

Neurostatistical Mapping: A Review and Unique Application Proposal, Exploration

By  
Michael Lawrence Haffner

A THESIS

submitted to  
Oregon State University  
Honors College

in partial fulfillment of  
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degree of

Honors Baccalaureate of Science in Industrial Engineering  
(Honors Scholar)

Presented May, 26, 2021  
Commencement June 2021



## AN ABSTRACT OF THE THESIS OF

Michael Lawrence Haffner for the degree of Honors Baccalaureate of Science in Industrial Engineering presented on May, 26, 2021. Title: Neurostatistical Mapping: A Literature Review and Unique Application Proposal, Exploration

Abstract approved:

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James Molyneux

Advancements in neurological data capturing and mapping have provided scientists with exponentially more information, allowing for more detailed analysis of neurological behaviors and their translations to cognitive, emotional, and physical function. Inspired by the difficulty in diagnosing brain damage, this project seeks to leverage statistical modeling with EEG data to explore a unique approach to modeling and representing neurological activity. The application of the Cox Point Process, a variation of the more commonly know Poisson Point Process, yielded simulation results that allow us to generate heat maps representing neural activity - suggesting that this application of statistical point processes potentially have promise in analyzing neurological data.

Key Words: neuroscience, point processes, Cox process, Poisson process, applied statistics

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Honors Baccalaureate of Science in Industrial Engineering project of Michael Lawrence Haffner presented on May, 26, 2021.

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Michael Lawrence Haffner, Author

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# 1 Introduction

## 1.1 A Personal Connection

At 17 years old, while playing football, I sustained a severe concussion. The injury caused a considerable amount of disruption to my daily schedule, necessitating stints of physical, speech, and occupational therapy, as well as prolonged exposure to vision therapy. Initially being removed from school for roughly two months, the catching-up process brought with it a number of curious root causes that result in otherwise generic symptoms - deficiencies in reading speed and comprehension, difficulty retaining information and concentration, frequent headaches and dissipated mental stamina, as well as sensitivity to light and noise.

The initial severity of my concussion ultimately proved beneficial. Milder injuries are often diagnosed and addressed by a general practitioner or an athletic trainer; bedrest and a brief respite from play are usually the prescribed treatments. While this does help, and in many cases the patient recovers fully, others can experience prolonged suffering and even permanent impairment, as the specific damage to the brain is never specifically addressed. In this regard, I am lucky, as the severity of my concussion necessitated a sports injury specialist, resulting in in-depth diagnoses, and referrals to specialists for treatments of my injury.

The largest culprit of my cognitive impairments were ultimately based out of the visual system. My eyes themselves are completely fine - the way my brain processes the information and stimulus from them, however, is not. Visits with an ophthalmologist eventually identified saccadic eye movement, exophoria, and convergence insufficiency as three of the main atypical ocular behaviors identified as causing my difficulty in reading and retaining information.

Discovering this root cause of my concussive symptoms was surprising, and fascinating, as neither myself, nor close friends or family, were aware that concussions could cause such side effects. It inspired a casual yet ever-present interest in neuro-



ogy.

## 1.2 Project Aims and Goals

Narrowing down and identifying the root cause of concussive symptoms is challenging. The ability to statistically model neural activity using available, accessible technologies could improve the diagnosing capabilities of physicians and sports medicine specialists. This project aims to explore this problem, by investigating whether or not neurological activity data can be successfully modeled through existing point process software analysis.

As previously mentioned, the commonality of concussions, combined with the breadth of possible side effects, makes narrowing down and identifying the root cause of symptoms challenging. *What if, we suggest, you could improve the diagnoses procedure using neural activity data and the Cox process?* If a concussive patient undergoes a scan while performing a series of routine tasks, could we use this statistical analysis to determine significantly different levels of neural activity throughout certain regions, compared to some baseline? If, hypothetically, we could demonstrate a statistically significant difference in neural activity in the visual region of the brain, compared to some baseline, could that suggest the patient is having to expound more mental effort to perform a relatively rudimentary task, and therefore could have difficulty concentrating, remembering, or are plagued with fatigue? The patient therefore might benefit from targeted therapies to address the underlying deficiency. These *what ifs* are far from simple - would a massive repository of 'healthy' brain data be adequate to provide a generalized baseline? Or would each patient have to have their own individual "before" scan in order to accurately measure some deviation? While intuition allows us to formulate some guesses about these next progressive steps, we first need to demonstrate an ability to statistically model this neural activity.

Herein lies the goal of this exploratory analysis - to demonstrate an observable, significant difference in neural activity under different cognitive loads, or, difficulty

of task, using a spatial point process model - the Cox model. Success in this first step justifies further exploration to more advanced applications in visualization and analysis.

To explore this problem, we will briefly introduce the topic of concussions (Section 2), and the physiological damage they cause to the brain and the impacts they have on patients. We will also discuss the statistical concepts (Section 3) applied in our analysis, work through the processing of the data (Section 3.3.2, and demonstrate a simulation (Section 4.3) of the statistical models. We conclude with results and the practical ramifications of these results (Section 5.)

## 2 Concussions

The commonality of concussions betray the gravity and seriousness of the injury. Cells called *neurons* transmit electrical signals between one another, regulating sensory input and dictating response - such as motor commands to muscles or recollection of some piece of information or memory[1]. The electrical communication between these cells are referred to as neurons ‘firing’[23].

Over time, the order in which these neurons fire - the ‘path’ these electrical signals take - become familiar patterns. The more familiar the pattern, the easier the brain can produce the subsequent response, which we realize as learning[17, 23]. Familiar, everyday actions such as walking have deeply ingrained neural patterns. The strength of each neural connection on that path is much stronger than, say, the neural patterns that dictate information retrieval on a geography test. That being said, information retrieval can also have an incredibly strong neural pathways. For example, the name of your parents, or your home’s address are usually strong patterns. When the brain is damaged, the firing patterns - the neural pathways that ‘light up’ to produce some response, can be damaged. When neural cells (neurons) are damaged, they do not transmit electrical signals as effectively, weakening the neural pathway.

Every brain is different, with different firing patterns that elicit different responses, bits of information, emotions, etc. However, we know that certain areas of the brain control different responses[7]. Due to this, we generalize the structure of the brain - areas that light up in response to visual and auditory stimuli are consistent, and likewise consistently different from areas of the brain that process emotion, memory, or pain[18]. Therefore, despite the individuality of each brain, they all adhere to the same approximate structure.

## 2.1 What is a Concussion?

Following from the Queensland Brain Institute[28], your brain floats in your skull. Cerebrospinal fluid (CSF) suspends your brain within the cranial cavity protecting it from damage caused by everyday swelling due to blood pressure fluctuations[8]. CSF also protects it from small jostles and everyday movements that would otherwise stress a rigid spinal cord. However, violent acceleration of the skull - either cause by a direct impact to ones head, or a whiplash effect from sudden bodily motion, can cause the freely-suspended brain to collide with the inner wall of the skull, damaging the tissue of the brain - commonly described as a ‘bruising’ of the brain, although this gentle term can betray the possible severity of such an injury. When this impact occurs, the brain always sustains some damage, even if minutely. This event is called a *concussion*[28], and are commonplace - especially among athletes. Medically speaking, some evidence suggests every time a person ‘heads’ a soccer ball, they sustain an (albeit) small concussion[27]. For a visual depiction of how a concussion occurs, see Figure 1.

