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Mathematical Model of Neuron Flows and Structures in the Brain

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Abstract

In this project, we investigate the use of a Bratteli diagrams as a structural model for neural activation in the brain. Proposed by Prof. Anthony Dooley, preliminary discussions suggest that the successive firing of connected neurons can be modelled by this kind of non-homogeneous Markov measure. Using measures on Bratteli diagrams to quantify neural activity and the flow of information in the brain. We attempt to provide a mathematical point of view for some big questions in neuroscience and how they relate to modern technologies such as fMRI's. The use of directed graphs and measure theory could potentially yield new insights into brain activity from this model of the behavior of neurons.

Introduction

One recent question in biology is explaining the mechanism of neural structures between neuroscience and behavioral therapy.

In this paper we attempt to bridge the gap between the two disciplines and provide a mathematical model how neurons are structured and rewired in the brain. By applying a measure to the path space of a directed graph we can get some understanding of how different activity and stimuli change the measure of neural activation.

In recent years, with the advancement of mathematics and neuroscience, stochastic models of single or a handful of neurons have been presented but large-scale mathematical models of neural structures in the brain is minimal. The Bratteli Diagram is used to a provide 2-dimensional model of neural circuits with implications of brain function and nervous system bodily functions. Directed graphs have been used to model neurons with neurons as vertices and edges as synaptic connects directed from the presynaptic to postsynaptic neurons. Reimann et al. [1] used algebraic topology [2] through directed graphs to analyze neuron activity. We will use Bratteli Diagrams, a type of directed graph and measures to model this activity.

Chapter 1

Neuroscience

The brain is made up of billions of neurons. Authors Williams and Herrup estimated the number of neurons in the human brain by compilation of partial numbers in the literature. They estimated the human brain to have 85 billion neurons, with 12-15 in the telencephalon [3], 70 billion in the cerebellum, as granule cells [4] , and fewer than 1 billion in the brainstem [5]. More recent estimates of the cerebral cortex increased that number to 21-26 billion neurons [6] and 101 billion neurons in the cerebellum [7], however, this would increase the total number of neurons in the human brain to over 120 billion neurons [8].

The generic neuron is comprised of several structures. The body or nuclei of the neuron is called the *soma*. *Dendrites* are the structure in which the neurons receive most of their information. Branch-like in structure; these dendrites are equipped with receptors that pick-up signals from other neurons which are of the form of chemicals called *neurotransmitters*. These signals from the neurotransmitters produce electrical charges in the neurons which are interpreted in the soma. The soma then processes and interprets these signals, then assembles this information in a structure known as the *axon hillock*. If the signal from the dendrites is strong enough, the electrical signal is sent through to the axon. The electrical signal is then called an action potential. The *axon* is a long tube-like structure which is covered by *myelin*, an insulator like material that helps prevent the signal from degrading. At the end of axons are the *axon terminals* or *synaptic boutons*. When the signal reaches the syntactic boutons, it can cause the release of neurotransmitters. At the other end of a synaptic bouton are dendrites from another neuron which pick up those neurotransmitters and the process repeats itself.

The *synapse* is an area of the neuron that allows communication to another neuron. The neuron where the signal is initiated is known as the *presynaptic* neuron and the recipient neuron is called the *postsynaptic* neuron. Located at the ends of the axon terminals, the presynaptic neuron releases neurotransmitters into the *synaptic cleft*, the space between the two neurons approximately 20 nanometers wide [9]. The neurotransmitters then bind to the receptors located on the dendrites of the post synaptic neuron.

Neuroplasticity

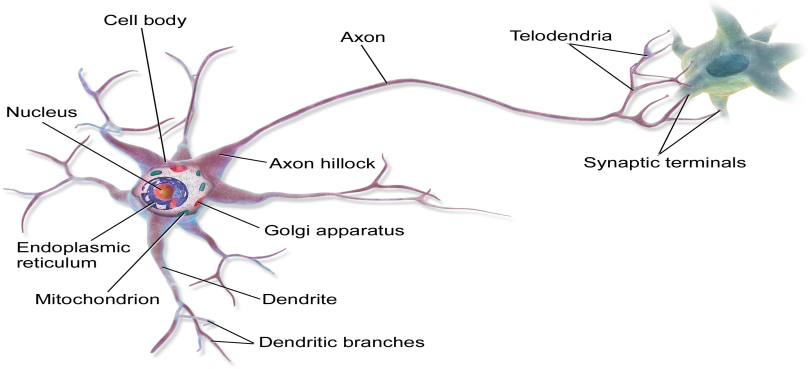

Figure 1: A generic neuron [10]

Neuron plasticity is the ability for neurons in the brain to change throughout an individuals life. This means that neural circuits in the brain are not hard wired and can be subject to rewiring in response to training or injury. The underlying basis of this principle is based on the idea that synaptic connections are constantly being removed or created. Draganski et al. showed that training induced stimulus made selective structural changes in brain areas that are associated with processing and storage of complex visual motion. We find that these individuals show a transient and selective structural change in brain areas that are associated with the processing and storage of complex visual motion [11].

