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Abstract

One way to analyze how our brains function is to apply statistical models to neural data. In 1998, Shadlen and Newsome observed that a balanced random walk integrate-and-fire model can model experimental data such as single-unit recordings of rhesus monkeys. Using this model, I study the spike count correlation of two neurons under varying conditions. Through simulations, I look at the relationship of spike count correlation across two neurons to firing input rate when the inputs are independent and correlated. After this step, I look at whether an oscillatory input affects synchrony, i.e., synchronous firing of two neurons in close temporal proximity. It is crucial to understand what affects synchrony as a little change in synchrony can cause huge impacts in cellular processes in the brain and in how neurons communicate with each other.
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1 Introduction

In the field of neuroscience, the general goal is to study how behavior is represented by neural activity. That is, how does the brain represent and process the actions, thoughts, and external inputs that will enable us, as individuals, to function? Computational neuroscientists want to analyze these neural activities. They want to understand the primary way neurons represent and communicate information in the nervous system such as spike trains, which are a sequence of neural discharges. One important contribution of statistics to the field of computational neuroscience is to apply statistical models to spike train data to distinguish the unknown signals from the noise. We apply these models so we can use their known properties to explain the data. Some existing computational models such as the leaky integrate-and-fire model or the Hodgkin-Huxley model are mathematical derivations from resistor-capacitor models.

In 1964, Gerstein and Mandelbrot introduced another model: the random walk model. They showed that the signal outputs for single neurons can be manifested in a simple integration of the incoming inputs from surrounding neurons to the target neuron. These inputs had equal strength on the target neuron to go up to the spike threshold, a threshold for which a neuron will spike, or go away from the threshold. Furthermore, M. Shadlen and W. Newsome (1998) then proposed the balanced random walk integrate and fire model (RW-IF) which could exhibit properties similar to those of experimental data. They showed that, with a few parameters, they could simulate spike discharges like those from single-electrode recordings from a single neuron.

Using models like the RW-IF model, computational neuroscientists can then study neural activity such as synchrony between neurons. In a recent study, M. Economo and J. White (2012) were interested in the irregular firing rates of neural discharges from cortical neurons. Using models, they looked at the increased oscillations that came from synchrony among neurons. It has been observed that many cortical neurons in close proximity often share oscillatory drive as well as having other independent inputs. They found that the oscillatory drive relative to the other inputs, which we consider “noise”, causes neurons to have excess synchrony. In addition, they found that the balance between the positive inputs that drive a neuron to spike and the negative inputs that does otherwise has an important effect on synchrony between neurons as well.

In this paper, I hope to illustrate the relationship for spike count correlations between two neurons for the balanced random walk integrate-and-fire model. A standard model for sequences of spike trains involves a “random walk of excitatory and inhibitory inputs (explained later). In particular, a simple balanced neural model (balanced between the amount of excitation and inhibition) can produce many characteristic features of recordings.

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1Kass, Eden, Brown (2013) p. 11, 22
2It should be noted that while these models can help understand the data better, it cannot fully represent it. Rather, the models help further explain, predict and understand the observable data. As the famous statistician G. Box once said, “All models are wrong but some are useful.”
3A probe that can measure the current from a single neuron
from rhesus monkeys (Shadlen & Newsome 1998). While there are many research papers that work with simple models such as the random walk integrate and fire model, there is very little research done in looking at the fluctuations in neural firing rates across two neurons and the correlated spiking behavior of those neurons. The main motivations for the project can be addressed in the following three questions of interest.

1. How do spike count correlations between neurons change as their firing rates fluctuate?

2. How does spike count correlation between neurons change when the inputs are themselves correlated with each other?

3. Does oscillatory inputs affect the synchrony of firing across pairs of neurons?

2 Background

Before I answer these questions, it is important to understand the biological background and terms related to the brain. In any given brain, there are more than $10^{10}$ neuron cells with $10^{14}$ connections (synapses) that link them together. Through these synapses, the neurons are able to send information signals from one presynaptic neurons (the neuron that sends a signal) to another postsynaptic neuron (the neuron that receives the signal). The signals from the presynaptic neuron stimulates an electrical shock in the postsynaptic neuron. This electrical pulse is called an action potential (see Figure 1a). The electrical potential of which the pulse goes up and down is called a membrane potential. The membrane potential will change dependent on the chemical ions that enter the postsynaptic neuron.