In extreme cases where damage to the brain is extensive, concussions are referred to as a traumatic brain injury. (TBI)

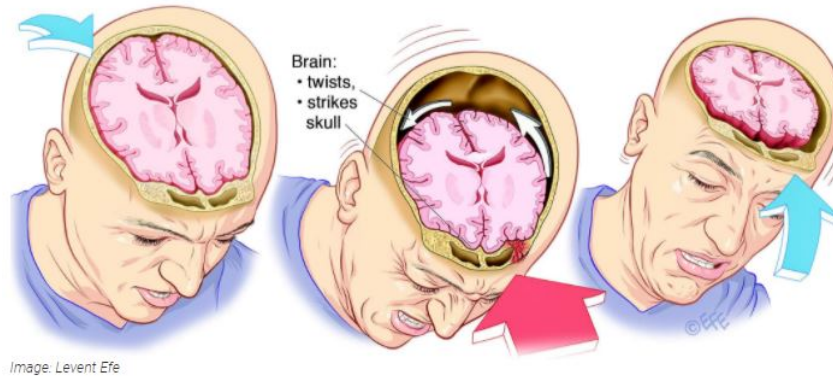


Figure 1: *Figure of a Concussion. Image courtesy of the Queensland Brain Institute, Australia[28].*

### 2.1.1 Symptoms, Damage and Diagnoses

The pathway that is damaged determines the type of symptom experienced by the patient. The list of possible symptoms from concussions is broad, and generalized. These symptoms include: fatigue (mental or physical), depression, anxiety, difficulty concentrating, memory problems, menstrual changes, poor motivation, impulsiveness, migraines, dizziness, sleep disturbances, mood swings, and, anger or irritability[13].

*There is no universally accepted definition of a concussion[28].* This is a large part of what makes them difficult to officially identify and diagnose. The breadth and commonality of these symptoms means they often go overlooked, and the concussions go undiagnosed and unassessed - 89 percent of concussions are undiagnosed[28]. These symptoms can be the manifestation of complex problems within the neural pathways that are damaged.

For example, difficulty concentrating as a result of a concussion might be a result of increased fatigue, difficulty with the neural pathway that commits to memory, or, as a result of damage to neural pathways within the visual system. In expansion of this last explanation - when concussions occur, they can impair the function of the eyes, making it difficult for fine motor control to direct the eyes to converge and therefore focus[11]. With the damaged neural pathway, the concussed individual unknowingly

expounds increased mental effort to successfully focus their eyes and read, sapping their mental stamina and ultimately making it more difficult to concentrate. In instances such as these, therapeutic efforts that target the visual system, such as vision therapy and exercises that practice fine eye movements and motor control, reinforce the previously damaged neural pathways involved, eventually reducing the effort required to read, and thus increasing concentration.

If, however, damage to the visual neural pathways was not recognized, the root cause of concentration problems might go undiagnosed, or mis-diagnosed. The patient continues to suffer from symptoms, greatly impairing quality of life.

The countless possible explanations for concussive symptoms represent a significant problem. The boilerplate treatment for concussions, especially mild ones, is bed rest - allowing the brain to heal itself, reduce swelling, and repair the damaged cells and the neural pathways. In instances of mild concussions, when pathways are not damaged significantly, recovery time is limited, and targeted treatment and therapy is not needed. However, more serious concussions can yield lingering symptoms, the causes of which are difficult to determine using traditional diagnoses methodologies. Failure to address the specific structural damage to the brain perpetuates symptoms.

Qualitative metrics, such as the Glasgow Coma Scale (GCS) are frequently used as an initial diagnosis metric due to the low complexity involved, making them perfect first passes for athletic trainers. The GCS measures a person's function in three basic areas: ability to speak, open eyes, and move[13]. Other questions can be presented to the patient - such as basic cognitive tasks or ability to read and write. These qualitative metrics are proficient in determining what symptoms a patient will experience, but do not actually determine the root cause for the patient[13].

Quantitative measurements can be made using functional Magnetic Resonance Imaging devices - fMRI, and Electroencephalogram (EEG)<sup>1</sup>. While both of these

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<sup>1</sup>How these technologies work and collect data is not within the scope of this project, but, more information can be found here: [https://books.google.com/books?id=HtkAfChTLToC&printsec=frontcover&source=gbg\\_ge\\_summary\\_r&cad=0#v=onepage&q&f=false](https://books.google.com/books?id=HtkAfChTLToC&printsec=frontcover&source=gbg_ge_summary_r&cad=0#v=onepage&q&f=false)

technologies provide useful information, they each have trade offs. fMRI provides superior spatial resolution, capturing more detailed information about neural activity within the brain, at the sacrifice of temporal resolution - less detailed information about neural activity over time. EEG, on the other hand, is the opposite. It provides superior temporal resolution at the expense of spatial resolution[24, 20, 15, 10].

As we will discuss later, we will be using EEG data in this project.

## 3 Applied Statistical Concepts

### 3.1 Point Processes

In considering how to represent neural activity in the brain, it is first important to touch on the concept of a point process. As the name suggests, a point process, in statistical terms, is merely a dispersion of singular data points across some sample subspace, usually, space and/or time[4, 16]. Consider how neural data is recorded - EEG readings, for example, are achieved by the placement of ‘nodes’ around a person’s head, with each node reading in neural activity at that specific point on the brain. We can see, then, how the data presents itself as a series of points over space and time, lending itself well to analysis via point patterns.

### 3.2 Stochastic Processes

A stochastic process is one which incorporates some element of randomness to the outcome. It is easier to think about this as the opposite of a deterministic process. In a deterministic process, a particular input (or inputs) will always result in the exact same output, because all parameters of the process are known, and consistent. We can *determine* exactly what the result will be, every time, without variation. An easy example of this is the simple equation:

$$y = mx + b.$$

The known parameters,  $m$  and  $b$ , mean that for each input value of  $x$ , we can calculate the exact value of  $y$ , every time. When dealing with stochastic processes, however, this is not true. Identical inputs, due to randomness, will likely yield slightly different results, but could yield vastly different results. Stochastic processes evolve randomly throughout time. This evolution, in turn, can be observed and recreated through probabilistic models, either through discrete time events or over continuous time intervals[14]. Statistical models are used to describe the randomness of these processes. For the sake of this project, we will be focusing on Poisson statistical models.

### 3.3 Poisson Process

The Poisson process is a statistical model used to represent the occurrence of points over some dimension or dimensions. Fundamentally, it is a *counting process*, as it returns the number (count) of events that occur over an interval of some dimension, often time or space. The process is easily thought of as rates of occurrence over time - such as how frequently car accidents happen on a stretch of freeway throughout the day. At different time intervals, the number of car accidents would be different. Poisson models can also count rates of change over space, such as the frequency of earthquakes in relation to some distance away from a fault line.

Specifically, the Poisson process describes the distribution of the number of events,  $Y$ , that occur over some sample space, be it time, space, distance, area, or volume[26]. Conceptually, the Poisson process is a *limiting* case of the binomial distribution. Similarly to how the binomial distribution measures the number of successes and failures for some number of finite trials, the Poisson process measures the existence of an event or not for an infinite number of trials. To stick with our freeway analogy example, a car crash either happened in such moment in time, or it did not. If then, for over some period of time, we use the binomial distribution to represent the number of events (successes) or not (failures), we can consider the number of repetitions to repeat this sampling  $n$  to be a number of sub-intervals within some given time period.

If we then take the time intervals to be infinitesimally small, such that only one event could possibly take place[26] within each subinterval, then either an event happens, or does not happen, but never do two events happen over the same sub-interval. In rationalizing this, one must realize that the requisite size of each sub-interval is so unbelievably small, that a functionally infinite amount of them occur over any given sub-interval of time. Therefore, to calculate a probability distribution for this event over the entire time period, we can take the limit of the binomial distribution as  $n$  approaches infinity. The resultant calculation of this limit is what we refer to as the Poisson distribution, modeling the rate of some event, over some dimension[26, 14].

