Realistic alpha oscillations and transient responses in a cortical microcircuit model

Andres A. Kiani^{2,4}, Theoden I. Netoff^{1,3}, Geoffrey M. Ghose^{1,2,4}

1 Center for Neuroengineering, University of Minnesota, Minneapolis, Minnesota, United States of America.

2 Center for Magnetic Resonance Research, University of Minnesota, Minneapolis, Minnesota, United States of America.

3 Department of Biomedical Engineering, University of Minnesota, Minneapolis, Minnesota, United States of America.

4 Department of Neuroscience, University of Minnesota, Minneapolis, Minnesota, United States of America.

* kiani006@umn.edu, tnetoff@umn.edu, ghose@umn.edu,

Abstract

Neural-mass modeling of neural population data (EEG, ECoG, or LFPs) has shown promise both in elucidating the neural processes underlying cortical rhythms and changes in brain state, as well as offering a framework for testing the interplay between these rhythms and information processing. Models of cortical alpha rhythms (8 - 12 Hz) and their impact in visual sensory processing have been at the forefront of this effort, with the Jansen-Rit being one of the more popular models in this domain. The Jansen-Rit model, however, fails in reproducing key physiological observations including the level of inputs that cortical neurons receive and their responses to visual transients. To address these issues we generated a neural mass model that complies better with

synaptic mediated dynamics, intrinsic alpha behavior, and produces realistic responses. The model is robust to many changes in parameter values but critically depends on the ratio of excitation to inhibition, producing response transients whose features are dependent on this ratio and alpha phase and power. The model is sufficiently flexible so as to be able to easily replicate the range of low frequency oscillations observed in different studies. Consistent with experimental observations, we find phase-dependent response dynamics to both visual and electrical stimulation using this model. The model suggests that stimulation facilitates alpha at particular phases and suppresses it in others due to a phase dependent lag in inhibitory responses. Hence, the model generates insight into the physiological parameters responsible for intrinsic oscillations and testable hypotheses regarding the interactions between visual and electrical stimulation on those oscillations.

Author summary

While there is increasing evidence of the fundamental role brain states play in shaping information processing in the cerebral cortex, a mechanistic understanding of how those brain states are manifested and alter the signals underlying sensory processing and decision making has proved challenging. To address this issue we have modeled a well established signature of inattention in visual cortex: synchronized low frequency (8 - 12 Hz) oscillations. To allow for inferences regarding the local generation of these rhythms within a cortical microcircuit we used a neural mass model approach that incorporates physiologically realistic interactions between 3 populations of neurons. Our model is able to explain a variety of experimental observations that previous neural mass models have not, including spontaneous rhythms in the absence of input, the faithful transmission of strong input transients, a range of oscillation frequencies, and phase dependent visual responses. The model is robust to a range of parameters, but critically depends on the balance between local excitation and inhibition.

Introduction

Many neural recording techniques, such as electroencephalograms (EEG), electrocorticogram (ECoG), and local field potentials (LFPs), record population-level activity that directly reflects information processing and behavioral state. These signals can exhibit complex spatiotemporal patterns in response to stimuli, cognitive state, and behavioral changes. [67] An understanding of the underlying dynamics producing these complicated signals is further complicated by the complex nature of the biological neural networks implicated in their production, with diverse classes of neurons whose interactions are mediated by the sophisticated interplay between channel, membrane, and synaptic dynamics. [32] The difficulty of the problem is moreover amplified by the interaction of these measured signals with non-neuronal tissues. [13]. Consequently, uncovering the underlying processes associated with these signals has been an immense challenge.

In view of these difficulties, researchers have turned toward the development of computational models that are able to capture the overlying dynamics including various phenomena such as the alpha rhythms of the visual cortex, whilst being capable of making inferences regarding underlying processes like excitation-inhibition balance. [23] Various modeling approaches have been endeavored in this domain, out of which the Neural-mass modeling approach has become widely popularized in the literature. [66] “Neural-mass” or “Mean-field” computational models, first introduced by Wilson and Cowan, adopt a reductionist approach by modeling the activity of interconnected populations of inhibitory and excitatory cells through coupled non-linear differential equations. [14] Such simplified models, when they include non-linear and delayed interactions between idealized sub-populations of neurons, can successfully model the dynamics of local field potentials. [61]

For example, Lopes da Silva et al., proposed one of the earliest models using coupled populations of excitatory and inhibitory neurons to simulate the generation of alpha rhythms (8 - 12 Hz), long associated with inattention in EEG studies. [59, 53] Jansen and Rit extended this model by adding a pyramidal population to explain how such rhythms within a local cortical population of the visual cortex are generated and how

the circuit is affected by incoming signals. [29, 31]

Although the Jansen-Rit (JR) model successfully models how alpha rhythms are modulated in a phase dependent manner by stimulation, it does not accurately simulate several aspects of cortical responses.[41] First, alpha rhythms are largely associated with relative quiescence [1, 15, 36]: the first observations of cortical alpha were obtained from subjects with their eyes closed in the absence of any visual stimulation.[6] However, stable oscillations in the absence of parameter manipulations emerges in the JR system when driven by drive above 155 Hz.[23, 30] Second, the stable oscillations come at the cost of realistic response dynamics to input transients, which are largely lost, or substantially altered, in the output of these models [41, 17]. For example, stable oscillations in the JR system are seen at input rates as high as 315 Hz, precluding the faithful transmission of high input transients.

A related issue is the mismatch between received input and output rates, which is a necessary self-consistency constraint given the interconnectedness of cortical columns both within and between areas. Cortical regions have roughly the same structure, consist of similar neuronal species, and exhibit similar dynamics at a local level. [21] While primary visual cortex (V1) receives primary input from thalamic neurons, all other visual areas, including V2, V4, MT, and IT, receive their input from other cortical areas [27, 47]. Therefore, input and output rates should be comparable, if not the same, in an adequate cortical model. As it stands, the upper bound imposed on a population output in the Jansen-Rit is 5 Hz. The average maximum single unit firing rate in both afferent V1 and efferent V4 neurons is around or below 50 Hz to a preferred stimuli [45, 65]. Therefore, it stands to reason that the average maximum output rate across a cortical population (representing thousands or millions of neurons) during a visually evoked response should be some factor lower than that of the average single unit due to the vast diversity of feature and spatial preferences represented across the ensemble.

Another critical flaw in the Jansen-Rit model is the linear mapping between input rates and synaptic drives, a clear contradiction from synaptic mediated dynamics and inconsistent with non-linearities observed in visual receptive fields [57]. The linear relationship between input rate and synaptic drive in potential results in linear response

dynamics that do not include thresholds, saturations, and attenuations, all of which are dynamical features observed in biological neural networks. The nonlinear nature of membranes and synapses is responsible for the emergence of multiple stable states and the ability to transition between distinct modes of response, all of which are hindered in this linear regime.

A final concern is related to the Jansen-Rit's inability to generate the different frequencies observed in neural recording data, even though the population time constants of the model were intended to serve this purpose. Previous studies have either introduced additional populations, or additional connections with associated connectivity factors, as an attempt to resolve this problem.

In this manuscript we show that simple physiologically informed modifications of the classic three population model can address all of these limitations. Like the Jansen-Rit model, this new model exhibits alpha rhythm activity that can be disrupted by input; however, in this model the rhythms are only prevalent when inputs are relatively inactive and are notably absent in the regime of strong input, allowing for a faithful transmission of input transients. The model is self consistent, in that input and output rates are comparable, and incorporates the fact that the effects of external drive on membrane potentials are synaptically mediated. Additionally, we formulate a relationship between the model's time constants and synaptic capacitances that effectively resolves the frequency problem, allowing the three population model to exhibit the myriad of physiologically relevant frequencies. Finally, because oscillations are only observed in particular coupling regimes, the model suggests that cortical function is strongly dependent on the maintenance of excitatory-inhibitory coupling ratios over a limited range.

Results

In this paper we introduce the self-consistent intrinsically oscillating microcircuit (SCIOM) model. The model is an extension of the Jansen-Rit three population model of cortical microcircuitry (Fig. 1) with a combination of structural and parameter changes

to create a more physiologically realistic model that is able to explain critical 89
 experimental observations. Unlike the classic Jansen-Rit model, it produces intrinsic 90
 alpha rhythms that are suppressed with increased external inputs. Unlike the 91
 Jansen-Rit, there are both physiological bounds on inputs and consistency between 92
 input and output rates. And unlike the Jansen-Rit, responses to inputs are mediated by 93
 nonlinearities akin to the synaptic nature of biological neural networks. 94

Fig 1. Block diagram schematic of the Jansen-Rit model (A) and the SCIOM model (B). New components added to the Jansen-Rit model to create the SCIOM model are shown in the green box in panel B. The connection between populations is shown as a sequence of arrows. These arrows depict the drive from the presynaptic population to the postsynaptic population. Dashed arrows indicate drive in terms of rate, while solid arrows indicate drive in terms of electric potential. Blocks indicate transformation for synaptic and sigmoidal functions. Ellipses indicate coupling coefficients that linearly scale drive. Stadiums (interneurons) and triangles (pyramidal) symbolize the populations in terms of their second order membrane potential function. The operator symbol indicates the superposition of drives from afferent sources.

First, we will examine several representative equilibrium curves of the Jansen-Rit and the SCIOM systems that illuminate important features and highlight key differences

and similarities between the two models, with particular focus on the presence of spontaneous oscillations in the absence of external drive and their suppression with increased drive. We will then adopt an energy approach to gain insight into the parameters and features of the SCION model responsible for the emergence of intrinsic oscillations and the interactions of these oscillations with visually evoked transients. We use this energy approach to map how the power and frequency of those oscillations depends on particular parameters.

Equilibria and State Transitions

The first experimental observation we focus on is the presence of intrinsic oscillations, which occur in the absence of significant input and whose magnitude decreases with strong inputs so as to allow such inputs to propagate. Both of these observations are consistent with experimental observations in visual cortex. We therefore derive equilibrium curves of the local field potential, Y , as a function of external input levels, P (Eq. 25). Consistent with previous work, and experimental data demonstrating the dominance of synaptic potentials in local field potentials, we define this Y as the difference between the excitatory and inhibitory membrane potentials. Doing so allows one to partition equilibrium curves into four segments: the high and low fixed point segments (**Fix_L** and **Fix_H**), a separatrix (**SP**), and the stable limit-cycle segment (**LC**).

In Fig. 2A we see that the standard parameter Jansen-Rit equilibrium curve's **LC** domain does not include $P = 0$ (i.e., it does not intrinsically oscillate), but rather stable oscillations are only observed with high levels of input drive. Furthermore, within the first half-domain of **LC**, increasing P results in the strengthening of the system's oscillatory power ($\partial|\Psi| > 0$), indicated by the green shading. The waxing and waning of alpha power with increasing P across the two aforementioned subsegments of **LC** is observed in simulations of the standard system, shown in the inset. Moreover, the end of **LC** occurs at $Y(P = 320 \text{ Hz}) = 8.00 \text{ mV}$, which means that any external input function $P(t) \leq 320 \text{ Hz}$ will be effectively attenuated by the system.

These failures to reproduce appropriate oscillatory or limit cycle behavior led us to alter the coupling parameters of the original Jansen-Rit model to investigate how intrinsic

oscillations that decay with strong input might be created.

With respect to inhibition, we found that increases in either of the inhibitory coupling weights (the inhibitory to pyramidal connection and the pyramidal to inhibitory connections: C_{IP} and C_{PI}) produce a positive translation in the **LC** segment about the P coordinate of the Y vs. P phase-space portrait, implying that greater external drive, P , is required for producing stable oscillations. Thus there is an upper bound, $C_{\Sigma I}^{\text{FixL}}$, for net inhibition beyond which intrinsic oscillations ($P = 0$) do not occur and the system inevitably converges to **FixL**. Increased inhibition also increases the range over which **LC** is observed and there is therefore a lower bound, $C_{\Sigma I}^{\text{FixH}}$, for net inhibition, below which **LC** is not observed and the system inevitably converges to **FixH**.

As would be expected given the definition of LFP signals as the difference between excitatory and inhibitory potentials, changes in excitation have largely opposite effects than to inhibition. Increases in either of the excitatory coupling weights (the excitatory to pyramidal connection and the pyramidal to excitation connection: C_{EP} and C_{PE}), produce a negative translation in the **LC** segment about the P coordinate of the Y vs. P phase-space portrait, suggesting that less external drive, P , is required for producing stable oscillations. Thus there is a lower bound, $C_{\Sigma E}^{\text{FixL}}$, for net excitation beyond which intrinsic oscillations ($P = 0$) do not occur and the system converges to **FixL**. Decreased excitation also increases the range over which **LC** is observed and there is therefore an upper bound, $C_{\Sigma E}^{\text{FixH}}$, for net excitation, above which **LC** is not observed and the system converges to **FixH**.

Irrespective of class (excitatory or inhibitory), there is a slight asymmetry in the impact that pyramidal to interneuronal couplings have on **LC** because the firing-rate nonlinearity, S_F , operates on C_{PE} and C_{PI} , but not on C_{EP} and C_{IP} in the Jansen-Rit formulation. Specifically, because $|\partial S_F| > 1$ for most of the responsive domain of S_F , C_{PE} and C_{PI} are more sensitive than C_{EP} and C_{IP} . Note that this is not the case in standard parameter Jansen-Rit.

These observations characterize the dependency of the system's oscillatory behavior on the coupling between the three populations. They were utilized to derive coupling combinations that result in intrinsic oscillations that decay in power with the

introduction and intensification of input and that disappear within $P \leq 2e_o$, where $2e_o$ is the upper bound of, S_F .