In the case of transmitting information, the presynaptic neurons send certain chemicals called neurotransmitters that can open ion channels that line the cell membranes of the synapses. The channels control the flow of ions such as $K^+$, $Na^+$, $Ca^{2+}$, and $Cl^-$ into the postsynaptic neuron. Some neurotransmitters trigger excitatory drive to the spike threshold, a threshold where it will trigger a spike, from the resting potential, the normal membrane potential state in the postsynaptic neuron. This is called an excitatory postsynaptic potential (EPSP). Some other neurotransmitters causes inhibitory postsynaptic potential (IPSP) in which the chemicals act as inhibitory inputs which drive the membrane potential down to the resting potential.

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4 For further information, refer to Trappenberg (2002)
5 Image from http://psychlopedia.wikispaces.com/action+potential
Figure 1: An action potential occurs when the membrane potential reaches the spike threshold. The spike threshold will reset to the resting threshold or some step below. In most cases, there will be a refractory period before the process restarts again. Each spike can be then assimilated into a spike train which is a binary representation of spike events in time where ones represent a spike and zeros represent a non-spike period.

The spikes from the postsynaptic neurons can be assimilated into a spike train (see Figure 1b). A spike train is a time-series of recorded spikes from an individual neuron in the brain. It is a binary representation of spikes on a time scale where ones will indicate a spike and zeros as non-spike. It is widely accepted that the brain decodes the spike rates and number of spikes in order to generate information about the outside world. Therefore, some computational neuroscientists seek to also decode the spike trains by utilizing statistical models to fit them to the electrical signals and analyze the spike trains.

These computational models, as mentioned before, can usually be derived from an electrical circuit. While they may some differences that make them unique, they all tend to follow the standard integrate-and-fire model. The change in membrane voltage can be derived as a function of the input current (I(t)) to the postsynaptic neuron and the capacitance C_m, the concentration of ions on either side of the cell membrane. The ions are integrated to find a net drive for the membrane potential.

\[ \frac{dV_m}{dt} = \frac{I(t)}{C_m} \]

In some cases, models include a refractory period, a period for which the membrane potential does not move for the duration of the period, so it can limit the frequency of spikes from occurring. In the case of the random walk integrate-and-fire model, it also has the same as the standard integrate-and-fire model. It differs in that, using a random walk towards the spike threshold, the membrane potential can go up and down with equal strength.

I begin by looking at theories about spike count correlation. A spike train analysis done

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6A process which, at each time step, the process moves randomly

7Further detail will be described
by R. Kass and V. Ventura (2006) revealed that spike count correlation between two simultaneously recorded neurons will often increase with time bins in recorded studies due to trial-to-trial variation. Through a careful proof about the formula for spike count correlation as a function of time bins and firing rate inputs, they were able to show the effects of trial-to-trial variation and length of time bins on spike count correlation. These trial-to-trial variation are often the results of “noise” in the models. The noise can be considered as the small fluctuations around the signals that the models generate. It is due to noise that there are varied spike counts for each trial. Without noise or trial-to-trial variation, the spike correlation becomes uncorrelated.

In addition, research by G. Vinci (2013) further revealed that the relationship between neurons cannot be simply explained by spike count correlation. Even if there is high correlation between firing input, the spike count correlation can still be close to zero. This is called the “attenuation effect”. This is important as it causes a phenomenon called “reversal problem” where the spike count correlation between two neurons will be greater when the firing input rate is lower than when the firing input rate is higher.

In the Assessment of synchrony in multiple neural spike trains using loglinear point process models, R. Kass, R. Kelly and W. Loh attempts to provide a statistical procedure to analyze synchrony. Using experimental primary visual data for an anesthesized monkey, they provided a quantifiable measure, $\zeta$, that would be the proportional gain in synchrony between two neurons’ spike trains when assuming that the expected gain is independent.[8] The equation for the quantifiable measure can be seen as a function of the spiking history $H$ and the observed number of synchronized spike:

$$\hat{\zeta}_H = \frac{N}{\int \lambda_1(H_1^1|t)\lambda_2(H_2^2|t)dt}$$

When $\zeta_H < 1$, there is suppressed synchrony (less synchrony than expected from the model) while $\zeta_H > 1$ indicates excess synchrony. Finally, when $\zeta_H = 1$, the synchrony is well explained by the model.