Two types of neural plasticity can occur, *synaptic* or *structural* neural plasticity. Synaptic neural plasticity is the process when cells change at the level of the synapse. This change can happen in several ways; i.e. the change of the amount of neurotransmitters released by the synaptic neuron, the number of neural receptors in the target neuron or type of neural receptor in the target neuron.

Structural neural plasticity is the process when the structure of the cell changes. The changes in the total number of synapses between two neurons. This can be described as the growth (sprouting) or lose (pruning) of axon terminals and dendrite spines from the pre-synaptic neuron and post synaptic neuron respectively. [12]

Overall, **potentiation** is the strengthening of neural pathways (synaptic or structurally) over time and **depression** is the weakening of neural pathways (synaptic or structurally) over time. Neuroplasticity can happen over milliseconds to minutes (short term) or minutes days, months and years (long term). Structural neuroplasticity tends to occur over long-term periods of time and synaptic neuroplasticity tends to occur over both long and short periods of time [13].

Neuroplasticity is the basis of all behavioural therapies and occurs in many instances of learning a new skill or being in a new situation. This is particularly emphasized in developing brain, although adult brains also exhibit neuroplasticity. A familiar example is the increased amount of grey matter in certain areas of the brain in individuals who practice long-term meditation [14]. This aspect of the brain is crucial when providing a mathematical model of how neurons are structured. Representing how neurons are rewired and new circuits

Neurogenesis

First recognized in the 1960s and substantial researching in the 1990s, *neurogenesis* is a process by which neurons are produced by neural stem cells. The ability for a brain to produce new neurons occurs in almost all species of animals. In the human brain, one paper estimates 700 new neurons are added to the each hippocampus every day which corresponds to a annual turnover of about 1.75% of the neurons within the renewal fraction, with a moderate decline with age [15].

Neurogenesis also plays a major part in keeping a fit brain. "Use it or lose it" by Authors Shors, Anderson et al. (2012) showed that engaging in certain activities will not only increase neurogenesis but also how new neurons are kept alive and integrated into neural circuitry by "effortful learning, a process that involves concentration in the present moment of experience over some extended period of time". On the other hand, cells will die unless they are engaged in some sort of effortful learning when new cells are approximately one week of age. "Concurrent and synchronous activity provides a mechanism whereby the new neurons become integrated with the other neurons. This integration allows the present experience to become integrated with memories from the recent past in order to learn and predict when events will occur in the near future. In this way, neurogenesis and learning interact to maintain a fit brain." [16]

Literature has become extensive on neurogenesis of late, and much more can be said about neurogenesis and neuroplasticity and the mechanisms which explain them, but the integration of these processes will be crucial when modeling the neural structures of the brain.

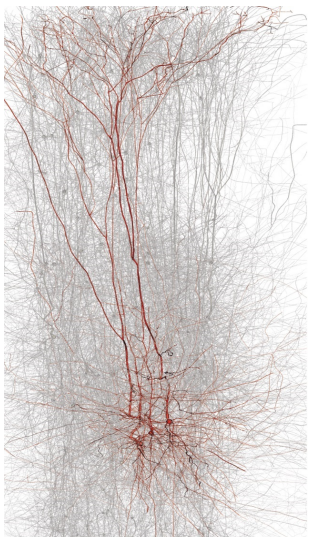

Figure 2: A reconstructed microcircuit produced using the model of neural activity. A 5-neuron clique is shown in red [1].

Chapter 2

Properties of the Bratteli Diagram

We define properties on the Bratteli Diagram that are ubiquitous in the literature. The Bratteli Diagram was first introduced by Ola Bratteli in 1972 [17]. This diagram is combinatorial in structure with Vertices at level n and edges connecting the vertices n to vertices $n+1$. We say $B = (V, E)$ is a Bratteli diagram with Vertices $V = \{V_n \mid n \in N\}$ and Edges $E = \{E_n \mid n \in N\}$. The vertex set $V = \coprod_{n=0}^{\infty} V_n$ and edge set $E = \coprod_{n=0}^{\infty} E_n$ are both countable disjoint unions of non-empty finite sets [18]. Properties of the Bratteli Diagram [19]:

- Let $B = (V, E)$ be a Bratteli Diagram
- The first vertex V_0 is a singleton $\{v_0\}$ at level $n = 1$
- The source map $s : E_n \to V_n$
- The Range map $r : E_n \to V_{n+1}$
- Let X_B be the is the set of all infinite paths starting at v_0
- The Bratteli Diagram B has finite rank if $|V_n| \leq k,\ k \in N$

Note: A Bratteli Diagram with defined ranged and source maps are sometimes given by the quadruple (V, E, r, s)

A Bratteli Diagram is *simple* for any level n, there is an $m > n$ such that each pair of vertices $(v, w) \in (V_n, V_m)$ is connected by a finite path [4]. I.e. every vertex at level n is connected to another vertex at level $n + 1$ by at least one edge [19].