The equilibrium curve in Fig. 2B illustrates the common dynamical features possessed by Jansen-Rit coupling configurations that support: intrinsic oscillations, as indicated by the intersection of **LC** and the vertical axis at $P = 0$); their decay ($\partial|\Psi| < 0$) with increased input, indicated by the pink shading across the positive P domain of LC; and their dissolution within the bounds of the firing-rate nonlinearity, S_F , illustrated by the termination of the **LC** segment within $P \leq 2e_o$.

Fig 2. Equilibrium curve transition from the standard Jansen-Rit model to the SCIOM model. The standard Jansen-Rit's equilibrium curve, Y^*_{JRSTD} , as a function of external input, P . The curve is divided into the four segments: low amplitude fixed point, Fix_L , the bistable region SP , the stable limit-cycle LC , and high amplitude fixed point Fix_H . The curve exhibits an initial Hopf-Bifurcation $Y(P = 89) = 6.74$ and then a subsequent Hopf-Bifurcation at $Y(P = 320) = 8.10$, defining the boundary of the limit-cycle. The LC is further divided into two segments according to the relationship between oscillatory power ($|\Psi|$) and external drive, P . The green shading indicates a positive relationship between $|\Psi|$ and P such that the oscillation grows with increasing P , while the pink segment indicates a negative one, where the oscillation decreases with increasing P . The inset shows time-series corresponding to points on the the equilibrium curve with constant input-rates of $P \equiv 0, 89, 200$, and 320 Hz. (B) The parameter adapted Jansen-Rit model, $Y^*_{JR_e}$ exhibits an initial Hopf-Bifurcation $Y(P = -34) = 6.56$ and a subsequent Hopf-Bifurcation $Y(P = 45) = 7.20$. For reference, Y^*_{JRSTD} is plotted in gray. (C) The parameter adapted Jansen-Rit model, $Y^*_{JR_eo}$, with an adapted S_F nonlinearity in black, plotted over the standard S_F nonlinearity in grey. Note that the input drive range in this panel has been narrowed to the range of physiological drive. (D) Presents a SCIOM equilibrium curve, Y^*_{SCIOM} . Note the stepwise structure between the partitions: Fix_L and LC , divided by steps: II_1 , II_2 , and II_3 where the system evolves independently of P .

Although there are many integer-valued coupling configurations with identical net
excitation and inhibition coupling ratios (perhaps infinitely many) that satisfy these
three conditions on LC , there are very few coupling configurations with different

164
165
166

integer-valued coupling ratios that do. Choosing to raise S_F 's upper bound from the
 $2e_o = 5$ Hz to $2e'_o = 9$ Hz so that outputs better reflect the magnitude and dynamics of
inputs, also increased the range of excitatory-inhibitory coupling ratios that result in
intrinsic oscillatory behavior. The left inset in Fig. 2C compares the graph of S_F with
an upper bound of 5 Hz to that with an upper bound of 10 Hz. And in Fig. 2C we
produce an example equilibrium curve of parameter modified Jansen-Rit systems with
this heightened S_F that satisfy the conditions we have set for intrinsic oscillatory
behavior.

Even though a number of conditions are satisfied with these parameter modifications, as
we will discuss later, they are not sufficient to replicate the dynamics of realistic
response transients. In particular, such systems are not capable of producing nonlinear
transformations of input that are physiologically appropriate.

Therefore, we derive equilibrium curves of the local field potential, Y , as a function of
external input levels, P , for the SCIOM model (Eq. 36). Partitioning SCIOM
equilibrium curves in terms of the four equivalence relations: **Fix_L**, **SP**, **LC**, **Fix_H**;
highlights several distinctive features relevant to the SCIOM family of equilibrium
curves not seen in Jansen-Rit, as is shown in Fig. 2D. Particularly apparent are the
step-wise divisions between the input-dependent segments of Y_{SCIOM} . We mark these
input-independent segments as: **II₁**, **II₂**, and **II₃**. Such input-independence is not
observed in the Jansen-Rit model, which is influenced by external inputs across the
entire domain of P , even when the system is non-responsive to network interactions (see
Appendix D for more details). This behavior is responsible for the Jansen-Rit model's
unbounded replication of inputs, a point that we will investigate further in later result
sections.

By contrast in SCIOM, segments **II₁** and **II₃** are associated with the saturation of the
system's response to extra-network drive. Given the parameter values in Table 2, this
saturation point occurs at $P \leq 17$ Hz, while the segment **II₂** is associated with the
attenuation of extra-network drive, for $P \leq 2$ Hz. See Appendix D for a more rigorous
analysis of these features and saturation points.

In the SCIOM paradigm, the transition between **Fix_L** and **SP** happens at inputs lower

than or equal to the maximal firing-rate upperbound ($P \leq 2e'_o$), as P drives the system
towards $\mathbf{II}_1$. The system quickly crosses $\mathbf{SP}$, and stability is found within the $\mathbf{LC}$ and
 $\mathbf{Fix}_H$ segments.

Between $\mathbf{II}_2$ and $\mathbf{II}_3$ we find limit-cycle behavior ($\mathbf{LC}$) that includes $P = 0$. Within the
 $\mathbf{LC}$ segment's positive P domain, oscillatory power decreases with P . And $\mathbf{LC}$
segments terminate at $P \leq 2e'_o$, before $\mathbf{II}_3$ at $P = 17$ Hz. This ensures that large inputs
($P(t) > 2e'_o$) can drive the system out of limit-cycle behavior. If the input, P , is larger
than $\text{Dom}(\max(\mathbf{LC})) < \mathbf{II}_3$, then the system will converge to $\mathbf{Fix}_H$. However, for
 $P \geq 17$ Hz, the system enters into the $\mathbf{II}_3$ segment and immediately shoots up to the
steady-state response segment, $Y_{\text{SCIOm}}^{\text{ss}}$, the higher $\mathbf{Fix}_H$ segment. For such inputs
into the Jansen-Rit system, because there is no nonlinearity applied to inputs, no such
saturation occurs.

Energy Model of Underlying Dynamics

Although the bifurcation diagrams presented in Fig. 2 provide a qualitative overview of
the dynamical features most relevant to the presence of intrinsic oscillatory behavior
and its dissolution with external drive, they do not offer any insights into the internal
dynamics fundamental to those features. Therefore, to address the underlying dynamics,
we will introduce two new functions in our analysis: the system's gross excitation
function, $U_{\text{sys}}(Y)$ (Eq. 62), and the excitation-inhibition work function, $\Sigma W(Y)$ (Eq.
63). See Appendix E for the explicit derivation of $U_{\text{sys}}(Y)$ and $\Sigma W(Y)$.

The gross excitation function, $U_{\text{sys}}(Y)$, determines the difference in the amount of
"energy" stored in the excitation and inhibition firing rates of the network. Energy in
this context is conceptually synonymous with the net excitation across the population
under consideration. The population's average firing-rate is therefore a reflection of the
amount of this energy stored by the population. The excitation-inhibition work
function, $\Sigma W(Y)$, determines the amount of energy put into (or taken out of) the
system at any point in the domain, Y . In this section, we will see how the interplay
between $U_{\text{sys}}(Y)$ and $\Sigma W(Y)$ determines the behavior of the system. For instance,
given a particular **excitation landscape**, U_{sys} , the proclivity for a transition to occur

between two modes of behavior will depend on the sum of the excitation-inhibition
work, ΣW , performed by the network near a transition point.

Before examining the standard Jansen-Rit system, and how the parameter modifications
discussed in the previous section influence the system to satisfy the desired dynamics,
we will define several terms that we will use in the ensuing analysis and discussion.

The system is in a **fixed excitation-state** when its output $Y^s = V_E - V_I$ is in an
interval $\mathbf{Y}_D$ such that $\frac{\partial U_{\text{sys}}(Y^s \in \mathbf{Y}_D)}{\partial Y} = 0$. Therefore, fixed-excitation states are
states such that small changes in V_E or V_I do not change the system's net excitation,
formally defined as for an $\epsilon > 0$, the following holds: $\frac{\partial U_{\text{sys}}((V_E \pm \epsilon) - V_I)}{\partial Y} =$
 $\frac{\partial U_{\text{sys}}(V_E - (V_I \pm \epsilon))}{\partial Y} = 0$. Fixed excitation-states occur because changes in the energy
stored in the excitation firing rate of the network are offset by changes in the energy
stored in the inhibition firing rate of the network, and vice-versa. Note that the
segments $\mathbf{Fix}_L$ and $\mathbf{Fix}_H$ are each subsets of a respective interval of fixed
excitation-inhibition states; that is: $\mathbf{Fix}_L \subseteq \mathbf{Y}_{DL}$ and $\mathbf{Fix}_H \subseteq \mathbf{Y}_{DH}$. Furthermore,
fixed excitation-states are graded in terms of stability, and the most stable subset of
fixed excitation-states occur when $U_{\text{sys}}(Y^s) = 0$, corresponding to a minimum in the
system's net excitation.

The system is driven internally by the “work” performed on it by the positive-feedback
loop between the pyramidal and excitatory populations, and the negative-feedback loop
between the pyramidal and inhibitory populations (see Appendix E for a more precise
definition). The net work ΣW performed is therefore the sum of the work performed by
these loops and the external drive. If $\Sigma W \neq 0$, then a perturbation occurs in the system
state, ΔY . More precisely, if the system undergoes positive work then an increase in Y
is generated, associated with an increase in V_E , a decrease in V_I , or both. **Stable**
equilibrium occurs in a subset of fixed excitation-states, Y^{s*} , in which the work done
on the system at Y^{s*} is balanced, $\Sigma W = 0$.

External Drive and Stable Oscillations

In the standard Jansen-Rit model with no external drive ($P = 0$), the excitation-inhibition work on the system becomes increasingly negative immediately to the right of the equilibrium point, $Y_a^* \in \mathbf{Fix}_L$ (Fig. 3A). Consequently, if the system starts at the equilibrium point, Y_a^* , it will return to Y_a^* with small perturbations.

Fig 3. Interplay between the gross excitation function and the excitation-inhibition work function in the Jansen-Rit model.

Excitation-inhibition work analysis over the standard Jansen-Rit model excitation-landscape in (A) and the parameter modified Jansen-Rit's, $Y_{JR\hat{c}}^*$, in (B), where $\hat{c} = \{120, 100, 20, 20\}$. The point Y_a corresponds to the lower fixed point, $\mathbf{Fix}_L$, while the peak at Y_b corresponds to the separatrix region, and Y_c at the, $\Sigma W(Y)$, zero-crossing corresponds to either the high fixed point, $\mathbf{Fix}_H$ or the stable limit-cycle segment, **LC**. The standard Jansen-Rit system, under intrinsic drive, does not exhibit oscillatory behavior because the system's excitation-inhibition work cannot overcome the excitation barrier (A). Therefore, neither $\mathbf{Fix}_H$ or **LC** occur in the standard Jansen-Rit under this condition. On the other hand, the parameter modified system, $Y_{JR\hat{c}}^*$, demonstrates an excitation landscape where sufficient net positive excitation-inhibition work is generated to overcome the energy-barrier, producing a **LC** within the energy-valley about Y_c . In (C) the effects of external drive ($P = 7.5\text{Hz}$) on the excitation-landscape are shown for, $Y_{JR\hat{c}}^*$. The energy-valley disappears and the system converges to $\mathbf{Fix}_H$.

By contrast, the unstable **SP** region is characterized by a peak in U_{sys} , where $\frac{\partial U_{\text{sys}}(Y)}{\partial Y} > 0$, creating an **excitation barrier** at Y_b , as indicated in Fig. 3A-C. To overcome this excitation-barrier, sufficient net positive work is needed to overcome it, $\Sigma W(Y \in \mathbf{SP}) > U_{\text{sys}}(Y \in \mathbf{SP})$, but in the standard Jansen-Rit model, the excitation loop cannot produce sufficient net positive work and the inhibition-loop creates net negative work further preventing Y from increasing. Thus the only way to overcome the barrier is with strong external drive.

By increasing the excitatory coupling weights (C_{EP} and C_{PE}) of the Jansen-Rit model,

it is possible to strengthen the excitation-loop to produce sufficient positive-work on the system to overcome the energy-barrier in the **SP** region without large external drive. While increasing excitatory coupling also increases the excitation barrier (Fig 3B), the parameter regimes of the modified Jansen-Rit (Fig 2C) allows deviation from the fixed state.

Once over the **SP** energy-barrier the system begins to converge toward the equilibrium point $Y_c^* \in \mathbf{Fix}_H$, where ΣW undergoes a zero-crossing at the fixed excitation-state, Y_c^* . Overshoots or displacements, ΔY , across an equilibrium point are hallmarks of higher-order dynamical systems (e.g. the Jansen-Rit system). Likewise, in order two or higher dynamical systems, these overshoots about a stable equilibrium point result in unstable oscillations. The work done on the system by the excitation-inhibition loops fluctuates between net-positive and net-negative work while $\frac{\partial U_{\text{sys}}(Y^s \in \mathbf{Y}_D)}{\partial Y} = 0$, resulting in decaying oscillatory behavior that converges to Y_c^* as $\Delta Y \rightarrow 0$.

Stable oscillations emerge at a ΣW zero-crossing, Y_c , within an excitation environment in which displacements about Y_c in either direction produce $\frac{\partial U_{\text{sys}}(Y^c \in \mathbf{Y}_D)}{\partial Y} > 0$. We refer to this convexity in the excitation-landscape as an **energy-valley**. Displacements, ΔY , over Y_c within this energy-valley such that $\Delta U_{\text{sys}} = U_{\text{sys}}(Y_c + \Delta Y) - U_{\text{sys}}(Y_c - \Delta Y) = 0$, result in stable oscillations and the emergence of the **LC** segment. Ultimately, in the case of stable oscillatory behavior, the fluctuations in positive and negative work on the system are sustained by increases in the system's net excitation, unlike in the case of decaying oscillatory behavior.