3 Poisson Process

A stochastic point process is a mathematical representation of a random process that generates intervals between points according to a stochastic rule (i.e. probability distribution). These points are discrete events that occur in a continuous time (e.g. action potential). In neuroscience, the stochastic point process is used to represent spike trains and the time between spikes is called inter-spike intervals (ISIs). Generally, the point process has memory which means the probability that a spike or event occurs is affected by the past. In addition, the interarrival times between the events can but do not necessarily have to be exponentially distributed.

[8] Please refer to Kass, Kelly, & Loh (2011) for further and more complete explanation about the theory to measure synchrony
One important example of a point process is the Poisson point process. In the homogenous case, the probability of an event occurring is independent of the time. For a homogenous Poisson process with rate $\lambda$, the intervals between events are i.i.d. $Exp(\lambda)$ and are memory-less, the chance of having another draw is not dependent on the past draws\(^9\). In addition, the expected number of spike counts $\mu = \lambda \cdot \Delta t$ which means that the mean is proportional to the length of the specified time interval.

4 Models

In order to examine the neural firing rate properties, it is important to understand the parameters of the model themselves first. Here, I briefly describe the models proposed by Shadlen and Newsome as well as the Leaky Integrate-and-Fire model that is more widely studied and used to describe experimental data.

4.1 Random Walk Integrate-and-Fire model

We focus on a simplified integrate-and-fire model with decay introduced by Shadlen and Newsome based on a random walk. Similar to a normal integrate and fire model, the neuron fires when it reaches a spike threshold of -55 mV and will reset immediately back to its resting potential of -70 mV or one step (1 mV) below it. Unlike many other models, it is important to note that the RW-IF model does not have a refractory period other than the reset to the one step below the resting potential. There is an exponential decay with time constant $\tau$ (20 ms).

\[ E (+1) \quad I (-1) \]

Figure 2: For each neuron, we have a balanced network of inputs (neurons) that will be integrated into the target neuron. These 600 presynaptic inputs will have ISIs drawn from an exponential distribution to follow the Poisson Process

As seen in figure 2, we have a graphical representation of the inputs (surrounding neurons that affect the target neuron) that will have either have one EPSP/IPSP affect the target neuron. Each excitatory (orange circles) or inhibitory input (green circles) will increment/decrement by one step respectively towards the firing threshold for the target neuron. In addition, each input has an ISI drawn from an exponential distribution (refer to raster plot in Figure 3a) which follows the Poisson process. We enforce a bound in which the inhibitory input cannot hyperpolarize the neuron beyond the resting potential. It is important to note that we have a balanced network of excitatory and inhibitory (300 input neurons each) synaptic inputs to a neuron. With balanced inputs, the firing of spikes is caused by the irregular fluctuations. The balance of excitatory and inhibitory inputs along with the reflecting barrier creates the irregular spike patterns of those from experimental data.

4.2 Leaky Integrate-and-Fire model

The leaky integrate-and-fire model is one of the most widely used and studied models due to its simple spiking neuron model. A neuron can be modeled from a simple linear differential equation:

\[
\tau_m \frac{dv}{dt} = -\left( v(t) - E_L \right) + RI(t)
\]

where \( \tau_m \) is the membrane time constant, \( R \) is the membrane resistance, \( E_L \) the resting potential, \( v(t) \) the membrane voltage potential as a function of time \( t \), and \( I(t) \) the input current. As a "leaky integrator" of its input \( I(t) \), it can capture the spiking effects of the sodium and leakage channels as well as the voltage decay. Although the spiking property of resetting are not seen in the experimental neural data, the LIF model has similar effects where the membrane potential will "reset" to the resting potential once it reaches the spike
threshold. We can also add a refractory period where the leaky integration process will restart after some duration of the absolute refractory period delay.\[10\]