A Bratteli Diagram $B = (V, E)$ with Edges E_n can be represented by a $|V_n| \times |Vn - 1|$ by a *incidence matrix* $F_n = (f_{ij})$. If all incidence matrices for all n are the same, B is called *stationary*, $F_n = F_1, \ \forall n \geq 2$ [19].

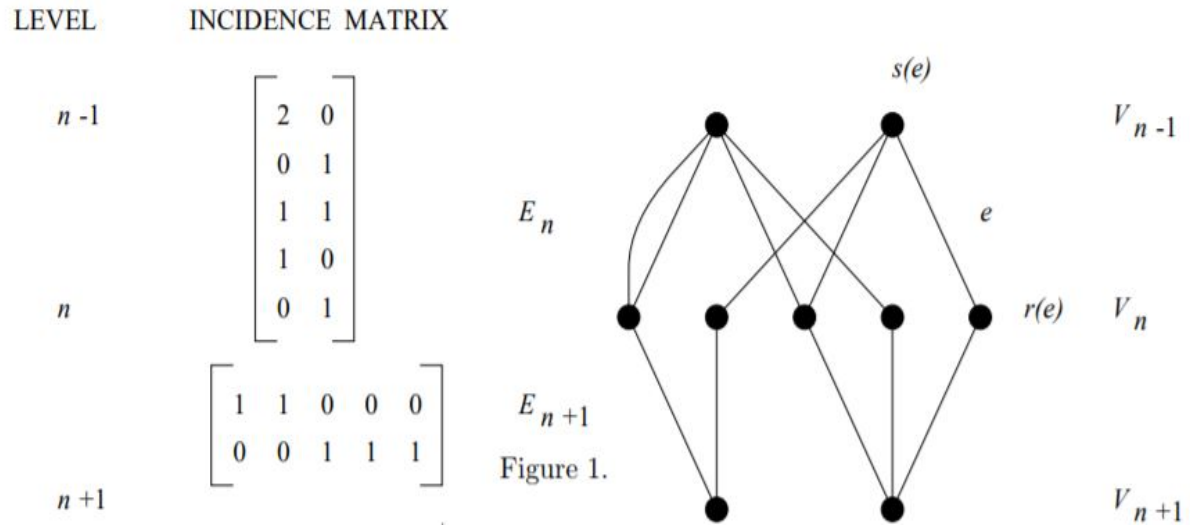

Figure 3: Example of a Bratteli Diagram [20]

Let $\alpha = (e_1, e_2, ..., e_k)$ be a finite path of X_B starting at v_0 and ending at some v_n. $(v_n \in V_n : n \in \mathbb{N})$. These finite paths are called cylinder sets. Formally, we topologize X_B by giving a basis of open sets, cylinder set [18]

$$U(\alpha) = U(e_1, e_2, , e_k) = \{(f_1, f_2,) \in X_B \mid f_i = e_i, 1 \leq i \leq k\} \tag{1}$$

Telescoping

Given a Bratteli Diagrams $(V, E, \geq)$ and let $m_0 < m_1 < m_2 < \dots$ be a sequence of non-negative integers. The telescoping of $(V, E, \geq)$ with respect to the sequence m_n is labelled a Bratteli Diagrams $(V, E, \geq)$. Where $V'_n = V_{m_n}$ and $E_n = E_{m_{n-1}+1} \circ E_{m_n+2} \circ \circ E_{mn}$. In other words, by multiplying the incidence matrix F_m by F_{m+1}, another Bratteli Diagram $(V, E, \geq)$ will be obtained with the same number of vertices on the first and last level of $(V, E, \geq)$, but different Edges connecting them. The inverse of this operation is called *microscoping* [18].

Ordered Bratteli Diagrams

We say an Ordered Bratteli Diagram $B = (V, E, \geq)$ is a Bratteli Diagram if we assignment a linear order '$\geq$' to the edges E_n . Naturally, the path X_{max} is the infinite path from all the maximum orderings of the edges. The converse is true for X_{min}. If (V, E) is simple then $X_{max} \cap X_{min} = \{\emptyset\}$ [18].

There is an isomorphism between Bratteli Diagrams (V, E) and (V, E), or a pair of bijections $f : V \to V$ and $g : E \to E$ if $fr = rg$ and $fs = sg$. This isomorphism preserves labeling and interweaving between range and source maps. In other words, there is changing the vertices within each level with keeping their labels and edges [18].