The effect of external drive on the excitation landscape, particularly the excitation-valley, is illustrated in Fig 3C. Two things occur simultaneously as external drive is added to the system. The first is the broadening of the excitation-valley, resulting in an increase in the displacement: $\Delta Y' > \Delta Y$ about Y_c , such that $\Delta U_{\text{sys}} = U_{\text{sys}}(Y_c + \Delta Y)' - U_{\text{sys}}(Y_c - \Delta Y') = 0$, within which stable oscillations occur corresponding to an increase in oscillatory power. The second is an increase in Y_c , as indicated by the right-ward movement of Y_c' with the introduction of external drive in Fig 3C. Increases in Y_c bring the system closer to the upper bound of S_F . Eventually, the increase in oscillatory power is capped by the interaction between the oscillations

and the upper bound of S_F . Further increases in external drive continue to push Y_c closer to the upper bound of S_F resulting in the reduction of oscillatory power.

The broadening of the excitation-valley with increasing external drive ultimately results in the elimination of the excitation-valley, and the reemergence of an interval $\mathbf{Y_D}$ such that $\frac{\partial U_{\text{sys}}(Y^c \in \mathbf{Y_D})}{\partial Y} = 0$. Accordingly, the zero-crossing point, Y_c , becomes a stable equilibrium point once again, indicating the reappearance of $\mathbf{Fix_H}$. The system begins to exhibit decaying oscillations toward Y_c^* , as before.

E/I Balance and Stable Oscillations

In the bifurcation analysis we made several observations regarding the occurrence of intrinsic oscillations within a narrow range of excitatory-inhibitory coupling balance. In this section we will take a closer look at how oscillatory power varies over the 4-dimensional coupling coefficient space, $\mathbf{C_W}$, and use the energy approach to describe the geometry of the intrinsic oscillatory region in $\mathbf{C_W}$. In particular, we focus on identifying the constraints on network coupling necessary to produce strong intrinsic oscillations.

Fig 4. Dependence of alpha rhythms on E/I couplings. Normalized alpha-power (8 - 12 Hz) metrics over the inhibitory surface $C_{I\phi}$ (A) given $\hat{c}_E^* = [100, 120]$ and $C_{E\phi}$ (B) given $\hat{c}_I^* = [20, 20]$. We map the coordinates of $\hat{c}_E^*$ used to generate $C_{I\phi}$ in (B), and map the coordinates of $\hat{c}_I^*$ used to generate $C_{E\phi}$ in (A). In the inset of (A) we include simulations of four sampled points in $C_{I\phi}$. In (C) we generate the $C_{E/I}$ map of the SCIOM system at intrinsic drive $P = 0$ with corresponding color map. Likewise in (D) we generate the $C_{E/I}$ map of the SCIOM system at $P = 5$.

We first consider the 2-D cross-section of the full parameter space in which the inhibitory coupling weights vary ($C_{IP} \times C_{PI}$), and focus our attention on the subregion wherein intrinsic oscillatory behavior manifests, $C_{I\phi}$ (Fig. 4A). The geometry of $C_{I\phi}$ is determined by the bounded interaction between C_{IP} and C_{PI} . We will delegate $C_{\Sigma I}$ to denote this interaction:

$$C_{\Sigma I} := C_{IP} S_F(C_{PI} \overline{V_P}) \quad (1)$$

where $\overline{V_P}$ is the average pyramidal membrane potential. Intrinsic oscillations occur
when $C_{\Sigma I}(C_{IP}, C_{PI})$ falls between a lower bound of $C_{\Sigma I}^{\text{FixH}}$ and an upper bound of $C_{\Sigma I}^{\text{FixL}}$:

$$C_{\Sigma I}^{\text{FixH}} \leq C_{\Sigma I}(C_{IP}, C_{PI}) \leq C_{\Sigma I}^{\text{FixL}} \quad (2)$$

The upper $C_{\Sigma I}^{\text{FixL}}$ and lower $C_{\Sigma I}^{\text{FixH}}$ bounds within which oscillations occur were detected
empirically in the bifurcation analysis according to the intersection of the **LC** segment
with the vertical axis in the Y versus P phase-space. However, by fixing a point, $\widehat{c_E^*}$, in
the complementary surface, then the upper bound on the interaction between the two
inhibitory coupling weights, $C_{\Sigma I}^{\text{FixL}}$, for intrinsic oscillatory behavior is determined as:

$$C_{\Sigma I}^{\text{FixL}} = \inf(\{C_{\Sigma I}(C_{IP}, C_{PI}) \mid \Sigma W(Y \in \mathbf{SP}; C_{IP}, C_{PI}) > U_{\text{sys}}(Y \in \mathbf{SP}; C_{IP}, C_{PI})\}) \quad (3)$$

The excitatory-loop's ability to produce sufficient net positive work to overcome the
inhibitory-loop's negative work and the excitation-barrier determines the upper bound
on $C_{\Sigma I}$. If the coupling between the inhibitory and pyramidal populations exceeds
 $C_{\Sigma I}^{\text{FixL}}$, then intrinsic oscillations do not occur and the system will converge to **FixL**.

On the other hand, the lower bound $C_{\Sigma I}^{\text{FixH}}$ on $C_{\Sigma I}$ for intrinsic oscillatory behavior is
determined as:

$$C_{\Sigma I}^{\text{FixH}} = \inf(\{C_{\Sigma I}(C_{IP}, C_{PI}) \mid \frac{\partial U_{\text{sys}}(Y \geq Y_c; C_{IP}, C_{PI})}{\partial Y} \neq 0\}) \quad (4)$$

the smallest $C_{\Sigma I}$ for which the excitation-landscape admits an energy-valley about Y_c .
The coupling between the inhibitory and pyramidal population must match or surpass
 $C_{\Sigma I}^{\text{FixH}}$ to support the presence of an excitation-valley in $U_{\text{sys}}(Y)$ about Y_c . Otherwise,
intrinsic oscillations do not occur and the system will converge to **FixH**.

Alpha (8 to 12 Hz) power over $\mathbf{C}_{I\Phi}$ indicates that increases in $C_{\Sigma I}$ result in increases in
the system's oscillatory power, as shown in Fig. 4A. This observation is explained by
the fact that: $\frac{\partial U_{\text{sys}}(Y \geq Y_c; C_{IP}, C_{PI})}{\partial Y} > 0$ as $\partial C_{\Sigma I} > 0$; the rise in the
excitation-valley results in an increase in the displacement, $\Delta Y' > \Delta Y$, that the system
undergoes about Y_c , such that $\Delta U_{\text{sys}} = U_{\text{sys}}(Y_c + \Delta Y') - U_{\text{sys}}(Y_c - \Delta Y') = 0$.
Increases in $C_{\Sigma I}$ push the system to the left, along the Y coordinate in the energy
landscape, U_{sys} , driving the system away from the upper-bound of S_F . Interactions
with the upper-bound of S_F create the waxing and waning effect that is observed when
increasing external drive or excitatory coupling as the system is pushed toward the
upper-bound of S_F . Consequently, because the system is pushed away from the
upper-bound of S_F as inhibitory coupling is increased, waxing and waning of alpha
power is not observed over $\mathbf{C}_{I\Phi}$. Rather, the system's oscillatory power is strengthened
with continued increases in $C_{\Sigma I}$ over $\mathbf{C}_{I\Phi}$ until the system converges to **FixL** before
interacting with the lower bound of S_F .

Next, we consider the 2-D cross section of the full parameter space in which the
excitatory coupling weights vary ($C_{EP} \times C_{PE}$), with focus on the subregion wherein
oscillatory behavior manifests, $\mathbf{C}_{E\Phi}$ (Fig. 4B). Akin to $\mathbf{C}_{I\Phi}$, the geometry of the
excitatory surface, $\mathbf{C}_{E\Phi}$, is determined by the bounded interaction between C_{EP} and
 C_{PE} . We delegate $C_{\Sigma E}$ to denote the interaction between the two excitatory coupling
weights:

$$C_{\Sigma E} := C_{EP} S_F(C_{PE} \overline{V_P}) \quad (5)$$

Intrinsic oscillations occur when C_{EP} falls between a lower bound of $C_{\Sigma E}^{\text{FixL}}$ and an
upper bound $C_{\Sigma E}^{\text{FixH}}$:

$$C_{\Sigma E}^{\text{FixL}} \leq C_{\Sigma E}(C_{EP}, C_{PE}) \leq C_{\Sigma E}^{\text{FixH}} \quad (6)$$

Fixing a point, $\hat{c}_I^*$, in the inhibitory surface, then the upper bound on the interaction
between the two excitatory coupling weights, $C_{\Sigma E}^{\text{FixL}}$, is determined as:

$$C_{\Sigma E}^{\text{FixH}} = \inf(\{C_{\Sigma E}(C_{EP}, C_{PE}) \mid \frac{\partial U_{\text{sys}}(Y \geq Y_c; C_{EP}, C_{PE})}{\partial Y} \neq 0\}) \quad (7)$$

An excitation-valley is necessary for stable oscillations and increases in $C_{\Sigma E}$ result in
 $U_{\text{sys}}(Y \geq Y_c) \rightarrow 0$, causing the system to converge to **FixH**. Therefore, the annihilation
of the energy-valley with increased coupling strength between the excitatory and
pyramidal populations determines the upper bound, $C_{\Sigma E}^{\text{FixH}}$, on $C_{\Sigma E}$.

On the other hand, the lower bound $C_{\Sigma E}^{\text{FixL}}$ is determined as:

$$C_{\Sigma E}^{\text{FixL}} = \inf(\{C_{\Sigma E}(C_{EP}, C_{PE}) \mid \Sigma W(Y \in \mathbf{SP}; C_{EP}, C_{PE}) > U_{\text{sys}}(Y \in \mathbf{SP}; C_{EP}, C_{PE})\}) \quad (8)$$

The excitatory-loop has to be strong enough to satisfy the excitation-barrier and to
contend against the inhibitory-loop. This requirement imposes a lower bound $C_{\Sigma E}^{\text{FixL}}$ on
 $C_{\Sigma E}$. Otherwise, the system will converge to **FixL**.

Alpha power changes with $C_{E\Phi}$, for low $C_{\Sigma E}$ alpha increases, and for high $C_{\Sigma E}$ alpha
power decreases, as shown in Fig. 4B. The dynamics behind this behavior is similar to
the dynamics underlying the waxing and waning of alpha power that was observed when
increasing external drive. Increases in $C_{\Sigma E}$ result in the broadening of the energy-valley,
which results in an increase in the displacement $\Delta Y' > \Delta Y$ that the system undergoes
about Y_c , such that $\Delta U_{\text{sys}} = U_{\text{sys}}(Y_c + \Delta Y') - U_{\text{sys}}(Y_c - \Delta Y') = 0$. Therefore, the
oscillatory power of the system increase as $C_{\Sigma E}$ is increased. At the same time,
increases in $C_{\Sigma E}$ push the system to the right, along the Y coordinate in the

excitation-landscape, $U_{\text{sys}}(Y)$. Eventually, the increasing oscillatory amplitude begins
interacting with the upper-bound of S_F , imposing a cap on the height of the oscillations,
and with the mean of the oscillations moving closer to the upper-bound of S_F , the
amplitude of the oscillations begins to shrink.

The similar dependence in $\mathbf{C}_{I\Phi}$ on the two inhibitory coupling coefficients C_{PI} and C_{IP}
results in the near symmetry seen in Fig. 4A. This occurs because of the mapping of
 $\overline{V_P}C_{PI}$ onto a near linear regime of S_F , which matches the linear dependence on $\overline{V_P}C_{IP}$.
By contrast the dissimilarity in the dependence of $\mathbf{C}_{E\Phi}$ on the two excitatory coupling
coefficients C_{PE} and C_{EP} results in the asymmetry seen in Fig. 4B. This occurs because
 $\overline{V_P}C_{PE}$, unlike $\overline{V_P}C_{PI}$, maps onto the nonlinear regime of S_F .

The difference between the operating ranges of C_{PE} and C_{PI} arises from $A_i\tau_i \gg A_e\tau_e$,
and, as established earlier, the excitatory interaction must be strong enough to produce
sufficient net positive work to overcome the excitation-barrier in order for the
emergence of oscillatory behavior. Unlike C_{PE} , C_{PI} does not interact with the bounds
of S_F within $\mathbf{C}_{I\Phi}$. This is because $A_i\tau_i \gg A_e\tau_e$ mandates that large changes in C_{PI}
toward either of the bounds of S_F drive the system out of the critical balance for
oscillatory behavior defined earlier.