5 Spike Count Correlation

5.1 Derivation

As stated in the introduction, I am interested in using simulations to illustrate the relationship between spike count correlations with firing rate inputs and time bins. Using the following two simple assumptions introduced from Kass and Ventura (2006), Vinci derived a general equation for correlation of two spike counts \( Y^1_r, Y^2_r \) that is dependent on time bins \( T \), trial-to-trial firing rate correlation \( \rho \), conditional expectation and variance of spike counts on the input firing rate \( X^1_r, X^2_r \) for simulation trial \( r \).\[11\]

1. Within trials, the expected spike counts increase proportionally to \( T \)

2. The within-trial variance is proportional to the within-trial expectation

Since we are working with Poisson process, we can assume that the fano factor \( k = 1 \).

We first start with the conditional expectation and conditional variance:

\[
E[Y^i_r | X^i_r] = TE[X^i_r] \tag{2}
\]

\[
Var[Y^i_r | X^i_r] = k \cdot E[Y^i_r | X^i_r] = kTE[X^i_r] \tag{3}
\]

From these definitions of conditional expectation and variance, we can then derive the variance and covariance:

\[
Var[Y^i_r] = E[Var[Y^i_r | X^i_r]] + Var[E[Y^i_r | X^i_r]]
= kTE[X^i_r] + T^2Var[X^i_r]
= T^2 \left( \frac{k}{T} E[X^i_r] + Var[X^i_r] \right)
\]

\[
Cov[Y^1_r, Y^2_r] = E[Cov[Y^1_r, Y^2_r | X^1_r, X^2_r]] + Cov[E[Y^1_r | X^1_r], E[Y^2_r | X^2_r]]
= 0 + Cov[E[Y^1_r | X^1_r], E[Y^2_r | X^2_r]]
= T^2 Cov[X^1_r, X^2_r]
\]

Finally, we have the general correlation equation to be:

\[
Cor(Y^1_r, Y^2_r) = \frac{Cov(Y^1_r, Y^2_r)}{\sqrt{Var[Y^1_r]Var[Y^2_r]}}
= \frac{T^2 Cov[X^1_r, X^2_r]}{\sqrt{T^2(\frac{k}{T} E[X^1_r] + Var[X^1_r]) T^2(\frac{k}{T} E[X^2_r] + Var[X^2_r])}}
= \frac{\rho T}{\sqrt{(T + \omega^1)(T + \omega^2)}}
\]

\[10\] Trappenberg (2010) p. 54
\[11\] Kass and Ventura (2006)
where:

- $\rho = Cor(X^1, X^2)$
- $\omega^i = \frac{E[X^i]}{\text{Var}[X^i]}$

From the correlation equation, I expect that the correlation with spike counts will increase with input firing inputs. In addition, I believe that the correlation of spike counts will increase with time bins and rho.

5.2 Test of Assumptions

As stated from the derivation of the spike count correlation, the spike count correlation formula is based on two assumptions. In papers from Shadlen & Newsome (2006) and Averbeck & Lee (2003), observed data from Macaque monkeys and many other studies have shown that variance of spike counts is proportional to the expected spike count. In addition, as time bins increases, the expected spike counts increase. To test this, I randomly drew 300 time bins and simulated 100 trials per time bin. For each time bin, I calculated the mean spike counts and plotted against the time bin inputs. As seen below, the expected spike counts increase with time bins.

![Expected spike count against Time bins (mu = 50)](image)

Figure 4: Expected spike count against time bins
In order to show this, I randomly simulated firing rates from 20 to 60 (spikes/second) and repeated for 100 trials. As seen below, the variance of the spike counts increase as firing rates increase which is consistent with Assumption 2.

Figure 5: Variance against expected firing rate
5.3 Input Firing Rate Equals Output Firing Rate

One important assumption in the simulation is that the firing rate of the inputs will result in the same firing rate from the target neuron. In the simulation, I will increase the expected firing rate of the inputs ($E[X^r_i]$) and we expect that the spike count correlation to change as well. However, I needed to check whether there were any additional effect on the output firing rate as I changed the input firing rate. As seen in Figure 6, we see that the output firing rate increases linearly with the input firing rate.