Markov Odometers [21]

Let $l_i \geq 2$ be a sequence of integers and the infinite product space $X = \prod_{i=1}^{\infty} \mathbb{Z}_{l_i}$ and we write $\mathbb{Z}_{l(i)} = \{0, 1, 2, \dots, l(i) - 1\}$. Also let $X_m^n = \prod_{i=m}^{n} \mathbb{Z}_{l(i)}$ with $X^n = X_1^n$. We denote $|X^n| = \prod_{i=1}^{n} l(i)$.

We assume X is of *bounded type* if there exists m such that $l(i) \leq m$ for all $i \in N$. We can define cylinder sets similar to that of 2.1 which generate standard σ-algebra B on X. An *Odometer* T acts on X by $Tx = y$ where y is the smallest element greater than x in a lexicographic order. If the path $l = (l(1), l(2), \dots, l(n), \dots)$, then $T(l) = 0 = (0, 0, 0, \dots)$.

We choose a probability measure μ_i on each coordinate space $\mathbb{Z}_{l(i)}$ when a weights of the edges $\mu_i(\{\alpha\})$ sums to 1. Suppose X is equipped with the usual infinite product measure $\mu = \bigotimes_{i=1}^{\infty} \mu_i$.

This is the most basic Odometer $X = \prod_{i=1}^{\infty} \{0, 1\}^{\mathbb{N}}$ with two edges between every vertex and probability α on the edges. Equipped with the probability product measure μ, a transformation T can be defined as a *finite coordinate change* of a path on the Odometer. This takes a non-maximal edge and maps it to the first successor edge, or maps the maximal edge to the minimal edge. The transformation T changes the measure μ of each path, provided $\alpha \neq .5$. For example:

$T(1, 0, 1, 0, 0, 1, \dots) = (0, 1, 1, 0, 0, 1, \dots)$
$T(0, 1, 1, 0, 0, 1, \dots) = (1, 1, 1, 0, 0, 1, \dots)$
$T(1, 1, 1, 0, 0, 1, \dots) = (0, 0, 0, 1, 0, 1, \dots)$

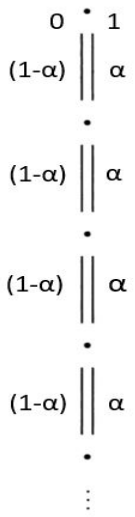

Figure 4: Basic {0,1} Markov Odometer with probabilities α

Dooley and Hamachi (2003), proved that any non-singular dynamical systems, Bratteli diagrams and Markov odometers [21].

Vershik Map [19]

The *Vershik transformation* φ maps the first non-maximal edge in a path on X_B to its successor edge, and if the path is the maximum, it maps X_{max} to X_{min}.

This generalized the notion of Markov Odometers to Bratteli Diagrams by having more than one vertex at each level.

More formally, the Bratteli-Vershik Diagram is a Bratteli Diagram with a topologized dynamical system and transformation called the Vershik Map acting on its path space [ref]. We introduce a transformation φ_B: $X_B \to X_B$ where B is a ordered Bratteli Diagram $B = (V, E, \geq)$ if it satisfies the following three condition:

1. φ is a homeomorphism on X_B
2. $\varphi(X_{max}(w)) = X_{min}(w)$
3. If $x = (e_1, e_2, ...) \in X_B$ and xX_{max}, then let k be the smallest integer such that e_k is not maximal. Let f_k be the successor of e_k such that $r(e_k) = r(f_k)$ and let $(e_1, ..., e_{k-1})$ be the unique minimal path from v_0 to $s(f_k)$. Then,

$$\varphi_B(e_1, e_2, ...) = (e_1, ..., e_{k-1}, f_k, e_{k+1}, e_{k+2}, ...)$$

Bratteli Vershik Systems are usually denoted as (B, φ) with $B = (V, E, \geq)$

Given two infinite paths, $x, y \in X_B$ we say that x and y are *cofinal* or *tail equivalent* if there is an $N \in \mathbb{N}$ such that $x_k = y_k$ for all $k \geq N$, in other words, the tails of the paths are the same from a certain point on. Observe that if xX_{max}, then x and $\varphi_B(x)$ are cofinal or tail equivalent. [1]

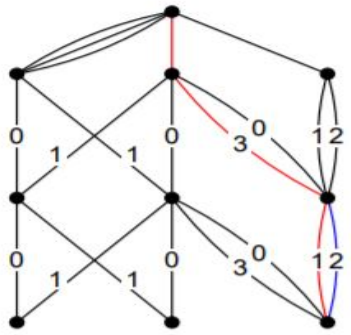

Figure 5: Example of a Vershik Transformation on a Bratteli Diagram [22]

Applying a Vershik transformation (blue) to the cylinder set (red) maps the red edge, non maximal to the blue edge (successor)

We define a full group of finite coordinate changes [21]

$$P_k^0(v) = (e_i) \in X : r(e_k) = v \tag{2}$$

with each $P_k^0(v)$ as a totally ordered set. We can also define a cyclic transformation $S = S_k$ on P_k^0 by:

$$S(x_1, \ldots, x_k) = (y_1, \ldots, y_k) \tag{3}$$

where, if r is the least integer such that x_r is not maximal, the elements $y_1, \ldots, y_{r-1}$are minimal, y_r is the successor of x_r and $(y_{r+1}, \ldots, y_k) = (x_{r+1}, \ldots, x_k)$. If all x_r are maximal, then we take all the y_r to be minimal.

