This talk studies how macroscopic spontaneous brain activity arises out of neural networks. It focuses on understanding how coherent oscillatory neural activity can arise spontaneously throughout the network through stochasticity in the transmission of information between neurons being filtered by the nonlinear neuronal dynamics.

It has long been known that in the absence of sensory stimulus, the brain spontaneously switches between periods of asynchronous quiescent ‘DOWN’ states, and active ‘UP’ states. These transitions have been observed in a variety of systems and conditions: including the primary visual cortex of anesthetized animals, during slow-wave sleep, and in cortical slices from mice. There have been two not-necessarily-incompatible proposed explanations for this switching: slow adaptation variables gradually altering the network state until a large jump occurs, or stochastic switching between bistable network states. It is known that the transitions tend to occur synchronously, and that the UP state often demonstrates synchronization that may attenuate in time.

Resting-state brain dynamics exhibits spontaneous bursting oscillations at a range of frequencies, particularly in the alpha frequency range of $8 - 10\, Hz$. The amplitude and duration of these oscillations are typically not fixed, but vary in a seemingly stochastic manner. Indeed evidence from EEG recordings suggests multistability: the low amplitude alpha rhythm can be characterized as diffusion about a fixed point, and the high amplitude alpha rhythm as a limit cycle. The variation in amplitude and duration of these oscillations can be seen through the fact that they are often distributed according to a power law.

Brain networks are distinguished by the complexity of the dynamics by which neurons communicate with each other. Briefly, the presynaptic neuron fires a pulse of electrical activity down its axon, which then passes through a synaptic cleft, and continues down the dendrites of the post-synaptic neuron. Crucially, the transmission of the spike through the synaptic cleft can be extremely noisy - basically due to the low copy numbers of the vesicles involved in the transmission process. Secondly, the transmission of information is not instantaneous, but is delayed through the relatively slow response of some aspects of the synaptic transmission. This slow response is often thought to be fundamental to the emergence of coherent network-wide oscillations, such as the well-known Gamma rhythm.

In most studies of dynamical systems on networks, the dynamics lies on the nodes, and the connections are merely a means for the nodes to instantaneously communicate with each other. By contrast, in the network to be analyzed in this talk, there is complex dynamics on both the nodes and the connections. The fundamental aim is to understand how this complex connection dynamics can shape emergent coherent oscillations in the entire network.

Model Construction

In the model, there is dynamics on both of the nodes and the connections.

- The neurons (i.e. the nodes in the network) are indexed by numbers between 1 and $5N$.
- Neurons with indices between 1 and $4N$ are stipulated to be excitatory, and neurons with indices between $4N + 1$ and $5N$ are chosen to be inhibitory.
- Connections between neurons are directed, and sampled randomly from a fixed probability distribution.
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- The internal dynamics of each neuron is excitable and governed by the Fitzhugh-Nagumo equation.
- Constants $\{A_{ab}^N\}_{a,b \in \{\,c,i\,\}}$ dictate the efficacy of the synaptic inputs: varying according to whether the neurons are excitatory or inhibitory, and the total number of neurons $N$. 
The inputted current from presynaptic neuron $k$ to postsynaptic neuron $j$ is given by $g^{jk}(t)$.

$$\frac{d}{dt} X^j(t) = G(X^j(t)) + H \left( \sum_{k \in E_j} A^N_{ab} g^{jk}(t) \right), \quad (1)$$

where the inputted current from neuron $k$, and the constants. The synaptic dynamics (on the connections) is modeled as

$$\frac{d}{dt} g^{jk}(t) = -\frac{1}{\tau_{ab}} g^{jk}(t) + h^{jk}(t) \quad (2)$$

$$h^{jk}(t) = h^{jk}(0) - \frac{1}{\tau_{ab}} \int_0^t h^{jk}(s) \, ds + S^{jk}(t), \quad (3)$$

- The variable $S^{jk}$ is defined to denote the number of times neuron $k$ successfully induced a postsynaptic response in the dendrite of neuron $j$. $S^{jk}$ is taken to be a $\mathbb{Z}^+$-valued Poisson counting process with a stipulated intensity function $\lambda_{jk}(X^k(t))$.
- The variables $g^{jk}, h^{jk}$ model the delayed postsynaptic response to the afferent inputs. It is generally thought that the delays are essential to emergent oscillations.
- Our reason for modeling the delays through introducing auxiliary variables, rather than an explicit delayed equation, is that it yields Markovian dynamics. This greatly facilitates the analysis.

**Analysis**

The linearity of the post-synaptic response allows one to separate the inhibitory and excitatory connections and sum the net afferent input on neuron $j$ to be

$$\tilde{g}^e_j(t) = \frac{1}{4N} \sum_{k \in [1,4N] \cap E_j} g^{jk}(t),$$

$$\tilde{g}^i_j(t) = \frac{1}{4N} \sum_{k \in [4N+1,5N] \cap E_j} g^{jk}(t),$$

$$\tilde{h}^e_j(t) = \frac{1}{4N} \sum_{k \in [1,4N] \cap E_j} h^{jk}(t),$$

$$\tilde{h}^i_j(t) = \frac{1}{4N} \sum_{k \in [4N+1,5N]} h^{jk}(t).$$

The scaling factors of $N^{-1}$ and $(4N)^{-1}$ have been inserted to ensure that these converge to $O(1)$ for large $N$. The central aim is to understand the emergent dynamics of the ‘empirical processes’ $\tilde{\mu}_{e,t}$ and $\tilde{\mu}_{i,t}$, i.e.

$$\tilde{\mu}^N_{e,t} = \frac{1}{4N} \sum_{j=1}^{4N} \delta(X^j(t), \tilde{g}^e_j(t), \tilde{g}^i_j(t), \tilde{h}^e_j(t), \tilde{h}^i_j(t)) \quad (4)$$

$$\tilde{\mu}^N_{i,t} = \frac{1}{N} \sum_{j=4N+1}^{5N} \delta(X^j(t), \tilde{g}^e_j(t), \tilde{g}^i_j(t), \tilde{h}^e_j(t), \tilde{h}^i_j(t)) \quad (5)$$

The empirical process is a device used in statistical mechanics to represent the collective behavior of the entire network. One can think of it as a frequency histogram, assigning mass of $1/N$ to each of the states of the neurons in the network.

In the large $N$ limit, the dynamics of the empirical process converges towards that of a PDE. Many of the coefficients in the PDE depend on integrals (over space) of the solution to the PDE. This PDE is often referred to as a population density. Next, a Large Deviations Principle is derived, giving the asymptotic probability (for large $N$ - the number of neurons in the system) of the system switching between the quiescent DOWN state and the oscillatory UP state. It yields a ‘rate function’: the minima of the rate function indicates the most likely states of the system. The ‘shortest path’ between two minima (as dictated by the rate function) indicate the path most likely followed by the system as it transitions between the state.

**Results**

- The effects of synaptic depression and synaptic facilitation on the spontaneous switching between the different states is explored. Synaptic depression is a decrease in the probability of a particular synapse transmitting signals, due to a lot of previous activity.
- The results are also applied to study orientation-tuning patterns in the visual cortex.