This is applicable to the research question, because the event of a neuron firing is likewise binary - a neuron either fires or it does not. Herein lies justification for modeling neural activity with a Poisson process.

### 3.3.1 Intensity Function for Poisson Processes

An inhomogeneous Poisson point process has intensity function  $\lambda(u)$  in which the expected number of points  $\mu$  falling within a region B is the integral:

$$\mu = \int_B \lambda(u) du$$

where  $\lambda$  is the *intensity* of the process - the average number of points per unit of area[4]. The intensity function  $\lambda(u)$  is important - it drives the behavior of the point distribution over a certain area, and this characteristic will be used later on in the simulation.

### 3.3.2 A Brief Note about the Exponential Distribution

The exponential distribution is closely tied to Poisson point processes. It is the probability distribution which describes the length of time in the intervals between successive Poisson event[21]. Its parameter,  $\lambda$ , is considered the *rate* of the distribu-



tion. Given as the expected, or, average value of the distribution  $\mu$  can be expressed as the inverse of the rate[21], we can derive the exponential rate function by inverting the mean:

$$\lambda = 1/\mu$$

This relationship between the exponential distribution and Poisson process will come into play during the simulation.

### 3.3.3 Homogeneous versus Inhomogeneous Poisson Processes

Homogeneous Poisson point processes are both stationary, and isotropic, unlike inhomogeneous Poisson point processes[16]. In this regard, stationary point processes have distributions that remain consistent under translation of a point throughout the sample space. Ergo, the distribution of points is consistent regardless of where they are observed within the area in question[16]. Similarly, isotropicity exists if the point process' distribution is consistent regardless of rotations about the origin of the area in question[16]. In considering neural behavior, we can intuitively recognize that the relative frequency of neural activity is dependent upon the neuron's location within the brain, as well as the task being performed. Therefore, its behavior cannot be statistically described as stationary *nor* isotropic, meaning the describing point process must be inhomogeneous. The difference between these two processes can be seen in Figure 2, which a realization of both a homogeneous and inhomogeneous point process, in space, graphically. The homogeneous point process in the left pane exhibits a completely random pattern while the inhomogeneous point process in the right pane exhibits a preference for points to occur in the upper-right hand corner of the plot.

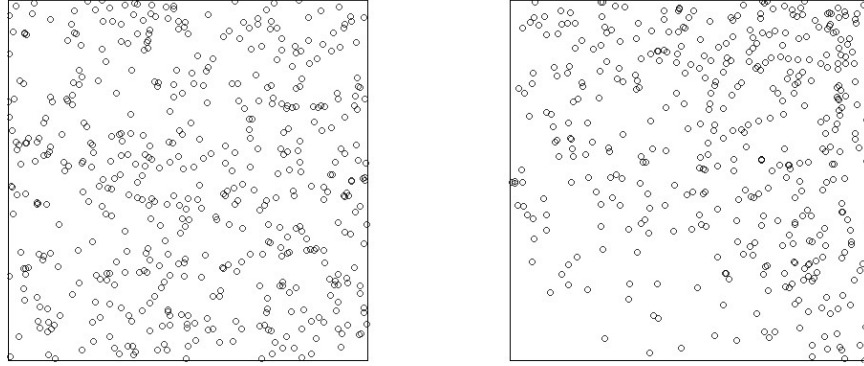


Figure 2: *An example of a homogeneously distributed Poisson point process (left) and an inhomogeneously distributed point process(right). Notice the bias of the points in the inhomogeneous graph - clustering near the upper-righthand corner of the plot.*

### 3.4 Cox Processes

Furthermore, it is not appropriate to simply use the Poisson process to model neural activity. A key assumption of the Poisson process is that each point within the process is independent of one another. That is to say, the existence of one event bears no influence on the existence of another event[4]. From what we understand about the structure of the brain, with certain regions controlling certain functions, and neural pathways existing as a linked chain of neurons, this assumption could not possibly be met. (Positive) dependency between points/events is referred to as *clustering*, and clustered point processes are so prevalent in the real world that models have been developed to represent them. For the purposes of analyzing neural data, we will consider the Cox process - a modification of the Poisson process.

The modification present in the Cox process is the randomization of the intensity function, which, for spatial point processes, is a randomness attributed to spatial covariance[4]. This covariance, this variation and randomness in the Poisson intensity function, causes points to be spread inhomogeneously across space. The practical consequence of this is that points could be more abundant in some areas of the relevant region than in others - a ‘clustering’ of the points, again, due to some spatial covariance[4].

When we consider the structure and function of the brain, we can intuitively draw parallels between neural activity and the behavior of point processes. As we discussed above, different regions of the brain are responsible for different functions. Therefore, we suggest that this innate structural attribute can be comparatively used as the spatial covariance that drives the randomization of the Poisson intensity function - the Cox process. Thinking practically, this makes sense - when performing tasks focused on fine motor control, such as typing an Honors College thesis, one would not expect the region of the brain that is connected to taste buds to be particularly active. Rather, one would (reasonably) expect to see clustering of neural activity present in spatial regions associated fingers and hands, as well as critical thinking and memory, to name a few.

Of course, the introduction of some appropriate stimulus (say, for example, drinking black coffee - a necessary companion to the often late-night activity of Honors thesis writing) should, in turn, be observed with increases in neural activity in the appropriate region of the brain. It is with this that we adopt a somewhat banal and axiomatic hypothesis about neural activity:

*If the consumption of some stimulus or performance of some action consistently results neural activity in a particular brain region, then absence of neural activity in said region likewise results in the absence of the stimulus/action.*<sup>2</sup>

## 4 Methodology

### 4.1 Data

The EEG data used in our analysis was found on a public GitHub repository<sup>3</sup> of various types of EEG data. Aside from being publicly-accessible, this data set was

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<sup>2</sup>It is important here to take pause and note the limitations of the scope of this thesis - the focus of which is purely exploratory statistical analysis, and not functional neurology. However, the above self-evident statement serves as the kernel for the (hopeful) statistical application.

<sup>3</sup>The repository[5] can be found here: <https://github.com/pbashivan/EEGLearn>

chosen because it is feature-rich. The data set contains both spatial information, as well as temporal information, providing 3-dimensional Cartesian coordinates for each EEG sensor node, as well as recording activity for a variety of time windows. Furthermore, the data set provides the activity readings of each sensor node over a four different brain loads, as well as recording three different wave forms: alpha, beta, and theta. These wave forms are dependent on the *frequency* of the readings, and reflect different types of neural activity: The wave form alpha has oscillations between 8 and 12Hz, and is generated when a person’s mind is at rest - such as in a meditative state. Conversely to beta, alpha wave forms represent the absence of mental activity, and has been described as an ‘idling rhythm’[12, 24]. The wave form beta has oscillations between 15 and 30Hz, and is generated with the brain is actively engaged in mental activities, such as giving a speech or presentation. This arousal response of the brain suppresses slower EEG rhythms, and, in simultaneous EEG-fMRI studies, a correlation was noted between beta rhythms and strong fMRI readings in areas of the brain associated with high levels of introspection<sup>4</sup> and mental exercise[12, 24]. The wave form theta has oscillations between 4 and 7Hz, and is generated when the brain is daydreaming, or, in a ”zoned out” state, such as the ‘autopilot’ that sometimes happens when people are driving on the freeway[12, 24].

In considering the goal of our analysis - demonstrating the applicability of Cox models to measure and compare neural activities under various conditions - this data set is uniquely positioned to allow for analysis across various states of mental engagement. However, for preliminary investigation, we will focus on the beta wavelength as it best demonstrates engaged mental activity.