A representation of $\mathbf{C}_W$ that allows us to evaluate the collective influence of the
coupling coefficients on oscillatory power is attained by considering the ratio between
 $C_{\Sigma I}$ and $C_{\Sigma E}$:

$$C_{E/I} = \frac{C_{\Sigma E}}{C_{\Sigma I}} = \frac{C_{EP}S_F(C_{PE}\overline{V_P})}{C_{IP}S_F(C_{PI}\overline{V_P})} \quad (9)$$

where $\overline{V_P}$ is the average pyramidal membrane potential. The space of $C_{E/I}$, presented
in Fig. 4C, substantiates the observations made in the bifurcation analysis regarding
the occurrence of oscillations in a narrow range of excitatory-inhibitory coupling
balance. The range of excitatory-inhibitory coupling balance associated with oscillatory
behavior is bounded from above by $C_{E/I}^{\text{FixH}}$, and from below by $C_{E/I}^{\text{FixL}}$.

$$C_{E/I}^{\text{FixL}} \leq C_{E/I} \leq C_{E/I}^{\text{FixH}} \quad (10)$$

The upper-bound $C_{E/I}^{\text{FixH}}$ on $C_{E/I}$ for intrinsic oscillatory behavior is determined as:

$$C_{E/I}^{\text{FixH}} = \inf(\{C_{E/I}(C_{\text{EP}}, C_{\text{PE}}, C_{\text{IP}}, C_{\text{PI}}) \mid \neg[\frac{\partial U_{\text{sys}}(Y \geq Y_c; C_{\text{EP}}, C_{\text{PE}}, C_{\text{IP}}, C_{\text{PI}})}{\partial Y} = 0]\}) \quad (11)$$

That is, a coupling balance $C_{E/I} > C_{E/I}^{\text{FixH}}$ results in the system converging to **FixH**;

again, due to the annihilation of the excitation-valley. Conversely, the lower-bound

$C_{E/I}^{\text{FixL}}$ on $C_{E/I}$ for intrinsic oscillatory behavior is determined as:

$$C_{E/I}^{\text{FixL}} = \inf(\{C_{E/I}(C_{\text{EP}}, \dots, C_{\text{PI}}) \mid \Sigma W(Y \in \mathbf{SP}; \dots) > U_{\text{sys}}(Y \in \mathbf{SP}; \dots)\}) \quad (12)$$

Where a coupling balance $C_{E/I} < C_{E/I}^{\text{FixL}}$ results in the system converging to **FixL**;

again, due to the inability of the excitatory-loop to satisfy the excitation-barrier.

Lastly, the effect of external drive on the oscillatory region in Fig. 4C is illustrated in

Fig. 4D. External drive reduces the size of the oscillatory region, further narrowing the

range of excitatory-inhibitory balance that generates oscillations. This is because

increases in external drive cause $U_{\text{sys}}(Y \geq Y_c) \rightarrow 0$, and cause the system to move closer

to the upper-bound of S_F .

Oscillation Frequency

In the Jansen-Rit model's formalism, the ratio between the average population

capacitance, A_η , over the lumped time constant, τ_η , (incorporating the average

membrane time constant, synaptic delays, and other temporal structures that shape the

temporal dynamics) maps rates in Hz to synaptic drive in mV, $\frac{A_\eta}{\tau_\eta}$. Together, the

average population capacitance and the upper-bound on the firing-rate nonlinearity, 413
determine the maximum synaptic drive, V_F , to a population: $\max(V_F) = A_\eta \lim_{X \rightarrow \infty}$ 414
 $S_F(X) = 2e_o A_\eta$. However, the average population capacitance and the lumped time 415
constant are treated as independent quantities in the Jansen-Rit formalism. In the 416
previous section, we saw that oscillations occur in a tight range of excitation-inhibition 417
coupling balance, $C_{E/I}$. We can extend this notion of a balance between excitation and 418
inhibition underlying oscillatory behavior to synaptic drive readily: 419

$$V_{F_{E/I}} = \frac{V_{F_E}}{V_{F_I}} = \frac{\frac{A_e}{\tau_e} C_{EP} S_F(C_{PE} \bar{V}_P)}{\frac{A_i}{\tau_i} C_{IP} S_F(C_{PI} \bar{V}_P)} = \frac{A_e \tau_i}{A_i \tau_e} C_{E/I} \quad (13)$$

Likewise, as in the coupling coefficient discussion, oscillations occur within upper $V_{F_{E/I}}^{\text{FixH}}$ 420
and lower $V_{F_{E/I}}^{\text{FixL}}$ bounds: 421

$$V_{F_{E/I}}^{\text{FixL}} \leq V_{F_{E/I}} \leq V_{F_{E/I}}^{\text{FixH}} \quad (14)$$

where $V_{F_{E/I}}^{\text{FixH}} = \frac{A_e}{\tau_e} C_{E/I}^{\text{FixH}}$ and $V_{F_{E/I}}^{\text{FixL}} = \frac{A_i}{\tau_i} C_{E/I}^{\text{FixL}}$, according to results from the previous 422
section. In the Jansen-Rit's treatment of A_η and τ_η as independent quantities, one can 423
see how this balance can be easily disrupted. Consequently, when attempting to change 424
the resonance structure of the system by changing the time constants, oscillations of 425
different frequencies are not observed. The system either oscillates at 12 Hz or it does 426
not oscillate at all across the majority of the parameter space. This is a critical 427
limitation given the frequency variability of oscillations between different studies, and 428
even different individuals. Likewise, the A_e versus A_i parameter space in Fig. 5A 429
exhibits 10 to 12 Hz in a region that looks similar to the oscillatory region in Fig. 4C, 430
but oscillations of different frequencies do not occur, and the system does not oscillate 431
across the majority of the parameter space. 432

Fig 5. Dependence of oscillation frequency on E and I capacitances. The Jansen-Rit model's frequency map as a function of excitatory and inhibitory capacitances (A). The SCIOM model's frequency map as a function of excitatory and inhibitory capacitances (C). Sample points in the A_e vs. A_i capacitance space at coordinates: (3.25, 22), (3.75, 41), (4.29, 50.4), (5.00, 63.7), (5.89, 78.1), and (6.53, 92), were used in time-course simulations in (B) for the Jansen-Rit map, and in (D) for the SCIOM map.

In contrast, in the SCIOM model, the function $\mathcal{T}(A_\eta)$ relates the average capacitance 433
 A_η to the lumped time constant τ_η of the population; therefore, the two quantities are 434
dependent in this model. Their dependence, as defined in the methods section, is such 435
that an increase in average capacitance results in an increase in the lumped time 436
constant, and vice-versa. These two quantities are related by a conversion factor: $\frac{T_\eta^{-1}}{a_\eta}$ 437
(see methods). Given two different capacitances, $A_e \neq A'_e$, then the following holds true: 438

$$\frac{A_\eta}{\mathcal{T}_\eta(A_\eta)} = \frac{a_\eta}{T_\eta^{-1}} = \frac{A'_\eta}{\mathcal{T}_\eta(A'_\eta)} \quad (15)$$

Therefore, for any choice of the conversion factor, $\frac{T_{\eta}^{-1}}{a_{\eta}} > 0$, in which the balance between excitatory and inhibitory synaptic drive satisfies the bounds for intrinsic oscillatory behavior changes, in either population conductance or lumped time constants, will produce oscillatory behavior of a different frequency because:

$$V_{F_{E/I}}^{\text{Fix}_L} \leq \frac{A_e \mathcal{T}_i(A_i)}{A_i \mathcal{T}_e(A_e)} C_{E/I} = \frac{A_e C_{E/I}}{a_i T_i \mathcal{T}_e(A_e)} = \frac{A_e \mathcal{T}_i(A'_i)}{A'_i \mathcal{T}_e(A_e)} C_{E/I} \leq V_{F_{E/I}}^{\text{Fix}_H} \quad (16)$$

Consequently, by choosing a conversion factor such that the balance between excitation and inhibition drive satisfies Eq. 16 we can produce the frequency map shown in Fig. 5C by varying A_{η} to generate oscillations of different frequencies but of similar spectral widths. Thus modest changes in the SCIOM parameters are sufficient to create different oscillation frequencies.

Response Transients

A final advantage of the SCIOM model is the incorporation of synaptically mediated input drive. Given that inputs from a cortical microcircuit are mediated by synapses, this is of obvious benefit, but it also allows to accurately reproduce the non-linear response dynamics seen in physiological data. By contrast, even a parameter modified Jansen-Rit model that supports intrinsic oscillations (Fig 6B), is limited to linear responses. By contrast the SCIOM model (Fig 2C) responds to input with an appropriate non-linearity. The parameters are set such that input rate functions $P(t) < 7$ Hz are attenuated; hence, the system attenuates input function, $P_1(t)$.

Fig 6. Transmission of input transients. The standard Jansen-Rit model's response to the input functions presented in the inset of (A). The parameter optimized Jansen-Rit model, allowing the transmission of transients, response to the same input functions in the inset of (A) is presented in (B). The SCIOM model's response to the same input functions is presented in (C). Raw LFP data over the V4 of a trained adult monkey performing a visual shape detection task illustrates prominent alpha-rhythms preceding the onset of a noisy stimulus in its visual space, followed by a shape stimulus, followed by V4 response, and a saccade.

The SCIOM model exhibits several modes of response depending on the state of the system and the strength of the input function. First, **subliminal responses** are those that are attenuated by the system. **Intermediary responses** are those that produce a sharp transient that are sometimes followed by a broader, decaying transient. **Suprathreshold** responses are ones in which the system generally produces an initial sharp transient that is followed by a steady-state response, $Y_{\text{SCIOM}}^{\text{ss}}(t)$.

Fig 6C illustrates the SCIOM model's response to the set of five input-rate functions that were used in the preceding Jansen-Rit simulations in Fig 4. In this case, the

model's parameters were set to attenuate input-rate functions $P(t) < 7$. What we see is the model's attenuation of $P_1(t)$, but to $P_2(t)$ it produces an intermediary response. This intermediary response consists of a sharp transient that's followed by a slower decay. To input functions: $P_3(t)$, $P_4(t)$, and $P_5(t)$, the model responds with a suprathreshold response consisting of an initial sharp transient, that is followed by steady-state components. The steady-state component for $P_5(t)$ lasts the longest. These response dynamics are compared to a segment of a raw V4 LFP recording taken from a behaving adult male Macaque, performing a visual task (Fig 6D). A segment of strong alpha oscillations precedes the onset of a visually evoked transient that consists of a sharp transient followed by a steady-state response, comparable to the SCIOM model's suprathreshold response behavior.

Interaction between intrinsic oscillations and transient responses

In this section, we will examine the SCIOM model's steady-state response component as a function of the internal state of the system (its alpha-power), and examine the initial sharp transient component as a function of alpha-phase. From here on, for brevity, we will use the term **fast-component** when referring to the initial sharp transient of the response. Furthermore, we will use the term **oscillatory state** in reference to the power of alpha in the system.

We can solve for the SCIOM's steady-state response function, $Y_{\text{SCIOM}}^{\text{ss}}(t)$, precisely by taking the upper limit of S_F in Eq. 34:

$$Y_{\text{SCIOM}}^{\text{ss}}(t) = 2A_e\tau_e \left(C_X e_f + C_{\text{EP}} S_s [2e_o] + \frac{(C_{\text{EP}} + C_X)}{2} \lambda(t) \right) - A_i\tau_i C_{\text{IP}} S_s [2e_o A_e\tau_e C_{\text{PI}}] \quad (17)$$

By examining Eq. 17, we see that the steady-state behavior, $Y_{\text{SCIOM}}^{\text{ss}}(t)$, is strongly determined by the inhibitory influence. This is due to the fact that $A_i\tau_i \gg A_e\tau_e$.

Fig. 7A illustrates the SCIOM model's responses to a visual input in the presence of 3 phase aligned oscillations of different strengths (weak (Ψ_{W}), moderate (Ψ_{M}), and

strong ($\Psi_{\mathbf{S}}$). The three oscillations were obtained through changing coupling
interactions in the network. The respective steady-state (post transient) responses
 $Y_{\text{SCIOm}}^{\text{ss}; \mathbf{W}}(t)$, $Y_{\text{SCIOm}}^{\text{ss}; \mathbf{M}}(t)$, and $Y_{\text{SCIOm}}^{\text{ss}; \mathbf{S}}(t)$ are anticorrelated with the oscillatory state of the
system: the greater the alpha power of the system, the smaller the amplitude of its
steady-state response, and vice-versa.

To explain these observations, we take another look at the Y vs. P phase space (Fig.
7C). The mean of the steady-state function, $Y_{\text{SCIOm}}^{\text{ss}}$, corresponds to a segment of
equilibrium points in excitation-inhibition, E - I. The trajectory of the system in its
phase space when undergoing a suprathreshold response involves an initial displacement
from excitation-inhibition equilibrium at some $P \geq \mathbf{II}_3$ corresponding to the
fast-component of the response. When the system's phase space trajectory falls back
down to the equilibrium curve, if $P \geq \mathbf{II}_3$ still holds, then the system maintains a
steady-state response, $Y_{\text{SCIOm}}^{\text{ss}}(\Delta t)$ for the duration that $P(\Delta t) \geq \mathbf{II}_3$. Moreover,
 $Y_{\text{SCIOm}}^{\text{ss}}$ is negatively correlated with alpha-power simply because inhibition, as was
established earlier, is positively correlated with alpha-power, and increased inhibition
lowers $Y_{\text{SCIOm}}^{\text{ss}}(t)$.

Fig 7. Alpha phase dependence of transient responses. The SCIOM model's response to a fixed input function at three different oscillatory states: strong, moderate, and weak, with all other parameter conditions equal, induces three different steady-state response amplitudes (A). Steady-state response amplitude is also anti-correlated to the power of alpha oscillations in the system. In (B) the SCIOM model is presented with the same input-function in (A) but at four different phases: 0, $\frac{\pi}{2}$, π , and 2π . The phase effect is reflected in the amplitude of the initial transient that precedes the steady-state response, and is related to the time lag in the inhibitory population's response in curving excitation. The phase-space representation reconciles both the phase-dependent and the amplitude dependent effects (C). The initial transient represents an overshoot across the equilibrium segment Y_{SCIOM}^{ss} , and the height of the overshoot is phase dependent. The system then eventually settles back down to equilibrium in Y_{SCIOM}^{ss} . In (D) we demonstrate the full phase response curve associated with the three oscillatory conditions in (A). The phase-dependency scales with oscillatory power.