If we extend S_k to a transformation on the subsets

$$\{(x_1, \ldots, x_k, x_{k+1}) \in P_{k+1}^0 : (y_1, \ldots, y_k) \text{ is not maximal}\} \tag{4}$$

by letting

$$S_k(x_1, \ldots, x_k, x_{k+1}) = (S_k(x_1, \ldots, x_k), x_{k+1}) \tag{5}$$

Then it coincides with S_{k+1} on that subset. The Vershik Transformation $\varphi = S_k \in P_k^0$.

We denote the cyclic group Z_k of finite k coordinates generated by S_k. Let $Z = \bigcup_{k=1}^{n} Z_k$ and we denote $Z_k(v)$, $v \in V^{(k)}$ with the orbit $\{\zeta x : x \in P_k^0$ with $r(x) = v$ and $\zeta \in A_k\}$ [21].

Measures on a Bratteli Diagram [21]

We say that a matrix

$$P^{(n)} = \{P^{(n)}_{(v,e)}\}_{(v,e)\in V^{(n-1)}\times E^{(n)}} \tag{6}$$

is a **stochastic matrix** if it satisfies the following two conditions:

(i) $P^{(n)}_{v,e} \geq 0 \Leftrightarrow s(e) = v$

(ii) $\sum_{\{e\in E^{(n)}:s(e)=v\}} P^{(n)}_{v,e} = 1, \forall v \in V^{(n+1)}$

Given a sequence $P^{(n)}$ of stochastic matrices and a probability measure on ν_0 on V_0 such that

$$\nu_0(v) > 0, \forall v \in V^{(0)} \tag{7}$$

We define a measure μ on the cylinder sets by

$$\mu([e_1, e_2, \dots e_n]_1^n) = \nu(s(e_1))P^{(1)}_{s(e_1),e_1}P^{(2)}_{s(e_2),e_2}\cdots P^{(n)}_{s(e_n),e_n} \tag{8}$$

This measure is called a **Markov Measure** and gives the dynamical system $(X, \mathcal{B}, T, \mu)$

Chapter 3

Modeling

Using a *finite* properly ordered Bratteli Diagram $B = (V, E, r, s, \geq)$, we say each vertex represents a neuron. The connections between these neurons, i.e. axon terminals, the release of neurotransmitters, and receptors on the dendrites of the post synaptic neuron are represented by the edges of the vertices from level n to $n+1$. As stated in the introduction, directed graphs have been used with some success to model neural flows in the brain. The arrows in the diagram give an idea about how information is flowing through neurons in the brain. Introducing the Bratteli Diagrams with vertices as neurons, edges as connections between neurons and assigning subsets of neurons to levels $(n, n+1, n+2, \dots, n+m : m \in \mathbb{Z})$ gives a naturally ordered structure to these neural circuits. Assigning probabilities to the edges of neurons from level n to $n+1$ gives an idea about the activity of certain paths. The

first vertex v_0 represents a sensory neuron, which picks up a stimulus and activates a path down the Bratteli diagram. Just like the probabilities measures μ on Markov Odometer, we will use this measure to analyze activation of neural pathways.

Full group of finite coordinate changes

Once a stimulus ζ_i arrives, i.e. picked up by the sensory neuron v_0, neural activity is modelled by a set of cylinder sets

$$C_i = \{\bigcup U(\alpha_n) \subset X_B \mid \alpha_n = (e_1, e_2, ..., e_k) : i, k, n \in \mathbb{N}\} \tag{9}$$

Indexed cylinder sets α_n make up the set of cylinder sets C_i starting at v_0 and ending at differing v_n. These are different paths being "lit up" when a stimulus arrives. We can apply transformations to cylinder sets to get a realistic model of neural activity.

As a new stimulus arrives, the cyclic group of finite coordinate changes Z_k will show how paths of the Bratteli Diagram are changed. We take a specific finite coordinate change of a cylinder set $U(a_n)$ from the group Z_k. The union of finite coordinate changes of each cylinder set $U(a_n) \in C_i$ will represent a new set of cylinder sets C_k, i.e. the change of neural activity in the brain.