Under ideal circumstance, two sets of data would be used for analysis - each with the same participants, collecting the same information, while performing the same tasks, with the only difference between the two is that one set of data be collected before a concussion, and the second being collected after. Understandably,

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<sup>4</sup>The emphasis here is inward thinking, not a response to external factors.

the difficulty of obtaining such a set of data organically precludes us from being able to conduct such an analysis, these are not ideal circumstances. Brain scans of the same subject both before and after a concussion are rare, and even if enough could be uncovered to create a data set, the lack of standardization between them would invalidate most results. Ethical considerations likewise prohibit such a data set from being deliberately constructed. However, we believe that if we can demonstrate Cox models depicting different levels of brain activity, this justifies further exploration in using Cox models to describe and analyze neural activity.

## 4.2 Data Exploration

Looking at the EEG data in its raw form, it is not immediately clear how to report the data spatially, or as a point process. The de-facto output for EEGs is shown in Figure 3.

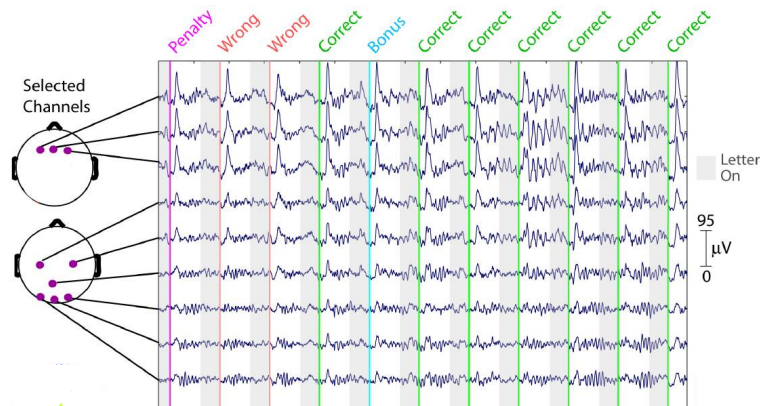


Figure 3: *Standard output of an EEG. Image courtesy of the University of California, San Diego[9].*

In order to approach this problem using a Cox process, some processing needs to be done. To aid in the visual analysis as part of our data exploration process, an interactive 3-dimensional plot of the data across various wave forms, brain loadings, and time windows was developed to serve as an analytical tool.<sup>5</sup> This application was

<sup>5</sup>The application can be access here: [https://haffnerm.shinyapps.io/EEG\\_3D\\_Plot/](https://haffnerm.shinyapps.io/EEG_3D_Plot/)

used throughout the analysis and simulation process as it helps to visualize the neural activity. It was used to spot-check our simulation to see if the generated offspring points accurately represent the associated level of brain activity. Figure 4 shows an image of the app. Each time window has multiple readings within said time window - they were averaged to achieve a mean reading for said time window. Exact continuous temporal data was not available, as each reading was grouped into a time window.



Figure 4: *The app used for exploratory analysis. The slider bars on the left allow the user to filter through the different subjects, time windows, and brain loadings. The top graph shows mean readings for each sensor node under the appropriate filters, whereas the bottom graph shows the standard deviation of each node reading under the appropriate filters.*

As the Cox process is a point process, some processing needed to be conducted to translate singular points to represent spatial regions of the brain. To address this issue, the Voronoi<sup>6</sup> method was used to mathematically generate cells around each EEG node location. We make the assumption that Voronoi cells are an adequate way

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<sup>6</sup>Voronoi Diagrams, also called Dirichlet tessellations[25], are a way to generate regions, or *cells*, around central points such that from any location within the cell, the closest point to that location is the one around which the cell was formed[3]. It is a way to define borders around which point is the closest to any region of space.

to represent spatial regions of the brain, and, that activity in that general region is picked up by the closest EEG node.

Most software packages generate Voronoi cells in 2D space, including the one we used in R. While there are some resources available online, there does not exist widely-accessible standard software to calculate Voronoi cells in 3D space. While a resource was found[22], its exclusive compatibility with C++ prevented it from being a viable choice for the purposes of this project, as R is the lingua franca of the parties involved. As most of the R packages and tools used to simulate and analyze point processes are optimized for use in 2D, the most reasonable course of action was to project the data into 2D.

### 4.3 Simulation

To consider the data set in 2D, it is not sufficient to simply use the Cartesian  $(x, y)$  coordinates of each EEG node. Ignoring the  $z$ -coordinate eliminates a dimension of the spatial relationship between points, which is a critical aspect of the research question. This can be effectively demonstrated by considering a graph of the just the  $(x, y)$  coordinates as shown in Figure 5. In ignoring the third dimension, compression of the points occurs around the perimeter of the brain. This loss in spatial resolution is unacceptable, and which is why we introduce projections to address this problem.

We use a stereographic projection to project our data from 3D to 2D. It is important to note that stereographic projections are not isometric, and do not preserve area[6]. However, it does preserve the angular relationship between points, creating a more faithful representation of the EEG nodes than just the  $x, y$  coordinates. Using this projection allows us to better preserve the spatial relationships between each EEG node. The formula used for the projection is as following[6]:

$$X_{stereo} = x_{cart}/(1 - z_{cart})$$

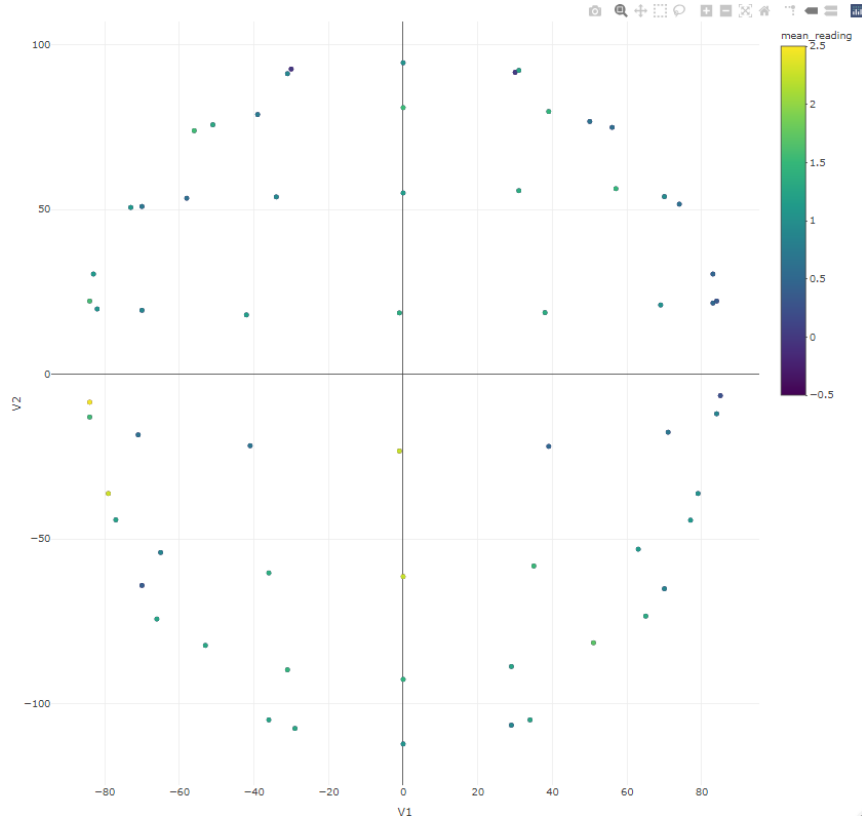


Figure 5: A top-down view of the EEG locations around the brain. Notice the compression around the edge, making the points appear to be much closer than they actually are. A comparison to Figure 4 demonstrates how the third dimension plays a crucial role in the spatial relationships between the points.

$$Y_{stereo} = y_{cart}/(1 - z_{cart})$$

The projected points now create a data frame we can use to effectively generate Voronoi cells, and simulate point processes.