The response of the SCIOM model to the fast-component is dependent on the phase of the alpha rhythm, as shown in Fig. 7B. In this figure, we also look at the excitatory and inhibitory time courses, and show that the excitatory population's response to $P_4(t)$ at different phases is virtually identical. By contrast, the inhibitory population displays a phase dependent time lag in generating a response. This results in a phase dependent population response (Fig. 7D), as defined by the max amplitude of the fast-component

at a particular phase over its average maximum amplitude across all phases. The phase response of the system is also dependent on the power of the oscillations. A larger phase effect is seen for the strong oscillatory state than for the moderate and weak, in that order.

Discussion

In order to understand how the prominent low frequency 8 to 12 Hz rhythms in cortex impact information processing, we adapted a classic neural mass model (Jansen-Rit) to model a cortical microcircuit which produces realistic activity dynamics and relies upon physiologically derived parameters. Our motivation was to address two critical issues in the original model: the absence of low frequency oscillations without strong input drive and the inconsistency between input drive and output activity. Both of these inconsistencies were addressed with modest changes in connectivity parameters and the addition of a input non-linearity consistent with the synaptically mediated inputs that cortical neurons receive and that govern connections within the microcircuit. We further demonstrate that these modest changes produce realistic non-linear responses to input transients and that, consistent with experimental observations, the magnitude of those responses depends on the phase of alpha rhythms. By deriving a potential energy approach to understanding oscillatory dynamics, in which stable oscillations occur in an energy valley, we found that in our model, unlike the original Jansen-Rit model, increasing external drive results in the reduction and ultimate disappearance of this potential energy valley, thus allowing strong input transients to be faithfully transmitted. These results are robust to a range of intracortical connectivities, but are critically dependent on the ratio of excitation to inhibition.

The presence of strong oscillations in the absence of strong input is consistent with the intrinsic oscillations reported in cortical slice preparations as well the prominence of alpha oscillations in states of relatively quiescence. Indeed, the first reports of such oscillations by Berger in 1930s [6] noted that such oscillations in visual cortex were most prominent when eyes were closed and largely absent when subjects were visually

engaged. Decades of subsequent study have demonstrated that the oscillations are
associated with a lack of attention and a corresponding drop in visual performance [39,
64, 52, 22].

In our model, oscillations are intrinsic to the circuit [20], and not reliant on an external
oscillatory signal, consistent with multi-site recordings in V4 and FEF [7]. However,
critical features of the cortical microcircuit, including laminar heterogeneities in both
connectivity and alpha rhythms [62], which are thought to originate infragranularly and
synchronize across laminae, are absent in the Jansen-Rit model.

While this circuit has distinct populations of projection pyramidal cells and excitatory
and inhibitory interneurons, it is clearly a simplification of the rich heterogeneity of cell
types and neurotransmitters in the cortex. While structural heterogeneity can strongly
affect network dynamics [37], recent evidence suggests that excitation/inhibitory ratios
in pyramidal cells are conserved at a cellular level [68], which is consistent with the
critical dependence of our model on these ratios. Excitatory/inhibitory imbalance has
been associated with cortical dysfunction both experimentally [16, 19, 33, 26] and
theoretically [9, 42, 70, 3, 49, 48, 69, 58, 12, 54].

The SCION model is robust to modest variations in the effective decay constant of
excitatory and inhibitory postsynaptic potentials. It is also robust to large variations in
inhibitory and excitatory coupling as long as their ratios are maintained. This is
consistent with recent studies in invertebrate model systems suggesting that it is
network behavior [50, 11], rather than the exact mixture of connections and
neurotransmitters, that is strongly conserved across systems and individuals. In our
model, significant excitatory/inhibitory imbalance leads to fixed points in the which the
system is “stuck” and unable to faithfully transform input transients. This is consistent
with the premise that excitatory/inhibitory balance is strongly conserved and necessary
for the accurate processing of incoming information in both sensory and cognitive
contexts.

Our model critically depends on input non-linearity. Non-linear input-output relations,
in addition to being physiologically well motivated given the synaptic nature of inputs
to a cortical microcolumn, have long been associated with critical functioning including

the maintenance of onset transients throughout a heirarchical system and allowing for
stochastic resonance. By contrast, the standard Jansen-Rit system does not transmit
inputs at all as they get attenuated by the ongoing oscillations and even the parameter
modified Jansen-Rit system responds linearly to input transients. This is particularly
problematic when considering the high input case in which, in the absence of any output
thresholds, output increases without limit.

In addition, SCIOM's response to input transients is strongly dependent on the phase of
alpha oscillations [60, 10], consistent with a variety of psychophysical studies involving
visual, auditory, and somatosensory detection [40, 38, 44, 18] and discrimination [25, 55,
5], and physiological studies highlighting the role of ongoing activity in explaining
response variability [34, 29]. The physiological basis for these effects has long been the
subject of modeling [51, 40, 41]. For example, models have typically assumed that the
inverse relationship between alpha waves and cortical excitability is governed by a
strong association between alpha waves and inhibitory neurons [36, 43], and that control
of alpha wave magnitude is governed by thalamocortical inputs [63]. However, a variety
of studies have pointed to an association of top-down feedback inputs [56, 46, 4], which
largely target excitatory cells [24], as critical to the regulation of intrinsically generated
alpha power [8].

SCIOM is consistent with such observations: the interactions between alpha and input
transients are a network phenomena, and are not solely due to a selective action upon
the inhibitory population. Changes in response transients occur because of a difference
of response latencies in the excitatory and inhibitory populations.

SCIOM makes an interesting prediction regarding the effects on alpha upon the response
dynamics to brief input transients: the sustained response that follows the initial
transient is dependent on the oscillatory state of the system, but, unlike the initial
response, not its phase. This is because the oscillatory state of a system is a reflection
of the underlying excitation-inhibition balance, and the amplitude of the steady-state
response is a function of the that balance. While such effects on response dynamics are
certainly consistent with experimental observations pointing to a complexity of response
effects with ongoing alpha rhythms [35, 28], verification of this prediction would depend

on delivery of impulse-like changes in input rates, which is unlikely to be observed, even
in the case of brief external inputs, in the case of sensory transduction mediated
stimulation, and may be require direct phase-specific electrical stimulation.

Because a major motivation of our approach was to impose consistency between input
and output regimes, a natural extension of would be construct 2-D arrays of these
microcircuits to study the spatiotemporal interactions between alpha and stimulation
across the surface of the cortex and thus make predictions about the spatiotemporal
generation and propagation of macroscopic signals that give rise to EEG observations.
Finally, should these studies result in data consistent with existing experimental data,
the model would have considerable predictive value for the design of multi-dimensional
microstimulation paradigms.

Methods

Computational Models

This section begins with a description of the Jansen-Rit model as originally defined,
placing particular emphasis on the two transformations that the model performs. Then,
we will introduce a third transformation that leads us to the SCIOM model whose
dynamics better simulates realistic response transients and alpha rhythms seen in
experimental data. Afterwards, we will order reduce the SCIOM model to arrive at a
single expression that encapsulates the entire equilibria set of the model.

Jansen-Rit

The Jansen-Rit (JR) system models a local cortical population with a set of three,
coupled, nonlinear ordinary differential equations of the second order that describe the
mean dynamics between three interconnected neuronal populations, namely: excitatory
(E), inhibitory (I) interneurons, and pyramidal (P) long range projection neurons.

In essence, the model approximates the dynamics of the aforementioned populations as

an equality between the second order derivative of their average membrane potentials
and their received net synaptic drive. In this process, each population performs two
transformations. The first is a transformation from membrane potential to **firing-rate**,
and the second is a transformation from pre-synaptic action potential rate **input-rate**
to postsynaptic potential.

A block diagram of the connectivity between these populations is shown in 1A. While
the mathematical definition of the model is as follows. Given $t \in [0; \infty)$:

$$\begin{aligned}\ddot{V}_P(t) + \frac{2}{\tau_e}\dot{V}_P(t) + \frac{2}{\tau_e^2}V_P(t) &= \frac{A_e}{\tau_e}S_F(V_E(t) - V_I(t)) \\ \ddot{V}_E(t) + \frac{2}{\tau_e}\dot{V}_E(t) + \frac{1}{\tau_e^2}V_E(t) &= \frac{A_e}{\tau_e}[C_{EP}S_F(C_{PE}V_P(t)) + P(t)] \\ \ddot{V}_I(t) + \frac{2}{\tau_i}\dot{V}_I(t) + \frac{1}{\tau_i^2}V_I(t) &= \frac{A_i}{\tau_i}[C_{IP}S_F(C_{PI}V_P(t))]\end{aligned}\quad (18)$$

The left hand sides of the equations determine the membrane potential dynamics of the
neuronal populations. The right hand sides describes their interactivity with each other
and with external sources, in terms of firing-rates. The average voltage (mV) for each
population is $V_j(t)$ for $j \in \{P, E, I\}$ and its first and second derivatives, $\dot{V}_j$ and $\ddot{V}_j(t)$.
The lumped parameter τ_η for $\eta \in \{e, i\}$ combines both the membrane time constant and
the synaptic delay together, for the excitatory (e) and inhibitory (i) populations,
respectively. A_η scales the net input-rate and determines the maximum synaptic
response to an impulse of input (see, “Transformation 2,” of the Computational Models
section). The ratio $\frac{A_\eta}{\tau_\eta}$ converts the net input-rate to an electric potential, which in the
Jansen-Rit model is unbounded. Lastly, the parameter C_γ is the coupling coefficient
between the different populations where $\gamma \in \{PE, EP, IP, PI\}$, and PE represents the
drive from the pyramidal population to the excitatory population. In the Jansen-Rit
model, these parameters: C_{EP} , C_{PE} , C_{IP} , and C_{PI} are each proportional to a global
coupling parameter C_G . That is $C_\gamma := \rho_\gamma C_G$, where ρ_γ is a scalar between 0 and 1, see
1.

Transformation 1: Membrane Potential to Firing-Rate. The input drive to each
population from the other populations is transformed through a sigmoid function S_F

from a potential to an average firing-rate in Hz. We'll refer to S_F as the **firing-rate nonlinearity**:

$$S_F(V_j(t)) := \frac{2e_o}{1+e^{r(V_o-V_j(t))}} \quad (19)$$

where e_o represents the half maximum firing-rate of the population (Hz), V_o is the potential (mV), that generates the half maximum firing-rate, and r describes the average membrane sensitivity (mV^{-1}). The coefficient $2e_o$ determines the upper-bound of the sigmoid output rate, and r is the slope at the half maximum point, V_o .

The external input to the system is $P(t)$, representing “pulse-density” (Hz), carries both upstream information and noise in the Jansen-Rit formulation. We will separate the input-rate function from intra-network sources, $F_N(t) := S_F(C_\gamma V_j(t))$, and extra-network sources, $F_X(t) := P(t)$. Hence, the net input-rate function to a population is the superposition of the intra-network and extra-network input-rate functions:

$$F_\Sigma(t) := F_N(t) + F_X(t) \quad (20)$$

All the standard values of the Jansen-Rit model's parameters are defined in 1. These parameter values were chosen by the authors of the Jansen-Rit to generate stable alpha-rhythms.

PSP		CC's		S_F	
Param.	Value	Param.	Value	Param.	Value
A_e	3.25 mV	C_{EP}	125	e_o	2.5 Hz
A_i	22 mV	C_{PE}	100	V_o	6 mV
τ_e	10 ms	C_{IP}	20	r	0.56 mV^{-1}
τ_i	10 ms	C_{PI}	20		

Table 1. Standard values of the Jansen-Rit model's parameters

Transformation 2: Input-Rate to Membrane Potential. The model's synaptic impulse response $H_\eta(t)$, (see Appendix A for derivation) is:

$$H_e(t) = \frac{A_e}{\tau_e} t e^{-\frac{t}{\tau_e}} \quad (21)$$

$$H_i(t) = \frac{A_i}{\tau_i} t e^{-\frac{t}{\tau_i}} \quad (22)$$

for the excitatory and inhibitory populations respectively. The excitatory postsynaptic-potential (EPSP) and inhibitory postsynaptic-potential (IPSP) impulse responses are shown in Figure 8.

Fig 8. Impulse response of the Jansen-Rit populations' EPSP (blue) and IPSP (red). The coefficients $A_e = 3.25$ mV and $A_i = 22$ mV scales the max response. The time constants are $\tau_e = 10$ ms and $\tau_i = 20$ ms.

Thus, from functional analysis, the membrane potential of a population j at time t is

the convolution of its impulse response function $H_\eta(t)$ with the net input-rate function $F_\Sigma(t)$ to the population:

$$V_j(t) = H_\eta(t) \otimes F_\Sigma(t) \quad (23)$$

Lastly, the output Y of the model approximates the measured local field potential of the microcircuit as the sum of the excitatory and inhibitory population potentials:

$$Y(t) = V_E(t) - V_I(t) \quad (24)$$

Equilibrium Expression for $Y_{\text{JR}}^*(P)$

An equilibrium equation for the Jansen-Rit model relating critical states $Y^* = V_E^* - V_I^*$ to external input-rate P has been previously presented [23, 2]:

$$Y_{\text{JR}}^*(P) = A_e \tau_e P + A_e \tau_e [C_{EP} S_F(C_{PE} A_e \tau_e S_F(Y_{\text{JR}}^*(P)))] - A_i \tau_i [C_{IP} S_F(C_{PI} A_e \tau_e S_F(Y_{\text{JR}}^*(P)))] \quad (25)$$

Equation (25) defines a relation from P to Y^* . The derivation of this expression involves standard order-reduction of the system (18) and substitution. We will not show these steps for the Jansen-Rit system; however, we will explicitly derive an equilibrium equation for the adapted model in the ensuing sections.