![Neuron’s output firing rate vs input firing rate](image)

Figure 6: Input firing rate for surrounding neurons in the RW-IF model result in the firing rate for the target neuron
5.4 Fano Factor of Spike Counts is One for Poisson Counts

Another important assumption in the correlation proof is that the fano factor of the spike counts given the input firing rates is one for Poisson counts. The fano factor (k in the correlation proof) is a measure of variation. In the general case, the fano factor is the variance over the mean:

\[ F = \frac{\sigma^2}{\mu} \quad (4) \]

In Poisson process, the fano factor would be one as the variance will linearly increase with the mean spike counts. This can be seen in the graph below.

![Fano Score: Mean vs Var](image)

Figure 7: We see a plot of the fano factor. We see that the mean spike counts increases linearly with the variance of the spike counts. For poisson process, the variance in the spike counts will equal roughly to the mean in the spike counts.

6 Simulation setup

6.1 Spike Count Analysis

In order to observe how spike count correlations between two neurons change given independent and correlated firing inputs, I ran four simulations. The first three simulations work with independent firing inputs and the fourth with correlated inputs. Each firing input (\(\mu\))
ranges from 20-80 impulses/sec by steps of five. For each run in the simulation, I set up 100 independent trials where each trial generates spike count outputs from two independent neurons and found the correlation between the two neurons spike counts. Each neuron is given a firing input rate that is determined as follows:

1. Constant Input
2. Inputs drawn from independent normal distribution: \( \lambda \sim N(\mu, 2) \)
3. Inputs drawn from independent multiplicative distribution: \( \lambda \sim \mu \cdot \exp(\alpha), \alpha \sim N(0, 2) \)
4. Inputs drawn from bivariate normal distribution: \( \lambda \sim N(\mu, \Sigma), \mu = \begin{pmatrix} \mu_1 \\ \mu_2 \end{pmatrix}, \Sigma = \begin{pmatrix} 4 & 4 \rho \\ 4 \rho & 4 \end{pmatrix} \)

For each firing rate, I ran the 100 independent trials 100 times to find an average correlation. I then plotted the average correlation against the firing input rate. It should be noted that the input firing rate variation are held constant for all cases (\( \text{Var}[X_i^r] \)). For future work, it will be interesting to explore varying the input firing rate variation and increasing the proportion of \( \omega \).

### 6.2 Synchrony Analysis

I also conducted simulations to study synchrony by introducing an oscillatory drive to the inhibitory inputs. Since I am interested in whether the oscillatory input affects the synchrony between neurons, I will look at the proportion of observed synchronous spikes over the total number of spikes from both neurons. I would expect that the proportion of synchronous spikes when the model has oscillation will be greater than the model without. I ran 100 independent trials for a certain amplitude (20 - 40). For each neuron, the excitatory inputs and a percentage of the inhibitory inputs are independent. The independent inputs have firing rate (\( \mu \)) set to 40 spikes per second. The remaining percentage of the neuron’s inhibitory inputs will share oscillatory input with the other neuron. To include an oscillatory input, I generated a cosine-tuning curves over 3000 ms with a base of 10 mV to avoid going below the resting potential:

\[
A \cdot \cos(2\pi ft + \phi) + 10
\]  

(5)

The frequency (f) was always set to 40 Hz. The starting phase value for both neurons (\( \phi \)) was drawn from an uniform distribution \([\pi, \pi]\) and it is redrawn for every trial. I apply the thinning algorithm to reject the spike events that were unlikely to occur given the oscillatory input. The thinning algorithm is an acceptance-rejection procedure in which we selectively take spikes in accordance to an oscillating input. The key idea is to choose a constant rate function \( \lambda_u(t) = \lambda_u \) for which we can generate a nonhomogenous Poisson process. In our case, this is our input firing rate. The acceptance-rejection algorithm for the thinning procedure will as follows:
1. Generate a sinusoidal wave
2. Draw inputs from an exponential distribution given time \( t \)
3. Generate uniform distribution \((u_1)\) independent of exponential distribution
4. Accept \( \lambda(t)/\lambda_u/ \) greater than \( u_1 \)