The projection can be visualized in the Figure 6. The projected EEG nodes with their respective Voronoi cells can be seen in Figure 7.

This projection allows us to simulate and analyze the readings of the EEG data as a Cox point process. To do so, we will be discussing three categories of points, so it is important to clearly delineate how we will refer to each group. Each of the EEG nodes are called the ‘parent points.’ From these parent points, we will randomly generate ‘background points.’ Finally, from these background points, we will generate



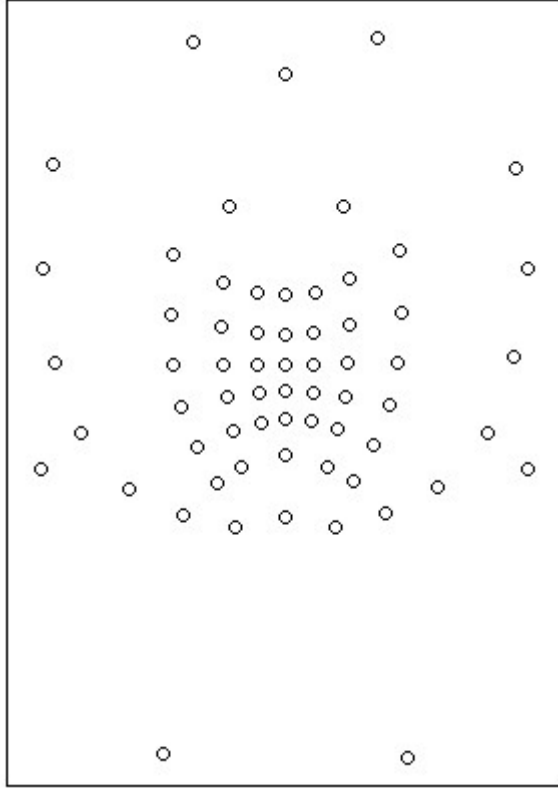


Figure 6: *The stereographic projection of the EEG nodes onto 2D. Compare against Figure 5 to see how spatial relationships are better preserved. These points are considered the ‘parent’ points.*

‘offspring points.’ The distinction between each of these three categories is important.

After projecting the nodes onto a 2D plane, each of the node’s stereographic  $(x, y)$  coordinates were read in as an existing Poisson point process, using the `ppp()` function in R’s `spatstat` package. Voronoi cells were generated for each point. Then, each cell was cycled through and an arbitrary number of background points were generated with an intensity function proportionally consistent with the size (area) of each cell<sup>7</sup>.

For each of the background points, the process is repeated. For each background point, offspring points are generated as a Poisson point process. However, this time, the intensity function driving the generation of offspring points within a cell is not

---

<sup>7</sup>As an aside, a function was written which returns the cell in which a point - the input parameter - is located. This function was used repeatedly throughout the simulation process, and further references linking points to a cell implicitly used this function in some capacity

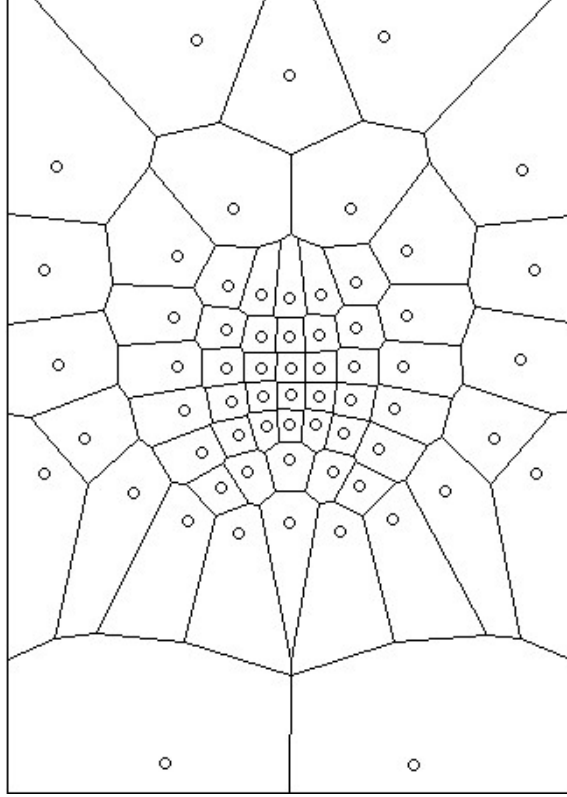


Figure 7: *The Voronoi cells calculated for the projected EEG locations.*

merely proportionally consistent, as it will with the background points.

To generate our offspring points, we use an intensity that takes a random value from an exponential distribution, due to the relationship discussed in Section 3.3.2.

$$\lambda_{Offspring,i} \sim \text{Exp} \left( (\text{EEG Reading}_i - \text{Shifting Value})^{-1} \right) \text{Area}_i^{-1}$$

As we know that the parameter for the exponential function, the rate, is merely the inverse of the average, or expected value, we calculated the exponential rate based on the initial EEG reading from the parent point of the cell. As you may recall, this EEG reading is the average value of the readings at this point over a given time window, and, as the dispersion of these offspring points are intended to represent the neural activity at that location over a certain window of time, this use of the

EEG reading is justified. The EEG reading is corrected for a shifting value<sup>8</sup>, and the result from the exponential distribution is generated. Before being passed to the intensity function parameter for the Poisson distribution, however, the value is lastly scaled proportionately to the size (area) of the Voronoi Cell  $i$  that the points are being generated within.

To pause and reaffirm: the background points are generated arbitrarily, and consistently - they merely represent part of the stochastic randomness that is crucial to the Cox process, and act as a ‘placeholder’ for the EEG nodes. As the placement of the EEG nodes is predetermined, and the number constrained to one per cell, the parent points are a decidedly *not* random point pattern. Using these parent points to spawn our offspring points would not adequately reflect the behavior of Cox point processes, nor the brain. Use of background cells provide a means to represent neural activity throughout an area. Furthermore, the background cells are generated consistently yet proportionately to the area of the Voronoi cell they are in, to avoid bias. The intensity with which to generate these background points was chosen arbitrarily, necessitating only act as a “happy medium” - we did not want too many background points generated so as to crowd, and therefore obscure, data, nor did we want there to be to be a significant risk upon repetition of cells being left empty - that is, no points generated for them by sheer unfortunate chance. The value 6 (divided by area, of course) was found to satisfy these requirements, and was both found and validated by trial-and-error.

$$\lambda_{Background,i} = 6/Area_i$$

The ultimate output of this simulation is a collection of offspring points whose

---

<sup>8</sup>Some of the EEG node readings reported negative values. To generate the offspring points in our simulation, the Poisson rate is normalized against the intensity of the relevant EEG sensor reading. However, given as a Poisson rate cannot be negative, we introduce a shifting factor to bump the rate to be just north of positive. This is based on another key assumption - we are assuming that a negative sensor reading reflects a very low level of neural activity. Therefore, we introduces a very small shift factor to ensure the subsequent Poisson rate is small, representing low activity, but still non-negative, to be mathematically possible.

density and dispersion - whose *clustering* - is representative of neural activity in the respective region. By using the R `loccit()` function, we can fit these points to a Cox point process model, and pass this `loccit` object through the `psib()` function to calculate the sibling probability<sup>9</sup> of each point that was fitted to the Cox model. A higher sibling probability means more neural activity, as a larger intensity function would result in more offspring points being generated for each background point, increasing the chances that two points are siblings.