SCIOM (Self-consistent intrinsically oscillating microcircuit) model

The SCIOM model differs from the Jansen-Rit model in 3 aspects (1): its input drive is synaptically mediated, there is a synaptic capacitance, and several coupling parameters are changed.

Synaptically mediated input The first change from the Jansen-Rit model is how

net-input rates $F_{\Sigma}(t)$ are treated and their linear transformation to synaptic drive. 685

Let $V_F(t)$ denote the synaptic drive. In the Jansen-Rit model, the conversion factor $\frac{A_{\eta}}{\tau_{\eta}}$ 686
maps rates in Hz to synaptic drive in mV linearly: 687

$$V_F(t) = \frac{A_{\eta}}{\tau_{\eta}} F_{\Sigma}(t) \quad (26)$$

Furthermore, because the membrane-potential dynamics of population j responds 688
linearly to synaptic drive: 689

$$\ddot{V}_j(t) + \frac{2}{\tau_{\eta}} \dot{V}_j(t) + \frac{1}{\tau_{\eta}^2} V_j(t) = V_F(t) \quad (27)$$

it therefore responds linearly to net input-rates $F_{\Sigma}(t)$ as well. There are three issues 690
with this behavior. First, it allows the populations to respond to physiologically 691
impossible negative input-rates. Second, there are no physiological constraints or 692
bounds on the synaptic drive and extreme events are possible. Although one could 693
avoid these predicaments by constraining $F_{\Sigma}(t)$ within a physiologically plausible bound, 694
the populations will still respond linearly to $F_{\Sigma}(t)$, and the output of the model 695
becomes a scaled version of the input. 696

This **response linearity** is neurophysiologically inconsistent with the nonlinear nature 697
of synaptic mediated dynamics. **Response nonlinearities** are necessary for multiple 698
stable states and the ability to transition to distinct modes of response dynamics. 699
Synaptic nonlinearities can also attenuate subthreshold inputs, such as noise, to enhance 700
relevant features resulting in improved noise rejection and information processing. 701

To address the issues caused by the linear responses in the Jansen-Rit model, we will 702
add a nonlinear transformation function S_s to the synaptic drive, that maps 703
presynaptic firing-rates in Hz to the rate of synaptic events in Hz. We'll refer to S_s as 704
the **synaptic nonlinearity**: 705

$$S_s(P(t)) := \frac{2e_f}{1+e^{s(f_o-P(t))}} \quad (28)$$

here e_f represents half maximum input-rate (Hz), f_o is the input-rate (Hz) that achieves half the maximum synaptic drive, and s is the synaptic membrane sensitivity (Hz⁻¹) to input-rates. The coefficient $2e_f$ determines the upper-bound of S_s , and s corresponds to the slope at the half maximum point, f_o .

The nonlinear transformation S_s on the input-rate functions $F_N(t)$ and $F_X(t)$ leads us to a new model of synaptic drive to population j :

$$V_F(t) = \frac{A_\eta}{\tau_\eta} [S_s(F_N(t)) + S_s(F_X(t))] \quad (29)$$

Consequently, the membrane-potential dynamics of population j , which depends linearly on their synaptic drive $V_F(t)$, as defined in equation (27), depends nonlinearly on input-rates. This change leads us to the “Self-consistent intrinsically oscillating microcircuit” (SCIOM) model. The block diagram of this new model is provided in 1B. The corresponding mathematical definition of the SCIOM model is as follows:

$$\begin{aligned} \ddot{V}_P(t) + \frac{2}{\tau_e} \dot{V}_P(t) + \frac{2}{\tau_e^2} V_P(t) &= \frac{A_e}{\tau_e} S_s[S_F[V_E(t) - V_I(t)]] \\ \ddot{V}_E(t) + \frac{2}{\tau_e} \dot{V}_E(t) + \frac{1}{\tau_e^2} V_E(t) &= \frac{A_e}{\tau_e} (C_{EP} S_s[S_F[C_{PE} V_P(t)]] + C_X S_s[P(t)] + (C_{EP} + C_X) \lambda(t)) \\ \ddot{V}_I(t) + \frac{2}{\tau_i} \dot{V}_I(t) + \frac{1}{\tau_i^2} V_I(t) &= \frac{A_i}{\tau_i} C_{IP} S_s[S_F(C_{PI} V_P(t))] \end{aligned} \quad (30)$$

where $\lambda(t)$ is a Poisson distributed random variable simulating exogenous noise. The parameter C_X weighs the average synaptic drive from external interactions. Observe that increasing either C_{EP} or C_X , the strength of the feed-forward process, increases the system’s noisiness.

Capacitance The next significant change from the Jansen-Rit model concerns the relationship between the lumped time constant τ_η and the synaptic capacitance A_η of a population. The Jansen Rit model treats them as independent quantities: changes in A_η do not result in changes in τ_η . By contrast, the SCIOM model treats these two

quantities as dependent in which the time constant of a neuron should be approximately
proportional to its synaptic capacitance, as a rough estimation:

$$\mathcal{T}_\eta(A_\eta) = \frac{A_\eta}{\alpha_\eta T_\eta} \quad (31)$$

Here $\alpha_\eta T_\eta$ serve as a conversion factor that converts a given synaptic capacitance A_η to
a particular time constant $\mathcal{T}_\eta$. In this paradigm, changes in the average synaptic
capacitance of a population result in a proportional change in its lumped time constant.
This is physiologically realistic because, on average, as the capacitance across a
population of neurons increases the amount of voltage change across the population
increases on average, and the length of time it takes for its restoration to baseline
increases as a result. Consolidating equation (31) with system (30):

$$\begin{aligned} \ddot{V}_P(t) + \frac{2\dot{V}_P(t)}{\mathcal{T}_e(A_e)} + \frac{V_P(t)}{\mathcal{T}_e(A_e)^2} &= \frac{A_e}{\mathcal{T}_e(A_e)} S_S[S_F[V_E(t) - V_I(t)]] \\ \ddot{V}_E(t) + \frac{2\dot{V}_E(t)}{\mathcal{T}_e(A_e)} + \frac{V_E(t)}{\mathcal{T}_e(A_e)^2} &= \frac{A_e}{\mathcal{T}_e(A_e)} (C_{EP} S_S[S_F[C_{PE} V_P(t)]] + C_X S_S[P(t)] + (C_{EP} + C_X) \lambda(t)) \\ \ddot{V}_I(t) + \frac{2\dot{V}_I(t)}{\mathcal{T}_i(A_i)} + \frac{V_I(t)}{\mathcal{T}_i(A_i)^2} &= \frac{A_i}{\mathcal{T}_i(A_i)} C_{IP} S_S[S_F(C_{PI} V_P(t))] \end{aligned} \quad (32)$$

Different oscillations of similar spectral-width but of different frequencies can be
generated through different choices of synaptic capacitance, A_η . Alternatively, with this
formulation, oscillations of different spectral-width but of the same frequency can be
produced by varying the conversion factor $\alpha_\eta T_\eta$, while holding the synaptic capacitance,
 A_η , fixed.

SCIOM parameter values. For convenience, all parameter modifications of the SCIOM
model in this manuscript will be in reference to 2. When presenting a parameter
modified SCIOM model, we will list the parameter changes that are different from the
list of parameters presented in this table.

PSP		CC's		S_F		S_S	
Param.	Value	Param.	Value	Param.	Value	Param.	Value
A_e	3.25 mV	C_{EP}	125	e_o	2.5 Hz	e_o	2.5 Hz
A_i	22 mV	C_{PE}	100	V_o	6 mV	V_o	6 mV
τ_e	10 ms	C_{IP}	20	r	0.56 mV ⁻¹	r	0.56 mV ⁻¹
τ_e	10 ms	C_{PI}	20				
		C_X	40				

Table 2. Values of the SCIOM model's parameters

SCIOM's Equilibria Set

Because of the nonlinearities in the SCIOM model, an analytical solution to Eq. 30 is unattainable. Nevertheless, analysis of the equilibria set can reveal behaviors exhibited by the model. Here we derive a minimal set of expressions that reduces the model's six-dimensional state-space that relates equilibria Y^* to the external constant input-rate function P .

Equilibrium Expression for $Y_{SCIOM}^*(P)$

We begin with the reduced-order representation of the SCIOM model, where the higher-order terms are expressed as first-order expressions of time, t :

$$\begin{aligned}
 \dot{V}_P &= Z_P \\
 \dot{Z}_P &= \frac{A_e}{\tau_e} S_S[S_F[V_E(t) - V_I(t)]] - \frac{2}{\tau_e} Z_P(t) - \frac{1}{\tau_e^2} V_P(t) \\
 \dot{V}_E &= Z_E \\
 \dot{Z}_E &= \frac{A_e}{\tau_e} \left(C_{EP} S_S[S_F[C_{PE} V_P(t)]] + C_X S_S[P(t)] + (C_{EP} + C_X) \lambda(t) \right) - \frac{2}{\tau_e} Z_E(t) - \frac{1}{\tau_e^2} V_E(t) \\
 \dot{V}_I &= Z_I \\
 \dot{Z}_I &= \frac{A_i}{\tau_i} C_{IP} S_S[S_F[C_{PI} V_P(t)]] - \frac{2}{\tau_i} Z_I(t) - \frac{1}{\tau_i^2} V_I(t)
 \end{aligned}
 \tag{33}$$

Equilibrium occurs within a subset (limit-set) of the solution space of (33) when the
differential terms become 0. We will refer to this space as the space of **critical-states**.
We can solve for said solution space, but uncovering the limit-set requires the use of a
bifurcation analysis software (we will use XPP-AUT). Nevertheless, solving for critical
states: V_E^* , V_P^* , and V_I^* , results in the system:

$$\begin{aligned} 0 &= Z_P \\ V_P^* &= A_e \tau_e S_S [S_F [V_E^* - V_I^*]] \\ 0 &= Z_E \\ V_E^* &= A_e \tau_e \left(C_{EP} S_S [S_F [C_{PE} V_P(t)]] + C_X S_S [P(t)] + (C_{EP} + C_X) \lambda(t) \right) \\ 0 &= Z_I \\ V_I^* &= A_i \tau_i C_{IP} S_S [S_F [C_{PI} V_P^*]] \end{aligned} \quad (34)$$

The equilibria set defined by (34) is 6-dimensional, however, three of these dimensions
are trivial: $Z_P \equiv 0$, $Z_E \equiv 0$, and $Z_I \equiv 0$. Thus, proceeding with a 3-dimensional
representation of (34) involving V_P^* , V_E^* , and V_I^* , results in no loss of information. We
therefore arrive at a final expression relating Y^* to external input-rate P , by
substituting V_E^* and V_I^* in the expression $Y = V_E = V_I$:

$$\begin{aligned} Y_{\text{SCIOm}}^* &= V_E^* - V_I^* \\ &= A_e \tau_e \left(C_{EP} S_S [S_F [C_{PE} V_P^*]] + C_X S_S [P] + (C_X + C_{EP}) \lambda(t) \right) - A_i \tau_i C_{IP} S_S [S_F [C_{PI} V_P^*]] \end{aligned} \quad (35)$$

and then substituting $A_e \tau_e S_F(Y_{\text{SCIOm}}^*)$ for V_P^* , results in:

$$\begin{aligned} Y_{\text{SCIOm}}^* &= A_e \tau_e C_X S_S [P] + A_e \tau_e C_{EP} S_S [S_F [C_{PE} A_e \tau_e S_F [Y_{\text{SCIOm}}^*]]] \\ &\quad - A_i \tau_i C_{IP} S_S [S_F [C_{PI} A_e \tau_e S_F [Y_{\text{SCIOm}}^*]]] + A_e \tau_e (C_{EP} + C_X) \lambda(t) \end{aligned} \quad (36)$$

Equation (36) encapsulates the entire equilibria set of the SCIOM model. We are
 primarily interested in studying the family of equilibrium curves of the form $Y_{\text{SCIOM}}^*(P)$
 relating external input-rates P to critical states Y^* .

Bifurcation Analysis of Curves $Y^*(P)$

This section outlines the bifurcation analysis used in this study to compare the family of
 equilibrium curves of the Jansen-Rit model, $\mathbf{G}_{\text{JR}}^{Y(P)}$, and the family of equilibrium curves
 of the SCIOM model, $\mathbf{G}_{\text{SCIOM}}^{Y(P)}$. Analysis of these curves will describe exactly how the
 changes in the SCIOM model result in more physiologically realistic dynamics. These
 curves are acquired by manipulating the four intra-network coupling weights. Therefore,
 it is useful to define the space of intra-network coupling coefficients, $\mathbf{C}_{\mathbf{W}}$:

$$\mathbf{C}_{\mathbf{W}} := \{(C_{\text{EP}}, C_{\text{EP}}, C_{\text{IP}}, C_{\text{PI}}) \mid C_{\text{EP}}, C_{\text{EP}}, C_{\text{IP}}, C_{\text{PI}} \geq 0\} \subseteq \mathbb{R}^4 \quad (37)$$

Now we can define the sets $\mathbf{G}_{\text{JR}}^{Y(P)}$ and $\mathbf{G}_{\text{SCIOM}}^{Y(P)}$ of equilibrium curves at different
 network coupling weights:

$$\mathbf{G}_{\text{JR}}^{Y(P)} = \bigcup_{\hat{c} \in \mathbf{C}_{\mathbf{W}}} \{(P, Y_{\text{JR}}^*(P; \hat{c})) \mid P \in \mathbb{R}\} \subset \mathbb{R}^2 \quad (38)$$

$$\mathbf{G}_{\text{SCIOM}}^{Y(P)} = \bigcup_{\hat{c} \in \mathbf{C}_{\mathbf{W}}} \{(P, Y_{\text{SCIOM}}^*(P; \hat{c})) \mid P \in \mathbb{R}_{\geq 0}\} \subset \mathbb{R}^2 \quad (39)$$

The rest of the parameters for each model are to be held fixed to the values defined in 1
 and 2 for the respective model, unless otherwise specified.