From figure 4b’s raster plot, we can see the inputs have been drawn accordingly to oscillating input. Once I have generated 100 independent trials, I implemented Pengcheng Zhou’s code to compute \( \zeta \), the measure of synchrony fit\(^\text{12}\). I then ran the same simulation again but this time gave the shared inhibitory inputs constant inputs with rate \((10 + \text{amplitude})\) spikes/second \((10 \text{ from the addition of a base in the cosine wave})\). I made sure that the percentage of shared inhibitory inputs that have constant, but slightly increased rate, in order to be able to compare to the model with oscillatory drive.

7 Results

After verifying the assumptions are met for the spike count correlation, we can now execute the simulations described previously. The results can be broken into two sections: spike count correlations and synchrony.

7.1 Spike Count Correlation relationships

From figure 9, we see that the spike count correlation seems to be independent of input firing rate when the input is constant among trials. Specifically, as input firing rate increases, the spike count correlations tends to approach a correlation of 0. This holds with results found by R. Kass and V. Ventura as there needs to be trial-to-trial variation. This also follows Giuseppe’s proposal in which there could be an “attenuation effect” when the correlation between firing rate inputs is one but there is zero correlation between the spike counts. This is due to the variability of the spike counts between trials to be close to zero.

\(^{12}\)The formula for \( \zeta \) can be found in the background section
When trial-to-trial variation is introduced when the firing inputs are drawn from the normal and multivariate distribution, we observe a negative association between spike count correlation and firing inputs. This again follows the correlation proof as we increase the firing rate input ($E[X_i^r]$) and keeping the time bins, firing input rate correlation and variance constant. It should be noted that the time bins was set to 3000 ms, sigma to $\sqrt{4}$, and rho as 1 (same inputs for both neurons) in all 3 of the simulations. In addition, if we were to increase the time bins ($T$) while keeping the rest of the variables constant, we observe an increase in spike count correlation which follows expectations.
(a) Firing rate inputs drawn from a normal with constant time bins (3000 ms) and variance(2)

(b) Firing rate inputs drawn from a multivariate distribution with constant time bins (3000 ms) and variance (exp(4))

Figure 9: Simulations of the two independent simulations follow the derived correlation equation. Left: Firing input rate drawn from a normal with constant variance of 2. Right: Firing input rate drawn from a multivariate normal with constant variance of 2

Figure 10: Spike count correlation as a function of increasing time bins with constant firing input rate and variance
From figure 12 below, when I correlated the inputs between neuron 1 and neuron 2, we see that the spike count correlation increases with rho as expected from the correlation equation. From Giuseppe’s paper, he observed that the SSCC is always less than FRC (rho). In the case of the RW-IF model, this was also observed as well. From figure 12b, we see that the correlation between firing inputs is always greater than the spike count correlation. In addition, when we increase the expected firing input rate, the spike count correlation decreases. It should be noted that while most literature indicates that the spike correlation increases with firing input rate, they are increasing the net firing input to the neuron. In my simulation, I increased the expected firing input rate to each presynaptic inputs and kept the variance constant. For future work, it will be interesting to increase the proportion of expected and variance firing rate input (ω), i.e. not keeping the input rate variance fixed. We would then expect the spike count correlation to increase with firing input.

(a) Firing rate input vs spike count correlation
(b) Correlated inputs (p) vs spike count correlation with rho and time bins held constant

Figure 11: Simulation with correlated inputs. Using inputs drawn from a bivariate normal, we can increase the correlation between firing rate inputs. As the correlation between the firing inputs increase, the correlation between spike counts also increase as well.
### 7.2 Synchronous neural activity

I then moved on to observe how introducing an oscillatory drive affects synchrony. After implementing a shared oscillatory input for the inhibitory inputs, I used Pengcheng Zhou’s code for analyzing synchrony to see the effects. When 40% of the inhibitory inputs (120 inputs) had shared oscillatory input and given amplitude of 30, the observed number of synchronized spikes can be seen:

```
################# synchrony resolution: 0.006 #################
```