Plotting this sibling probability gives us our most effective visualization tool - a heatmap of sibling probabilities across the different regions of the brain, quantifying neural activity through the use of a Cox point process model.

## 5 Results

The resultant heatmaps of our simulation suggest that the Cox point process model is an adequate way to model and represent neural activity under different loads. Figure 8 depicts each of the 4 different brain loadings for the Beta<sup>10</sup> waveform. The ribbon on the side of each graph maps the colors to the relative probability of sibling points. (They are all the same, standardized scaling.)

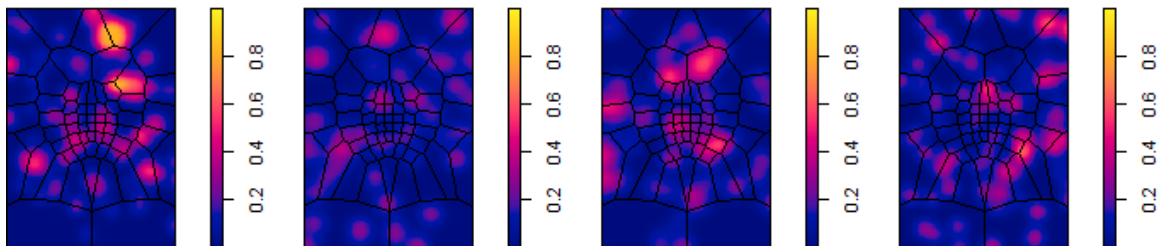


Figure 8: *From Left to Right, regional sibling probabilities for Loadings 1, 2, 3, and 4 of the brain for the Beta waveform.*

For clarity, Figure 9 shows only Loadings 1 and 4, recreated at a larger scale to

<sup>9</sup>In a Poisson point process, two points are called *siblings* if they were generated from the same background point[19].

<sup>10</sup>Again, we chose to focus on the Beta wavelength as it is associated with the highest levels of brain function, on average[24].

more easily draw distinction.

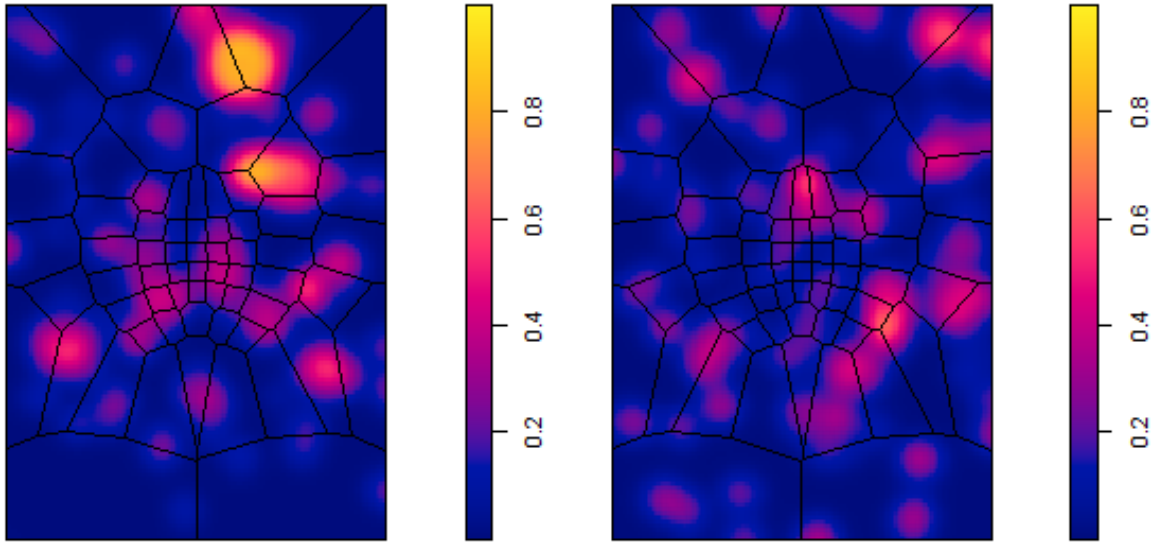


Figure 9: *Left, Loading 1 of the Beta waveform. Right, Loading 4 of the Beta waveform.*

If we compare these heat maps against the levels of activity demonstrated by the 3D EEG app, we can see relatively consistent parallels. This is best seen by comparing Loadings 1 and 2. For Loading 1, in Figure 10, we can clearly see higher levels of neural activity on the top-back portion of the brain, as well as front-right side. Considering the levels of activity represented in the 3D rendering of the EEG data for Loading 1, this pattern more-or-less holds. The same result, albeit opposite magnitude, can be seen for Loading 2, in Figure 11. Brain Loading 2 has muted activity pretty much throughout, a pattern which is corroborated with a comparison to the 3D output.

The parallels are not perfect, nor exact. And, for the other brain loadings, the correlations are not as strong. The comparisons between the 3D app and the heatmaps are not entirely fair - when simulating the data, we averaged over all of the time windows, and subject IDs, unlike in the app, where they are specified individually. These generalizations in the simulation were made for simplicity just as much as they were practicality. The fitting of the Cox model to the simulated points was a

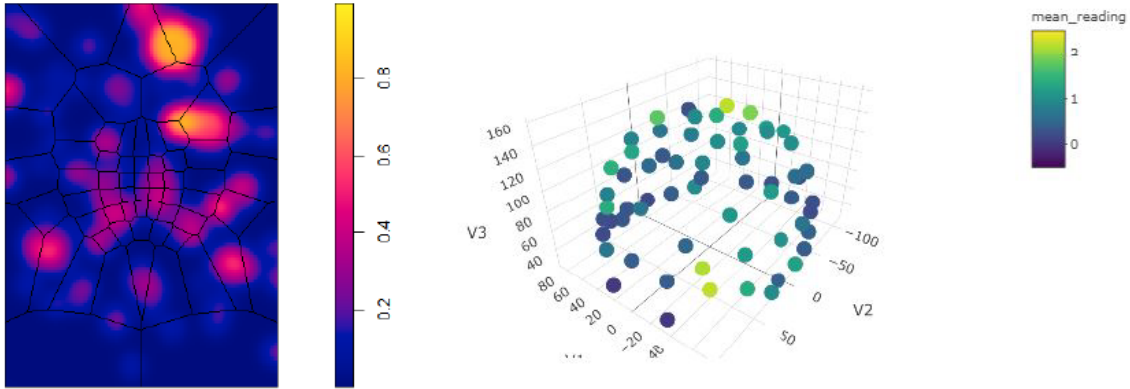


Figure 10: *Comparison of the heatmap (left) against the 3D app (right) for the Beta waveform Loading 1.*

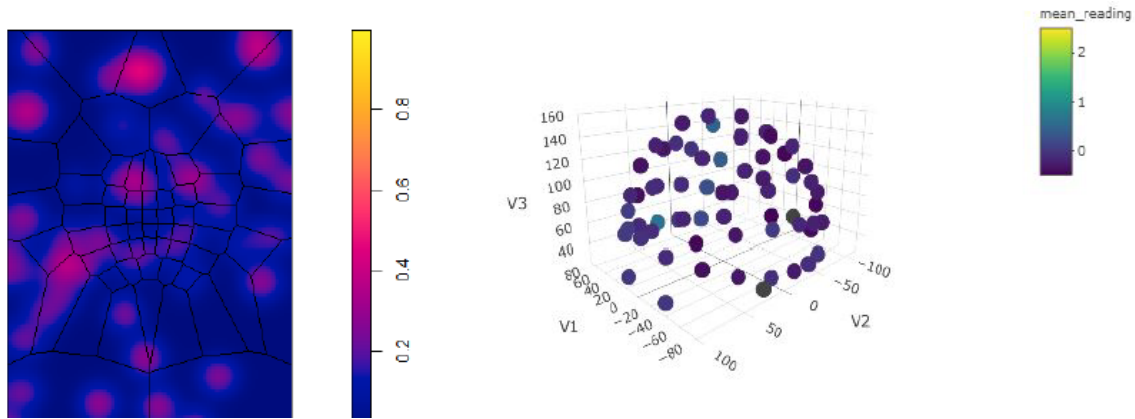


Figure 11: *Comparison of the heatmap (left) against the 3D app (right) for the Beta waveform Loading 2.*

fairly computationally and time intensive process. Individually simulating and fitting models to each subject over each time window was not realistically feasible on personal hardware, nor was reserving HPC<sup>11</sup> time considered to be very responsible until more evidence was provided on the merit of this project.