More specifically, however, we are interested in subsets of $\mathbf{G}_{\text{JR}}^{Y(P)}$ and $\mathbf{G}_{\text{SCIOM}}^{Y(P)}$ that

satisfy certain criteria corresponding to dynamical regimes that are consistent with
alpha-rhythm physiology in the visual cortex. We will discuss the criteria of these
subsets and their corresponding dynamics. We will then introduce four equivalence
relations over each equilibrium curve to outline the assortment of limit-set behaviors as
a function of external drive P .

Intrinsic Oscillations

To simulate the occurrence of alpha rhythms during quiescence we sought for a subset of
curves $\Phi_{JR} \subset \mathbf{G}_{JR}^{Y(P)}$ and $\Phi_{SCIOM} \subset \mathbf{G}_{SCIOM}^{Y(P)}$ that possess an initial supercritical Hopf
bifurcation at $P_{H_1} \leq 0$ and a subsequent Hopf bifurcation somewhere between
 $0 \leq P_{H_2} < 2e_o$. We also require for the decay of oscillatory power with increasing
external drive. These three conditions can be written in a more formal manner:

$$\mathbf{C}_1(y): \exists P_{H_1} \leq 0, P_{H_2} \in [0, 2e_o] \text{ s.t. } \lambda_{1,2} \in \Lambda_y(P \in [P_{H_1}, P_{H_2}]), \text{Re}(\lambda_{1,2}) > 0 \ \& \ \text{Im}(\lambda_{1,2}) \neq 0$$

$$\mathbf{C}_2(y): \forall P > P_{H_2} \implies \lambda_{1,2} \in \Lambda_y(P), \text{Re}(\lambda_{1,2}) < 0 \ \& \ \text{Im}(\lambda_{1,2}) \neq 0$$

Where $\Lambda_y(P)$ is the diagonal matrix containing the eigenvalue pairs in the
eigendecomposition of a system y 's Jacobian matrix at the specified point P (see
Appendix C for more details).

Conditions $\mathbf{C}_1$ and $\mathbf{C}_2$ ensure the existence of stable oscillatory behavior in the absence
of external drive. We refer to such oscillations as **intrinsic oscillations**. These two
conditions also ensure the dissolution of stable oscillations within an external drive
range of $[0, 2e_o)$. In addition to these conditions, we require a negative correlation
between oscillatory power and external input P . Therefore, we enforce a third condition:

$$\mathbf{C}_3(y): \forall P_a, P_b; P_{H_1} \leq P_a < P_b \leq P_{H_2} \implies \text{Re}(\lambda_{1,2}^a \in \Lambda_y(P_a)) > \text{Re}(\lambda_{1,2}^b \in \Lambda_y(P_b))$$

The set of equilibrium curves of the form $Y^*(P)$ from the Jansen-Rit and SCIOM
models that exhibit intrinsic oscillations are defined as such:

$$\Phi_{JR} = \{ \mathbf{Y}_y \in \mathbf{G}_{JR}^{Y(P)} \mid \mathbf{C}_1(y) \wedge \mathbf{C}_2(y) \wedge \mathbf{C}_3(y) \} \quad (40)$$

$$\Phi_{SCIO} = \{ \mathbf{Y}_y \in \mathbf{G}_{SCIO}^{Y(P)} \mid \mathbf{C}_1(y) \wedge \mathbf{C}_2(y) \wedge \mathbf{C}_3(y) \} \quad (41)$$

The sets Φ_{JR} and Φ_{SCIO} correspond to parameter regimes that permit intrinsic oscillations for each model and that possess the ability to transmit a response transient.

Partitions on $Y^*(P)$

We partition each curve in $Y_{JR}^*(P) \in \Phi_{JR}$ and $Y_{SCIO}^*(P) \in \Phi_{SCIO}$ into four segments: the high and low fixed point segments Fix_H and Fix_L , a separatrix, and the stable limit-cycle segment defined at its boundaries by an initial supercritical Hopf-bifurcation at one end and a subsequent Hopf-bifurcation at the other end. Examples of these curves with the following partitions indicated can be seen in 2. We'll define these constructs over curves in Φ_{SCIO} , but their implications over curves in Φ_{JR} will be synonymous.

Fix_L: The segment of fixed points $\text{Fix}_L \subseteq \text{Range}(Y_{SCIO}^*)$ are stable states in terms of excitation-inhibition where the eigenvalues of the Jacobian matrix λ_j for each population j is $\text{Re}(\lambda_j) < 0$ and $\text{Im}(\lambda_j) = 0$.

Fix_H: The segment of fixed points $\text{Fix}_H \subseteq \text{Range}(Y_{SCIO}^*)$ are stable states in terms of excitation-inhibition where the eigenvalues of the Jacobian matrix λ_j for each population j is $\text{Re}(\lambda_j) < 0$ and $\text{Im}(\lambda_j) \neq 0$. Although complex, the eigenpairs of the system y each have negative real parts, and so the system will converge to the fixed point.

Limit-cycle Segment (LC): The segment of stable points in the $\text{LC} \subseteq \text{Range}(Y_{SCIO}^*)$

are stable states in terms of excitation-inhibition where the eigenvalues of the Jacobian matrix λ_p and λ_i of the pyramidal and inhibitory populations each have $Re(\lambda) > 0$ and $Im(\lambda) \neq 0$ parts. This results in the manifestation of stable oscillations overlying an unstable fixed point indicated by the positive real part of these eigenvalues.

Separatrix Region (SP): The separatrix is a region in excitation-inhibition that exist between two stable segments. These are transient states that do not belong in the limit-set of the model.

Simulations and Numerical Methods

Numerical Methods. Models were solved numerically using a 4th order Runge-Kutta integration algorithm in MatLAB 2019b (Mathworks. Natick, MA). Equations were integrated at 1kHz. XPP-AUT was used to calculate bifurcation points.

Phase Response Curves. Phase response curves were acquired by simulating an impulse stimulus to the SCIOM model at different phases of the oscillation between 0 and 2π and measuring phase change from the unperturbed case.

Coupling-Coefficient Surfaces. The 4-dimensional space of coupling weights $\mathbf{C}_W$ can be divided into two orthogonal half-spaces of 2-dimensions each: an **excitatory surface** $C_{EP} \times C_{PE} \subset \mathbf{C}_W$, and an **inhibitory surface** $C_{IP} \times C_{PI} \subset \mathbf{C}_W$. Within these half-spaces it is useful then to define regions that give rise to intrinsic oscillations. This region within the excitatory surface is acquired by fixing a point $\hat{c}_I^* \in C_{IP} \times C_{PI}$, then,

$$\mathbf{C}_{E\phi} = \{ \hat{c}_E \in C_{EP} \times C_{PE} \mid Y_{SCIOM}^*(P; \hat{c} = [\hat{c}_E, \hat{c}_I^*]^T) \in \phi_{SCIOM} \} \quad (42)$$

Likewise, a similar region in the inhibitory surface is acquired by fixing a point $\hat{c}_E^* \in C_{EP} \times C_{PE}$, then,

$$\mathbf{C}_{I\phi} = \{ \hat{c}_I \in C_{IP} \times C_{PI} \mid Y_{\text{SCIOIOM}}^*(P; \hat{c} = [\hat{c}_E^*, \hat{c}_I]^T) \in \phi_{\text{SCIOIOM}} \} \quad (43)$$

Depending on the choice of c_η^* , the orthogonal submanifold $C_\eta^\perp$ may be empty. The rest
of the parameters of $Y_{\text{SCIOIOM}}^*(P)$ are held fixed to the values defined in 2, unless
otherwise noted.

Acknowledgements

We thank Audrey Sederberg for her helpful comments. This work was supported by
National Institute of Health grant R01-MH118487.

Appendices

A - Firing-rate to membrane potential operation

Each equation in system (18) serves to transform input-rates on the right-hand side to
field-potentials on the left-hand side through the use of a conversion factor $\frac{A_\eta}{\tau_\eta}$. Where
 A_η determines the maximum synaptic response to a unit impulse function δ , and τ_η
determines its time course. To elucidate the synaptic response to δ we express the
equation in terms of a second-order linear differential operator $\mathcal{L} = (\frac{\partial^2}{\partial t^2} + \frac{\partial}{\partial t} + 1)$:

$$\left(\frac{\partial^2}{\partial t^2} + \frac{2}{\tau_\eta} \frac{\partial}{\partial t} + \frac{1}{\tau_\eta^2} \right) V_j(t) = \frac{A_\eta}{t_\eta} F_\Sigma(t) \quad (44)$$

where $F_\Sigma(t) = F_N(t) + F_X(t)$ is the net input-rate to population j . In the case that
 $F_\Sigma(t) = \delta$ is a dirac-delta function, $V_j(t)$ becomes an impulse response function H_η :

$$\left(\frac{\partial^2}{\partial t^2} + \frac{\partial}{\partial t} + 1 \right) H_\eta(t) = \frac{A_\eta}{t_\eta} \delta \quad (45)$$

or more simply:

$$\mathcal{L}H_{\eta}(t) = \frac{A_{\eta}}{t_{\eta}}\delta \quad (46)$$

It can be inferred from (46) that the kernel of $\mathcal{L}$, the impulse response function $H_{\eta}(t)$ must take the form:

$$H_{\eta}(t) = \frac{A_{\eta}}{\tau_{\eta}}te^{-\frac{t}{\tau_{\eta}}} \quad (47)$$

Therefore, the membrane potential of a population j at time t is the convolution of its impulse response function $H_{\eta}(t)$ with the net input-rate function $F_{\Sigma}(t)$ to the population:

$$V_j(t) = H_{\eta}(t) \otimes F_{\Sigma}(t) = \int_0^t H_{\eta}(t-s)F_{\Sigma}(s) \, ds \quad (48)$$

C - Linearization and Stability

The linearization of either system (JR or SCIOM) principally rests upon finding an approximation to S_F about the point $\hat{X}^* = [V_E^*, V_P^*, V_I^*]^T$. For the SCIOM system we consider the linearization of:

$$\hat{Y}(\hat{X}^*) = [A_e\tau_e S_F(V_E^* - V_I^*), A_e\tau_e [C_{EP}S_F(C_{PE}V_P^*) + C_X S_S(P)], A_i\tau_i [C_{IP}S_F(C_{PI}V_P^*)]]^T \quad (49)$$

Its Jacobian $\mathbf{Dy}_{i,j}(x)$, then takes the form:

$$\mathbf{Dy}_{i,j}(x) = \nabla \hat{Y}(\hat{X}^*)^\top = \begin{bmatrix} \frac{\partial y_1}{\partial x_1} & \frac{\partial y_1}{\partial x_2} & \frac{\partial y_1}{\partial x_3} \\ \frac{\partial y_2}{\partial x_1} & \frac{\partial y_2}{\partial x_2} & \frac{\partial y_2}{\partial x_3} \\ \frac{\partial y_3}{\partial x_1} & \frac{\partial y_3}{\partial x_2} & \frac{\partial y_3}{\partial x_3} \end{bmatrix} \hat{Y}(\hat{X}^*)^\top = \quad (50)$$

For stability and bifurcation analysis the eigendecomposition of its Jacobian matrix is considered at each point $\hat{X}^*$,

$$\mathbf{Dy}_{i,j}(x) = Q\Lambda Q^\top \quad (51)$$

where Q is an orthogonal matrix of size 3 whose columns contain the corresponding eigenvectors of $\mathbf{Dy}_{i,j}(x)$,

$$Q := [\mathbf{q}_E, \mathbf{q}_I, \mathbf{q}_P] \quad (52)$$

where $\mathbf{q}_E$, $\mathbf{q}_I$, $\mathbf{q}_P$ are the eigenvectors corresponding to the excitatory, pyramidal, and inhibitory components accordingly. The diagonal matrix of size n , Λ contains the eigenvalues of the Jacobian:

$$\Lambda := [\lambda_E, \lambda_P, \lambda_I] \quad (53)$$

where λ_E , λ_P , and λ_I are the individual eigenvalues corresponding to the respective populations.

Of-course, because the nonlinearities of the model preclude analytical approaches, we used the numerical analysis software XPPAUT to detect both bifurcation points and stable regimes associated with the emergence and disappearance of oscillations within the domain of these curves.