Observed number of synchronized spikes: 312.000

Predicted number with PSTH+HISTORY model: 543.0000

\[ \text{zeta: 0.5746} \]

The average total number of spikes for all 100 trials for neuron 1 was 518 and neuron 2 was 520. We would then have a proportion of \( \frac{312}{1038} = 0.3006 \). When we have implemented the simulation but have a percentage of shared inhibitory inputs to have constant input (the base membrane potential of 10 + amplitude of 30 = 40), the observed number of synchronized spikes can be seen:

```
################# synchrony resolution: 0.006 #################
```

Observed number of synchronized spikes: 53.000

Predicted number with PSTH+HISTORY model: 49.0000

\[ \text{zeta: 1.0816} \]

The average total number of spikes for all 100 trials for neuron 1 was 156 and neuron 2 was 158. We would then have a proportion of \( \frac{53}{314} = 0.1688 \). From these results as well as many others, we see that the proportion of the synchronous spikes is greater with oscillations than when there is no oscillation in the model. We have similar cases when we changed the amplitude of the cosine wave. This shows that an oscillatory input drive will increase synchrony among neurons.

### 8 Discussion

We have shown that the RW-IF model, while a simple model, can be implemented as a model for experimental data. From the simulation results, the RW-IF follows results expected from studies. For a balanced random walk integrate and fire model, the spike count correlation increases with time bins. However, when there are no trial-to-trial variability, there will be no correlation between spike counts as firing input rates increases. When we impose correlation among the firing rate inputs, we see that spike count correlation increases as well.

As suggested by Vinci (2013), the input firing rate correlation was greater than the spike count correlation. This could lead to a behavior in which he calls “attenuation effect”. An
The extreme case of the “attenuation effect” is when the firing input correlation is one and the spike count correlation to be zero. When I increased the firing input rate and kept the variance constant, we only increased $\omega$ slightly so we do not see this case clearly when we correlated the inputs. However, if I were to increase $\omega$ by a large amount, I would be increasing the denominator of the spike count formula. At a certain stimulus, the input firing rate correlation would not effect the spike count correlation. This would lead to the problem called “reversal effect” where the spike count correlation does not increase with firing input. Instead, it will decrease as firing input increases.

While the RW-IF model is a simple model and concept, there are certain limitations for which the model excludes. As discussed by Shadlen and Newsome, many of the biophysical properties of an actual neuron have been ignored. The excitatory and inhibitory synaptic events count the same and therefore I do not consider any inputs that have no effect on the postsynaptic neuron. Another important aspect is that a refractory period is not implemented other than a one step below the resting potential when the membrane potential fires.

At first, I explored the effect of magnitude on synchrony when both excitatory and inhibitory had shared oscillatory inputs. However, there was no indication that there was excess synchrony when we introduced the oscillation. In fact, Shadlen and Newsome (1998) had also explored briefly the effect of shared constant inputs on synchrony. They found that there was no increase in synchrony when there were shared inputs. They attributed to the fact that this was a “high-input” regime$^{13}$ (where we have 300 excitatory and inhibitory inputs) where the abundance of inputs were effectively synchronous. However, in a later study by Salinas and Sejnowski (2000), they claimed that it was due to the correlation parameters that Shadlen and Newsome had set for the excitatory/inhibitory inputs that resulted in no increase in synchrony. They hypothesized that the fact that the correlation between excitatory and inhibitory were the same resulted in reduced variance. Therefore, from the literature and the results, we can see that the magnitude of the oscillatory drive as well as the parameters such correlation between excitatory/inhibitory inputs are very important in determining the output firing rate.

In the future, I hope to move forward with the simulations for synchrony. In particular, I hope to explore how correlated inputs for just excitatory or just inhibitory inputs affect the synchrony of firing across pairs of neurons. I will explore how the synchrony changes with the length of time observed as well as with firing rate input variation. Another interesting exploration would be to look at how shared inputs affect the synchrony of spikes. Since this was a process of learning the area of neural computation and spike train analysis, I was not able to fully apply all the model analysis and further work could be addressed in detail.

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$^{13}$In common situations, neurons only receive less than 100 inputs
References