*The fundamental takeaway is that the modeling and analysis of neural activity through the application of Cox Point Process models is capable of demonstrating significant differences of neural activity over a spatial region.*

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<sup>11</sup>High-Performance Computing - reserving time on Oregon State University's supercomputers.

## 6 Future Work

Throughout this project, some concessions were certainly made for the sake of simplicity and accessibility. However, we believe that the work demonstrated shows promise, and supports further exploration into this application. Throughout the project, we encountered countless rabbit holes or possible next steps to take, some of which are expanded upon below:

- We would like to explore the incorporation of fMRI data into our analysis. fMRI has superior spatial resolution, so the combination/contrast between the two would provide more information and insight into the levels of activity.
- The use of machine learning, classification, and neural networks to classify neural activity based on certain brain loadings is a logical next step. In addition to these classification methodologies, recent exposure to hierarchical clustering algorithms likewise inspires future applications.
- The current method of analysis realistically only incorporates the spatial dimension as a covariance factor. This was out of the sake of simplicity. However, a more complex, next iterative step would be to create a generalized additive model, with which to include the temporal dimension as well as spatial.
- One critique with the methodology used reflects the limitations of EEG as measurement medium. EEGs measure on the surface of the head, and as such, lack the cross sectional capabilities of MRI or fMRI to see ‘inside’ the brain instead of just the surface. While our method of projecting the EEG points into 2D and using Voronoi cells after is acceptable, we think the idea of using surface-area Voronoi cells could provide an added benefit to analysis. Surface-area Voronoi software, like 3D Voronoi cells[22], is not widely available in statistical packages such as `spatstat`. However, prior research on the use of Voronoi cells

for surface area[2] certainly exists, so it would be a fascinating avenue to explore further.

## 7 Conclusion

In considering the goals of the project, to demonstrate the applicability of the Cox point process in the modeling of neural activity, we feel as though we have successfully met this end goal. Excitingly, there seems to be multiple direct follow-up projects with which we could build upon the work demonstrated here. Concussions are debilitating, common injuries that can impair anyone unlucky enough to sustain one. We hope that the realized results - the unique visualization methodologies and analysis, can go on to improve the accuracy and frequency of concussion diagnosis, and aid in the treatment of brain injuries of all kinds.



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## 10 Appendix

### 10.1 Code

#### 10.1.1 3D App Code

```
library(tidyverse)
library(plotly)
library(readr)

#load("~/C:/Users/haffn/Documents/College/Thesis/Nuerostatistical Mapping/
Coding/Report Files/eeg.Rda")

load("eeg.Rda")

# Define UI for application that draws a histogram
ui <- fluidPage(

  # Application title
  titlePanel("EEG Data"),

  # Sidebar with a slider input for number of bins
  sidebarLayout(
    sidebarPanel(

      selectInput("wave_form",
                  label = "Choose a wave form to display",
                  choices = c("alpha",
                              "beta",
                              "theta"),
                  selected = "alpha"),

      sliderInput("subject_id",
                  "Select a Subject to View:",
                  min = 1,
                  max = 15,
                  value = 1),

      sliderInput("time_window",
                  "Select a Time Window to View:",
                  min = 1,
                  max = 7,
                  value = 1),
```

```

        sliderInput("brain_load",
                    "Select a Brain Load to View:",
                    min = 1,
                    max = 4,
                    value = 1)

    ),

    # Show a plot of the generated distribution
    mainPanel(
      plotlyOutput("mean_plot"),
      plotlyOutput("sd_plot")
    )
  )
)

# Define server logic required to draw a histogram
server <- function(input, output) {

  locations <- eeg %>%
    group_by(sensor_node) %>%
    summarize(V1 = unique(V1),
              V2 = unique(V2),
              V3 = unique(V3))

  #input filtering?
  plot_df <- reactive({eeg %>%
    filter(subject_id == input$subject_id,
           time_window == input$time_window,
           wave_form == input$wave_form,
           brain_load == input$brain_load) %>%
    group_by(sensor_node) %>%
    summarize(mean_reading = mean(reading),
              sd_reading = sd(reading)) %>%
    left_join(locations)
  })

  output$mean_plot <- renderPlotly({

```

```

        plot_ly(plot_df(), x = ~V1, y = ~V2, z = ~V3,
                 color = ~mean_reading) %>%
        add_markers() %>% colorbar(limits = c(-.5,2.5))
    })

    output$sd_plot <- renderPlotly({

        plot_ly(plot_df(), x = ~V1, y = ~V2, z = ~V3,
                 color = ~sd_reading) %>%
        add_markers() %>% colorbar(limits = c(0,4.5))
    })
}

# Run the application
shinyApp(ui = ui, server = server)

```

### 10.1.2 Simulation Code

```
# Load libraries
library(spatstat)
library(spatstat.local)
library(tidyverse)
library(plotly)
library(ggvoronoi)
library(patchwork)
library(ggthemes)

load("eeg.Rda")

locations <- eeg %>%
  select(sensor_node, V1, V2, V3) %>%
  group_by(sensor_node) %>%
  slice(1) %>%
  mutate(Xconv = V1/(1-V3),
         Yconv = V2/(1-V3))

speedy_calc <- function(waveform, brainloading){

  loading1 <- eeg %>%
    filter(wave_form == waveform,
           brain_load == 1) %>%

    group_by(sensor_node) %>%
    summarize(mean_reading = mean(reading)) %>%
    left_join(locations)

  loading2 <- eeg %>%
    filter(wave_form == waveform,
           brain_load == 2) %>%

    group_by(sensor_node) %>%
    summarize(mean_reading = mean(reading)) %>%
    left_join(locations)

  loading3 <- eeg %>%
    filter(wave_form == waveform,
           brain_load == 3) %>%

    group_by(sensor_node) %>%
```

```

    summarize(mean_reading = mean(reading)) %>%
    left_join(locations)

loading4 <- eeg %>%
  filter(wave_form == waveform,
         brain_load == 4) %>%

  group_by(sensor_node) %>%
  summarize(mean_reading = mean(reading)) %>%
  left_join(locations)

shift_value <- round(min(c(loading1$mean_reading, loading2$mean_reading,
loading3$mean_reading, loading4$mean_reading))-0.1, digits = 1)

loading <- eeg %>%
  filter(wave_form == waveform,
         brain_load == brainloading) %>%

  group_by(sensor_node) %>%
  summarize(mean_reading = mean(reading)) %>%
  left_join(locations)

df <- loading%>%
  rename(
    nodes = sensor_node,
    x = Xconv,
    y = Yconv,
    readings = mean_reading
  )

# Based on the nodes in our eeg data, we want to compute (1) the
# Voronoi tessellation for each point and (2) the area/size of each
# Voronoi cell

# Turn our points into a point pattern object
eeg_window <- owin(c(-2.75, 2.75), c(-4, 3.75))