Synaptic Neuroplasiticity

We use the time dependent stochastic matrix $P_n^{(t)}$ to model synaptic neuroplasicity. This will describe the increase or decrease in probabilities of edges of the Bratteli Diagram over time (potentiation and depression). We relax the constraint $\sum P_{v,e}^{(n)} = 1$ because each level n will have potentially millions if not billions of neurons at each level. As the matrix $P_n^{(t)}$ evolves over time, probabilities on the millions of edges between each vertex change. The set of null edges (edges with 0 probability) will change to 'activated'edges with non-zero probabilities and 'deactivated 'edges will go from a non-zero probability to 0 probability.This is analogous to circuits in the brain being created and destroyed (potentiation and depression).

How can we determine the change of probabilities on each edge associated with a response to a stimulus or activity?

We define two sets of all bounded real-valued transformation matrices:
1. Let $\{L_{v,e}^{(n)}\}$ be $m \times n$ matrices associated with each corresponding stochastic matrix $P_{v,e}^{(n)}$.

2. Let $\{R_{v,e}^{(n)}\}$ be $i \times j$ matrices associated with each corresponding stochastic matrix $P_{v,e}^{(n)}$.
Note: $P_{v,e}^{(n)}$ does not have to be square

Due to the non-commutative native of matrices, if $L_{v,e}^{(n)}$ is an $m \times n$ and $P_{v,e}^{(n)}$ is an $n \times k$ matrix with $m \neq k$ then by *left* multiplication of $L_{v,e}^{(n)} \cdot P_{v,e}^{(n)}$, the addition or deletion of rows explains edges being created or destroyed from a vertex at level n to connected vertices at $n+1$.

Right multiplication $P_{v,e}^{(n)} \cdot R_{v,e}^{(n)}$ can be define similarly, and gives rise to the addition or deletion of new columns. This can explain the mechanism of neurogenesis when adding edges to a new neuron (column) or destruction of circuits when a column is deleted.

This describes the changes of probabilities of edges leave each vertex in response to some stimulus or training activity over-time.

Note: if $m = n$ or $i = j$ and the transformation matrices $\{L_{v,e}^{(n)}\}$ and $\{R_{v,e}^{(n)}\}$ are invertible, then we can define them as the General Linear Group $GL_n(\mathbb{R})$.

We say the brains *response* to some activity T_k is a collection of $\coprod\{L_{v,e}^{(n)}\}$ and $\coprod\{R_{v,e}^{(n)}\}$ matrices such that they correspond to the collection of stochastic matrix $P = \coprod\{P_{v,e}^{(n)}\}$ at every vertex of B.

Time Dependent System

The Markov (memoryless) property perfectly describes the idea of "Use it or lose it" by Authors Shors, Anderson et al. (2012)

$$P(X_n = x_n | X_{n-1} = x_{n-1}, \ldots, X_0 = x_0) = P(X_n = x_n | X_{n-1} = x_{n-1})$$

We define an *evolving* Markov Chain $\{X(t)\}$ with finite states as $(P(i), (T_1P(i)), (T_2P(i)), \ldots (T_kP(i))$. Here, the new stochastic matrix $P(i)$ after each jump of the Markov chain where $i \in (0, 1, 2, \ldots n)$. This can be thought of as an *evolving* Markov Chain with collection of matrices P being multiplied by T_i when a jump occurs.

The stationary transition matrix (when a jump occurs)

$$P(i) = \begin{pmatrix} p_{11}(P(i)) & p_{12}(T_1P(i)) & \ldots & p_{1,n}(T_kP(i)) \\ p_{21}(P(i)) & p_{22}(T_1P(i)) & \ldots & p_{2,n}(T_kP(i)) \\ p_{31}(P(i)) & p_{32}(T_1P(i)) & \ldots & p_{3,n}(T_kP(i)) \\ \vdots & \vdots & \ddots & \vdots \\ p_{m1}(P(i)) & p_{m2}(T_1P(i)) & \ldots & p_{mn}(T_kP(i)) \end{pmatrix}$$

with $p_{ii} = 0$

This Markov chain describes when we apply a transformation T to the stochastic matrix P. This can be thought of as when preforming a stimulus/activity, how the brain changes (learns) in response to that activity which is the transformation $T_k P(i)$.

The generator matrix Q is the time until the transition out of state i. The waiting times are independent have are exponentially distributed

$$T_i \sim Exp(\nu_i)$$

. where the parameter $\nu_i \geq 0$ and $\mathbb{E}(T_i) = a_i = \frac{1}{\nu_i}$.

$$Q_{nm} = \begin{pmatrix} -a_1 & a_1 p(i)_{1,2} & a_1 p(i)_{1,3} & \dots & a_1 p(i)_{1,m} \\ a_2 p(i)_{2,1} & -a_2 & a_2 p(i)_{2,3} & \dots & a_2 p(i)_{2,m} \\ a_3 p(i)_{3,1} & a_3 p(i)_{3,2} & -a_3 & \dots & a_3 p(i)_{3,m} \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ a_n p(i)_{n,1} & a_n p(i)_{n,2} & a_n p(i)_{n,3} & \dots & -a_n \end{pmatrix} \tag{10}$$

The rows of Q sum to 0,

$$\sum_{m=1} q_{nm} = 0$$

The waiting time T_i is the waiting time until engaged with some activity or stimulus.

