region, we normalize the UE flow per region, the SO flow per region, and the ALFF values, by dividing each by the subject's mean value. This makes each of the values dimensionless as they now represent ratios. This makes it possible to compare the UE and SO flows per region to the ALFF values. To make the comparison, we computed the Euclidean distance of UE flow per region to ALFF, and the SO flow per region to ALFF. The idea is that the flows with shorter distance to ALFF, which is a measure of the actual brain activity, represent the true signal-traffic activity within the brain.

The Euclidean distance measure for subject s, which we label as d^s, is defined as:

$$d^s(UE^s, \text{ALFF}^s) = \sqrt{\sum_{r=1,\dots,87} (UE_r^s - ALFF_r^s)^2}$$

for UE and

$$d^s(SO^s, ALFF^s) = \sqrt{\sum_{r=1,\dots,87} (SO_r^s - ALFF_r^s)^2}$$

for SO. In other words, we treat the ALFF, UE, and SO flows for each subject as 87-dimensional points, and compute the distances between ALFF and UE, and ALFF and SO.

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[10] Lower scaling values were tested, but the results showed a very large degree of variance, which indicates that due to low capacity the results are insignificant.

[11] Recall that the UE and SO assignments give a flow for each directed edge.

We performed the comparison for four different β values (1, 2, 3, and 4). For all β values, the average distance to ALFF for SO was smaller than for UE (see Table 1). A paired T-Test was used to verify that these results are statistically significant. For the healthy subjects the T-test gave p<0.032 for all β values, 1, 2, 3, 4; which means that the conclusion is unlikely to be due to the particular choice of subjects.

TABLE I.   DISTANCE TO ALFF VALUES FOR DIFFERENT ASSIGNMENTS, SUBJECTS, AND BETA PARAMETER SETTINGS.

Subjects	Assignment	Beta			
		1	2	3	4
Healthy	UE	6.515	8.839	9.054	8.348
	SO	6.514	8.801	8.957	7.660
With Depression	UE	6.664	8.892	8.712	7.712
	SO	6.663	8.859	8.612	7.562

For the depressed subjects, the average SO values were also smaller than UE, but the differences were small and for $\beta = 4$, shown to be insignificant (for the paired T-Test, p=0.54).

The largest differences between UE and SO flows occurred for $\beta = 4$ and these results are shown in figures 3 and 4, below.
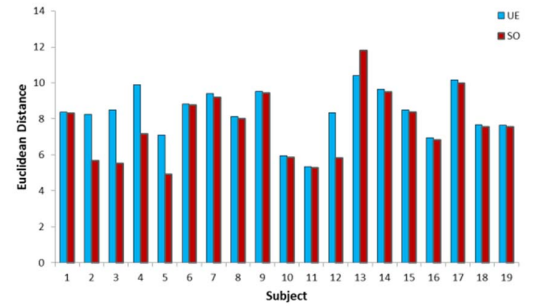
Fig. 3. Comparison of SO and UE traffic assignments to brain activity measured using ALFF, for each healthy subject.
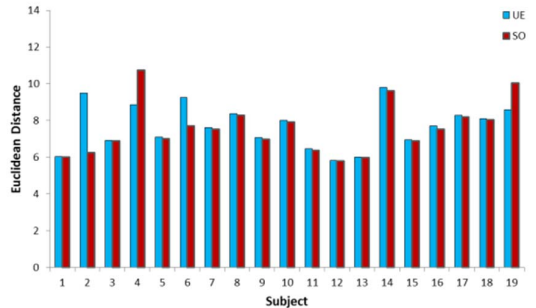
Fig. 4. Comparison of SO and UE traffic assignments to brain activity measured by ALFF, for each depressed subject.

As can be seen in fig. 3, in almost all cases, the distance between SO and ALFF is smaller than the distance between UE and ALFF. This suggests that the SO flows per region are closer to the actual measured brain activity. Therefore, it is more likely that the true signal transport activity within the brain is closer to System Optimum, than to User Equilibrium.

For the depressed subjects, the average distance was still smaller for SO than for UE, but the difference was insignificant. In particular, the average SO distance from ALFF was similar for healthy and depressed subjects; but the average UE distance differed substantially between healthy and depressed subjects. This suggests either that the routing of brain signals in depressed subjects is measurably different than in healthy ones, or that the structural connectomes are different. Determining which of these two options is true is the subject of future work.

V. CONCLUSION AND FUTURE WORK

In this paper we analyzed the communication in the brain using vehicular traffic tools. In particular, we were interested in the way the communication signals travel along the communication pathways in the brain. Traffic theory points to two fundamental assignments of vehicles to the road network: User Equilibrium and System Optimum. UE represents a state which lacks a global or centralized control, and in which vehicles act selfishly to minimize their travel time. Thus, in a UE state no vehicle can unilaterally deviate from the assigned route to improve its travel time. This anarchical situation should be contrasted with SO, which is analogous to a benevolent dictatorship that optimizes the global travel time. In an SO state, some individual vehicles can improve their travel time by rerouting; however, doing so would impede other vehicles by congesting their routes, and violating global optimality. Thus they are prevented by the dictator from such rerouting, i.e deviation from the assigned SO-route.

Our analysis of 38 subjects (connectomes) indicates that, given certain calibrations to bridge between traffic and neuroscience, UE and SO represent different states. This means that, for example, in the UE state, an edge (v,w), where v and w are brain regions, is traveled by many signals; whereas in the SO state edge (v,w) is traveled by few signals. Furthermore, communication in the brain is closer to an SO state rather than a UE state. This is surprising since the only means of communication in the brain that neuroscience knows are the signals (analogous to vehicles). Thus, in this model it is hard to see how individual signals can be controlled to achieve global optimality, and if so by whom. Is it possible that the global optimization is related to consciousness? A way to address this question is to repeat the experiments for comatose (i.e. unconscious) patients; these are left for future work. Furthermore, our experiments were performed on connectome data obtained from subjects in a resting state. Data is often obtained in other states, i.e. when subjects perform some task, and again, our results should be confirmed for those.

The techniques presented in this paper may be extendible to other types of networks that can be modeled in terms of transportation supply and demand. For example, the internet can be modeled as a transportation network carrying packets of data (vehicles) that flow on the physical communication (road) network. However, traffic flow on the internet is much better understood, and controlled, than the traffic of signals in the brain; thus this type of analysis is less interesting.

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