```



```

points <- ppp(df$x, df$y, window = eeg_window) #-> function ppp() creates a
point pattern

# plot(points, main = "EEG Parent Points")

# Compute the Voronoi cells for each point
cells <- dirichlet(points) #-> creates dirichlet (aka voronoi) tessellation
of points

# plot(cells, add = TRUE)

# Compute the area of each cell
areas <- tile.areas(cells)

# To get our simulation to work, what we need is a function which
# takes a set of random points and computes which Voronoi cell each
# point falls into. So to do this, I create the find_cell() function
#
# For this function, you input the x,y coordinate of the points, it
# turns those points into a point pattern object, computes the cell
# the points fall into and outputs the cells. These cells should have
# a 1-1 coorespondance with the sensor nodes. So "cell 5" should line-up
# with sensor node 5 for instance

find_cell <- function(x,y) {
  new_points <- ppp(x, y, window = eeg_window)
  cell <- cut(new_points, cells)$marks
  return(cell)
}

# You can demo the function below
find_cell(x = c(0.2, 0.45, 0.9), y = c(0.2, 0.45, 0.9))

# Now we initiate our simulation. This simulation should generate a
# point pattern which resembles a Cox process.

# First, we generate some "background" points which represent general
# neural activity across the regions of the brain.
#
# Note: When we perform our simulation on the real data, we might need

```

```

# to change lambda = 100 to be some other number. We can chat more about
# this later
# bg_pts <- rpoispp(lambda = 10, win = eeg_window)

store_x_bg <- c()
store_y_bg <- c()

# Iterate through each background point
for (i in 1:64) {
  # Generate some offspring for background event i and place those points
  # in the Voronoi cell which contains that point
  bg_by_cell <- rpoispp(lambda = 6/areas[i],
                        win = cells[i])

  # If no offspring are created, move on. Otherwise, put them into the
  # storage vectors
  if (bg_by_cell$n > 0) {
    store_x_bg <- c(store_x_bg, bg_by_cell$x)
    store_y_bg <- c(store_y_bg, bg_by_cell$y)
  }
  # readline(prompt="Press [enter] to continue")
}

# Once our simulation is done, we can store these points as a point
# pattern object so we can analyze them
bg_pts <- ppp(x = store_x_bg, y = store_y_bg, window = eeg_window)

# You can see our "background" points by plotting them. Notice that here,
# our neural activity is taking place randomly across the "brain"
# plot(bg_pts, main = "Background Points, Loading 3, Alpha")
# plot(cells, add = TRUE)

# For each of our simulated background points, we next want to compute
# which cell each point is in using our find_cell() function
bg_cells <- find_cell(bg_pts$x, bg_pts$y)

# bg_cells

# Next, we want to compute the "intensity" of the points for each point
# landing in each background cell. Our Voronoi cells are different sizes,
# so I think one thing that might be useful is to scale the intensity
# based on the area of the cells. This way, the intensity will be for each

```

```

# 'unit area'.
bg_intensity <- df$readings[bg_cells]

# Now, we simulate:
# Create some vectors to store our simulated points
store_x <- c()
store_y <- c()

# Iterate through each background point
for (i in 1:bg_pts$n) {
  # Generate some offspring for background event i and place those points
  # in the Voronoi cell which contains that point

  offspring <- rpoispp(
    lambda = rexp(1,
                  rate = 1 / (bg_intensity[i]-shift_value)) /
                  areas[bg_cells[i]],
    win = cells[bg_cells[i]]
  )

  # If no offspring are created, move on. Otherwise, put them into the
  # storage vectors
  if (offspring$n > 0) {
    store_x <- c(store_x, offspring$x)
    store_y <- c(store_y, offspring$y)
  }
  # readline(prompt="Press [enter] to continue")
}

# Once our simulation is done, we can store these points as a point
# pattern object so we can analyze them
simulated_points <- ppp(x = store_x, y = store_y, window = eeg_window)

# Finally, we can plot the points
# plot(simulated_points, main = "Simulated Points, Loading 3, Alpha")
# plot(cells, add = TRUE)

mod_loading <- loccit(simulated_points, sigma = 0.15)

return_list <- list("sim_points" = simulated_points,
                   "mod_loading" = mod_loading,

```

```

        "cells"=cells, "bg_points" = bg_pts,
        "points" = points)

return(return_list)
}

mod_loading1_alpha <- speedy_calc(waveform = "beta", brainloading = 1)
mod_loading2_alpha <- speedy_calc(waveform = "beta", brainloading = 2)
mod_loading3_alpha <- speedy_calc(waveform = "beta", brainloading = 3)
mod_loading4_alpha <- speedy_calc(waveform = "beta", brainloading = 4)

psib_l1_alpha <- psib(mod_loading1_alpha$mod_loading)
psib_l2_alpha <- psib(mod_loading2_alpha$mod_loading)
psib_l3_alpha <- psib(mod_loading3_alpha$mod_loading)
psib_l4_alpha <- psib(mod_loading4_alpha$mod_loading)

psib_list <- list(l1_alpha = psib_l1_alpha, l2_alpha = psib_l2_alpha,
                 l3_alpha = psib_l3_alpha, l4_alpha = psib_l4_alpha)

simpts_list <- list(smpts_l1_alpha = mod_loading1_alpha$sim_points,
                  smpts_l2_alpha = mod_loading2_alpha$sim_points,
                  smpts_l3_alpha = mod_loading3_alpha$sim_points,
                  smpts_l4_alpha = mod_loading4_alpha$sim_points)

lapply(psib_list, range)

range(psib_l1_alpha)
range(psib_l1_alpha$marks)

psib_range <- range(unlist(lapply(psib_list, range)))

plot(as.listof(psib_list), zlim = psib_range, ncols = 1)
plot(as.listof(simpts_list), add=TRUE, ncols = 2)

plot(psib_l1_alpha, zlim = psib_range, main = "Loading 1, Beta")
#plot(mod_loading1_alpha$sim_points, add = TRUE)
plot(mod_loading1_alpha$cells, add = TRUE)

plot(psib_l2_alpha, zlim = psib_range, main = "Loading 2, Beta")
#plot(mod_loading2_alpha$sim_points, add = TRUE)

```

```
plot(mod_loading1_alpha$cells, add = TRUE)

plot(psib_l3_alpha, zlim = psib_range, main = "Loading 3, Beta")
#plot(mod_loading3_alpha$sim_points, add = TRUE)
plot(mod_loading1_alpha$cells, add = TRUE)

plot(psib_l4_alpha, zlim = psib_range, main = "Loading 4, Beta")
#plot(mod_loading4_alpha$sim_points, add = TRUE)
plot(mod_loading1_alpha$cells, add = TRUE)

# mod_cox4 <- kppm(mod_loading1_alpha$sim_points, trend = ~1,
#                 clusters = "MatClust")
# summary(mod_cox4)
# mod_cox4

# Next steps:
# (1) We want to adapt this code to the eeg data
# (2) We want to simulate points for brain loadings 1 and 4
# (3) We want to analyze the points and demonstrate how
# they're different
```

### 10.1.3 Homogenous/Inhomogenous Example

```
library(spatstat)

par(mfrow=c(1,2))

#homogenous
plot(rpoispp(500, win=square(1)), ncols = 2)

#inhomogenous
lambda <- function(x,y) { 500 * (x^2+y) }
X <- rpoispp(lambda, win=square(1))
plot(X, win=square(1))
```