We define an orbit of the Transformation T_i as the sequence

$$Orbit_{T_n} = \{T_{k_1}(P(0)), T_{k_2}(P(1)), \dots, T_{k_i}(P(n)) \ | k_i = (k_1, k_2, \dots, k_i) \in \mathbb{Z}_0^+\}$$

Note: The Transformation T_k cannot be the same for two consecutive jumps $P(i)$ and $P(i+1)$.

One interesting question arises when talking about orbits, when does the $Orbit_{T_{k_i}} = Orbit_{T_{p_i}}$ for some k, p? In words, what stimulus/ learning activity provides the same neural changes in the brain?

Structural Neuroplasticity

With billions of neurons in the brain, Peter R. and Huttenlocher in 2003 showed synaptic density was constant throughout adult life (ages 16-72 years) with a mean of 11.05×10^8 synapses/cu.mm $\pm$

0.41 S.E.M. A slight decrease in synaptic density in brains of the ages (ages 74-90 years) with a mean of 9.56×10^8 synapses/cu.mm $\pm$ .28S.E.M. in 4 samples ($P < 0.05$). Human cerebral cortex is one of a number of neuronal systems in which loss of neurons and synapses appears to occur as a late developmental event [23].

Obviously, all matrices associated to these processes are very large, to represent the scale of neurons in the brain.

This is mirrored in the time-dependent incidence matrix $F_n^{(t)}$. With around 1 billion synapses per cubic millimeter, the entries for the incidence matrix $F_n^{(t)}$ will potentially be very large. $F_n^{(t)} = (f_{ij}^{(t)})$ will be the incidence matrix at time t from $V_n \to V_{n+1}$.

This is mirrored in the incidence matrix $F_n^{(t)}$

$$F_n^{(t)} = (f_{ij}^{(t)}) = \begin{pmatrix} f_{11}^{(t)} & f_{12}^{(t)} & f_{13}^{(t)} & \cdots & f_{1j}^{(t)} \\ f_{21}^{(t)} & f_{22}^{(t)} & f_{23}^{(t)} & \cdots & f_{2j}^{(t)} \\ f_{31}^{(t)} & f_{32}^{(t)} & f_{33}^{(t)} & \cdots & f_{3j}^{(t)} \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ f_{i1}^{(t)} & f_{i2}^{(t)} & f_{i3}^{(t)} & \cdots & f_{ij}^{(t)} \end{pmatrix}$$

By looking at the two incidence matrices at different time, this can explain how edges between vertices change, or how structural neuroplasiticity is at play.

Note: This incidence matrix indexed with discrete time t only shows edges of probability > 0 between vertices.

Evolving measures and Bratteli Diagrams

As seen from above, the brain is a sort of dynamical system. As the stochastic matrices change over-time, the measure μ changes. By defining a new measure, we can take into account neuroplasticity and neurogenesis i.e. how neurons and inter-neural connections change over-time. With the transformation of stochastic matrices, probabilities on the edges of the Bratteli Diagram B change accordingly. We define the time-dependent finite product measure $\mu^{(t)} = \bigotimes_{i=1}^{n} \mu_i^{(t)}$. Taken at discrete times, the measure $\mu^{(t)}$ shows the the change of probabilities overtime.This gives the system $(B, \mu^{(t)})$. We assign the time-evolving Bratteli Diagram $(B, \mu^{(t)})$ as neurons are created, destroyed and how connections between neurons change over time. $(B, \mu^{(t)})$ will be the basis of our model, with $\mu^{(t)}$ representing the time-dependent measure of the measure space X_B.

Radon-Nikodym Theorem [24]

Given two σ-finite measures μ and ν on a measurable space $(X, \mathcal{A})$, we say $\nu \ll \mu$ (ν is absolutely continuous with respect to μ) if there exists a measurable function $f : X \to [0, \infty]$, such that

$$\nu(\mathcal{A}) = \int_{\mathcal{A}} f d\mu, \quad \forall A \in \mathcal{A}$$

The function f is called the *Radon-Nikodym derivative*

$$f = \frac{d\nu}{d\mu} \tag{11}$$

If the R-N derivative exists then $\mu(A) = 0 \iff \nu(A) = 0$

Kakutani's Theorem [25]

The Radon-Nikodym Theorem leads to another important result of measure theory, *Kakutani's theorem.* It gives if and only if conditions to determine is two countable product measures are equivalent or mutually singular.

For each $n, k \in \mathbb{N}, n \neq k$, given two probability measures μ_n and ν_n on $\mathbb{R}$. Let $\mu = \bigotimes_{n\in\mathbb{N}} \mu_n$ and $\nu = \bigotimes_{n\in\mathbb{N}} \nu_n$ are product measure on $\mathbb{R}^\infty$ and let μ_n and ν_n be equivalent, $\mu_n \sim \nu_v$ (i.e. have the same null sets) for every $n \in \mathbb{N}$. The two measure μ and ν are said to be equivalent if the infinite product series

$$\prod_{n\in\mathbb{N}}^{\infty} \int_{\mathbb{R}} \sqrt{\frac{d\mu_n}{d\nu_n}} d\nu_n$$

converges, or the infinite sum series

$$\sum_{n\in\mathbb{N}} \sqrt{\frac{d\mu_n}{d\nu_n}} d\nu_n$$

converges

We use a finite product probability measure to measure of finite paths on the Bratteli Diagram. The evolving nature of the $(B, \mu^{(t)})$ means the time dependent measure $\mu^{(t)}$ can be analyzed with Kakutani's theorem to determine equivalence if the Radon-Nikodym derivative exists. In this context, for each t, the measure would be defined as $\mu^{(t)} = \bigotimes_{n\in\mathbb{N}} \mu_n$. This can give insights to some interesting questions in the context of neural activity as the brain changes overtime.

- With the same stimulus, are the same areas (measure of subsets) of the brain activated as a persons ages?
- With the same stimulus, is the amount (measure) of neural activity in the brain the same as a person ages?
- What behavioural change, action, or stimulus transforms the amount or areas of neural activity in the brain?

Discussion and future work

Random Walks on Bratteli Diagrams

A natural extension of modeling with Bratteli Diagrams is simulation. By simulating paths of a Bratteli Diagram given some stimulus, we can get a realistic idea of how neural networks fire in real-time. We can start with some stimulus ζ, which we assign a probability $p \in [0, 1]$. We use probabilities on the edges as paths from which an electrical signal travels. Since the probabilities leaving each vertex do not have to sum to 1, potentially thousand of paths can be "lit up". Also by defining the transformation matrices T_k we can how the same stimulus can "light up" different paths. With more research, the goal would be to simulate a 3-D model of neural circuitry in the brain using paths Bratteli Diagrams.

fMRI

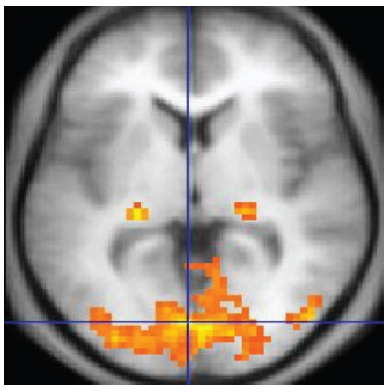

Figure 6: fMRI of the human brain [26]

Functional magnetic resonance imaging (fMRI) is the process which measures changes of brain activity by detecting blood flow. This relies on the fact that neural activation and blood flow are

coupled. The activation of an area of the brain means increased blood flow to that region [27]. Through hemodynamics, the dynamics of blood flow in the body, blood oxygenation and blood flow in the brain are synonymous. Hemoglobin in red blood cells carry oxygen O_2 molecules. Deoxygenated hemoglobin cells (oxygen poor cells) have a different magnetic resonance than oxygen rich blood cells, therefore fMRI can detect areas of the brain that exhibit increased blood flow. fMRI uses the Blood-oxygen-level-dependent (BOLD) signal to measure blood flow, blood volume and oxygen in the brain [28] [29].

This is related to fMRI's which map neural activity in the brain. The measure of paths on the Bratteli Diagram can correspond with the amount of brain activity exhibited in the fMRI. More research would have to be conducted by mapping specific areas of the Brain to subsets of the Bratteli diagram. Then defining the topology of the measure space X_B would give an idea how certain paths could be mapped to certain regions of the brain.

How do I know if I've been in this room before?

One big question of Neuroscience bought to our attention by Prof. Bryce Vissel is *How accurately do I know if I've been in this room before?* What areas of the brain activation, and what neural activity is present when assessing whether I've been someplace before? This question not only has significance in terms of neuroscience, but also forensics, criminology and psychology. Clearly, areas of the brain which are associated with memory, reasoning, and motor patterns are likely candidates for activation when determining if you have been somewhere before. Applying a maths prospective to this question could potentially yield some powerful insights. By thinking of neural circuits as probabilities of paths on a Bratteli Diagram, we see that perhaps the stimulus of that room activates an initial low probability path "dormant circuits". Analogous to repressed memories, perhaps that initial infrequently taken path leads to other paths of higher measure and the higher the measure, the more certain you are that you've been in this room before.

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