D - Mapping between output Y and input P

The addition of a second nonlinearity, S_s , in the SCIOM model imposes several segments in which the system evolves independently of external input, driven only by the network interactions. In the Jansen-Rit paradigm the situation was reversed, the system would evolve in several segments independently of network interactions, driven solely by external inputs. However, in the SCIOM, the function S_s distorts the map of P to Y^* such that $P \mapsto Y^*$ is no longer a function, nor smooth at various points of P . We consider the closed-form expression of the map, $P \mapsto Y^*$:

$$P = f_o - \log \left(\frac{\frac{Y^*}{A_e \tau_e} - C_{EP} S_s(S_F(C_{PE} A_e \tau_e S_F(Y^*))) + \frac{A_i \tau_i}{A_e \tau_e} [C_{IP} S_F(C_{PI} A_e \tau_e S_F(Y^*))]}{2e_f} - 1 \right) / s \quad (54)$$

we can write this in a more concise and meaningful manner by defining:

$$M(Y^*) := \frac{Y^*}{A_e \tau_e} - C_{EP} S_F(C_{PE} A_e \tau_e S_F(Y^*)) + \frac{A_i \tau_i}{A_e \tau_e} [C_{IP} S_F(C_{PI} A_e \tau_e S_F(Y^*))] \quad (55)$$

and noticing that $M(Y^*)$ constitutes the synaptic drive to the system that evolves from external interactions. Put in terms of equation (29) for the synaptic drive $V_F(t)$, the variable $M(Y^*) = S_s(F_X(t))$. Now, we can re-write the expression (54) more concisely, using (55):

$$P(Y^*) = f_o - \frac{\log \left(\frac{2e_f}{M(Y^*)} - 1 \right)}{s} \quad (56)$$

Equation (56) implies that the domain of synaptic drive, $M(Y)$, influenced by external inputs is restricted between the bounds: $0 < M(Y^*) < 2e_f$, because $P(Y)$ is undefined at $\lim_{M(Y) \rightarrow 0} \frac{2e_f}{M(Y)} - 1 = -\infty$ and as $\lim_{M(Y) \rightarrow 2e_f} \frac{2e_f}{M(Y)} - 1 = \infty$. Synaptic drive outside

of this dynamic range for external influences manifests from intranetwork interactions and from the relative internal state of the populations. Thus, in the SCIOM model, the system does change and responds with a signal Y in the absence of external influences at various points in its dynamics. Graphically, these points where Y changes independently of P fall within the vertical segments: $\mathbf{II}_1$, $\mathbf{II}_2$, and $\mathbf{II}_3$, illustrated in 2D.

On the contrary, the Jansen-Rit model is invariably influenced by external interactions. Even in domains where the network interactions are essentially shutoff or when the network is fully saturated. Notably, there are two half-lines that radiate away in a linear manner, mostly clearly see in 2A, from the dynamic core of the model. Here $\frac{\partial S_F(Y(P))}{\partial P} = 0$, but the system continues to evolve as a function of P and with $\left| \frac{\partial Y^*(P)}{\partial P} \right| = A_e \tau_e P$.

The saturation point of P is not obvious when examining equation (55). In other words, it is not clear when increasing external input, P , begins to have negligible effect on synaptic drive. Since we run simulations at time steps of 0.001 seconds, we'll consider a change of 0.001 in synaptic drive, $M(Y)$, to be a least change. That is to say, $\left| \frac{\partial M(Y^*)}{\partial P(Y^*)} \right| \leq 10^{-3}$ is consider negligible. Accordingly, $\partial M(P(Y^*) \approx 10^3) \approx 8.982$, therefore, $\lfloor P(M(Y^*) \approx 8.982) \rfloor = 17$ Hz is the upper limit of external-input P in terms of generating a meaningful synaptic drive in the SCIOM model.

E - Excitatory/Inhibitory influence on state transitions: an energy and work analysis of network dynamics

From the equilibrium equation (25) of the Jansen-Rit model and (36) of the SCIOM model, it is apparent that the respective equilibria sets of the two models principally rest upon the weighted interactivity of the populations across the three firing-rate transformation functions in the system, S_f . To study these interactions in more depth we define:

$$L_E(Y) := A_e \tau_e C_{EP} S_F(C_{PE} A_e \tau_e S_F(Y)) \quad (57)$$

$$L_I(Y) := A_i \tau_i C_{IP} S_F(C_{PI} A_e \tau_e S_F(Y)) \quad (58)$$

$$Y^* := L_E - L_I + F_X = \Delta L + F_X \quad (59)$$

The function L_E is the drive from the pyramidal population onto the excitatory population, and L_I is the drive from the pyramidal population to the the inhibitory population. 923
924
925

To analyze these equations it is tempting to simplify them by replacing the sigmoid nonlinearity with a piecewise linear function or by reducing the dimensionality of the system through singular perturbation methods. However, it has been observed by Grimbert and Faugeras that these approximations destroy the oscillatory behavior. Therefore it appears that the oscillations emerging from the interactions between the excitation and inhibition loops is determined by higher order dynamics: 926
927
928
929
930
931

$$F_E(Y) := A_e \tau_e \frac{\partial^2 L_E(Y)}{\partial Y^2} = A_e^2 \tau_e^2 C_{EP} \frac{\partial^2}{\partial Y^2} S_F(C_{PE} A_e \tau_e S_F(Y)) \quad (60)$$

$$F_I(Y) := A_i \tau_i \frac{\partial^2 L_I(Y)}{\partial Y^2} = A_i^2 \tau_i^2 C_{IP} \frac{\partial^2}{\partial Y^2} S_F(C_{PI} A_e \tau_e S_F(Y)) \quad (61)$$

where $\frac{\partial^2 L_\eta(Y)}{\partial^2 Y}$ is weighted by the relevant post-synaptic potential amplitude and time constant, $A_\eta \tau_\eta$. The function F_E is the rate of change of the excitatory drive and F_I measures rate of change of the inhibitory drive with respect to the model output, Y . 932
933
934

To better provide an intuitive analogy in understanding these equations, we employ an “energy function” to measure the stability across different output states of the system, Y : 935
936

$$U_{\text{sys}}(Y) = \int [\Delta L_N(Y) + F_X(P)] dY \int Y^* dY \approx \frac{1}{2} Y^{*2} + Y_o^* \quad (62)$$

We refer to $U_{\text{sys}}(Y)$ as the potential-energy of the system at output Y . The constant of integration Y_o^* is used to offset the vertical shift in the graph of $Y(P)$. Therefore, we take $Y_o^* = -\min\{U(Y)\}$, and sets our minimum value of $U_{\text{sys}}(Y) = 0$.

Along with the potential-energy function $U_{\text{sys}}(Y)$, we define work done by the network using (60) and (61):

$$W_{\eta}(Y) = \int F_{\eta}(Y)dY \approx F_{\eta}(Y)\Delta Y = \Delta U(Y) \quad (63)$$

where we denote W_{η} as the work done the loops within the network L_{η} . More specifically, we are interested in the sum of the work done on the system by the excitation and inhibition loop interactions; thus,

$$W_{\Delta}(Y) := W_E(Y) - W_I(Y) = \Delta U(Y) \quad (64)$$

where $W_{\Delta}(Y)$ is the difference between the work done on the system by the excitatory and inhibitory loop interactions which is equal to the change in the potential energy function of the system.

The amount of energy that is inserted into the system by the network interactions at output Y is calculated as:

$$U_N(Y) = \int W_{\Delta}(Y)dY \quad (65)$$

References

- [1] E D Adrian and B H C Matthews. “The berger rhythm: Potential changes from the occipital lobes in man”. In: *Brain* (1934). ISSN: 00068950. DOI: 10.1093/brain/57.4.355.

- [2] Saeed Ahmadizadeh et al. “Bifurcation analysis of two coupled Jansen-Rit neural mass models.” In: *PloS one* 13.3 (2018). Ed. by Gennady Cymbalyuk, e0192842. ISSN: 1932-6203. DOI: 10.1371/journal.pone.0192842. URL: <https://dx.plos.org/10.1371/journal.pone.0192842>
<http://www.ncbi.nlm.nih.gov/pubmed/29584728>
<http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC5870965>.
- [3] Alan Anticevic and John D Murray. “Rebalancing Altered Computations: Considering the Role of Neural Excitation and Inhibition Balance Across the Psychiatric Spectrum.” In: *Biological psychiatry* 81.10 (2017), pp. 816–817. ISSN: 1873-2402. DOI: 10.1016/j.biopsych.2017.03.019. URL: <http://www.ncbi.nlm.nih.gov/pubmed/28434614>.
- [4] K M Armstrong and T Moore. “{R}apid enhancement of visual cortical response discriminability by microstimulation of the frontal eye field”. In: *Proc Natl Acad Sci U S A* 104.22 (2007), pp. 9499–9504. DOI: 10.1073/pnas.0701104104.
- [5] Thomas J Baumgarten, Alfons Schnitzler, and Joachim Lange. “Prestimulus Alpha Power Influences Tactile Temporal Perceptual Discrimination and Confidence in Decisions”. In: *Cerebral Cortex* 26.3 (Mar. 2016), pp. 891–903. ISSN: 14602199. DOI: 10.1093/cercor/bhu247. URL: <http://www.ncbi.nlm.nih.gov/pubmed/25331603>.
- [6] H Berger. “Uber das Elektroenkephalogramm des Menschen. Arch. Pyschiatr. Nervenkr”. In: *Arch. Pyschiatr. Nervenkr* (1929).
- [7] A Bollimunta et al. “Neuronal Mechanisms and Attentional Modulation of Corticothalamic Alpha Oscillations”. In: *Journal of Neuroscience* 31.13 (2011), pp. 4935–4943. ISSN: 0270-6474. DOI: 10.1523/JNEUROSCI.5580-10.2011. URL: <http://www.jneurosci.org/cgi/doi/10.1523/JNEUROSCI.5580-10.2011>.
- [8] Anil Bollimunta et al. “Neuronal mechanisms of cortical alpha oscillations in awake-behaving macaques.” In: *The Journal of neuroscience : the official journal of the Society for Neuroscience* 28.40 (2008), pp. 9976–9988. ISSN: 1529-2401. DOI: 10.1523/JNEUROSCI.2699-08.2008. URL: <http://www.ncbi.nlm.nih.gov/pubmed/18829955>
<http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC2692971>.

- [9] L J Borg-Graham, C Monier, and Y Frégnac. “Visual input evokes transient and strong shunting inhibition in visual cortical neurons.” In: *Nature* 393.6683 (May 1998), pp. 369–73. ISSN: 0028-0836. DOI: 10.1038/30735. URL: <http://www.ncbi.nlm.nih.gov/pubmed/9620800>.
- [10] Michael E Brandt and Ben H Jansen. “The relationship between prestimulus alpha amplitude and visual evoked potential amplitude”. In: *International Journal of Neuroscience* 61.3-4 (1991), pp. 261–268. ISSN: 00207454. DOI: 10.3109/00207459108990744. URL: <https://pubmed.ncbi.nlm.nih.gov/1824388/>.
- [11] Nicolas Brunel and Xiao-Jing Wang. “What determines the frequency of fast network oscillations with irregular neural discharges? I. Synaptic dynamics and excitation-inhibition balance.” In: *Journal of neurophysiology* 90.1 (July 2003), pp. 415–30. ISSN: 0022-3077. DOI: 10.1152/jn.01095.2002. URL: <http://www.ncbi.nlm.nih.gov/pubmed/12611969>.
- [12] Daniel A Butts et al. “Temporal precision in the visual pathway through the interplay of excitation and stimulus-driven suppression.” In: *The Journal of neuroscience : the official journal of the Society for Neuroscience* 31.31 (Aug. 2011), pp. 11313–27. ISSN: 1529-2401. DOI: 10.1523/JNEUROSCI.0434-11.2011. URL: <http://www.ncbi.nlm.nih.gov/pubmed/21813691><http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC3197857>.
- [13] György Buzsáki, Costas A Anastassiou, and Christof Koch. “The origin of extracellular fields and currents—EEG, ECoG, LFP and spikes.” In: *Nature reviews. Neuroscience* 13.6 (2012), pp. 407–420. ISSN: 1471-0048. DOI: 10.1038/nrn3241. URL: www.nature.com/reviews/neuro<http://www.ncbi.nlm.nih.gov/pubmed/22595786><http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC4907333>.
- [14] Áine Byrne, Daniele Avitabile, and Stephen Coombes. “Next-generation neural field model: The evolution of synchrony within patterns and waves”. In: *Physical Review E* 99.1 (2019). ISSN: 24700053. DOI: 10.1103/PhysRevE.99.012313.
- [15] M H Chase and R M Harper. “Somatomotor and visceromotor correlates of operantly conditioned 12-14 c/sec sensorimotor cortical activity”. In:

- Electroencephalography and Clinical Neurophysiology* (1971). ISSN: 00134694. DOI: 10.1016/0013-4694(71)90292-6.
- [16] Vardhan S Dani et al. “Reduced cortical activity due to a shift in the balance between excitation and inhibition in a mouse model of Rett Syndrome”. In: *Proceedings of the National Academy of Sciences of the United States of America* 102.35 (2005), pp. 12560–12565. ISSN: 00278424. DOI: 10.1073/pnas.0506071102.
- [17] Olivier David, Lee Harrison, and Karl J Friston. “Modelling event-related responses in the brain”. In: *NeuroImage* (2005). ISSN: 10538119. DOI: 10.1016/j.neuroimage.2004.12.030.
- [18] Tolgay Ergenoglu et al. “Alpha rhythm of the EEG modulates visual detection performance in humans”. In: *Cognitive Brain Research* (2004). ISSN: 09266410. DOI: 10.1016/j.cogbrainres.2004.03.009.
- [19] André A Fenton. “Excitation-inhibition discoordination in rodent models of mental disorders.” In: *Biological psychiatry* 77.12 (2015), pp. 1079–1088. ISSN: 1873-2402. DOI: 10.1016/j.biopsych.2015.03.013. URL: <http://www.ncbi.nlm.nih.gov/pubmed/25895430>
<http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC4444398>.
- [20] Alexander C Flint and Barry W Connors. “Two types of network oscillations in neocortex mediated by distinct glutamate receptor subtypes and neuronal populations”. In: *Journal of Neurophysiology* 75.2 (1996), pp. 951–957. ISSN: 0022-3077. DOI: 10.1152/jn.1996.75.2.951. URL: <https://www.physiology.org/doi/10.1152/jn.1996.75.2.951>.
- [21] David C Godlove et al. “Microcircuitry of agranular frontal cortex: Testing the generality of the canonical cortical microcircuit”. In: *Journal of Neuroscience* (2014). ISSN: 15292401. DOI: 10.1523/JNEUROSCI.5127-13.2014.
- [22] I C Gould, M F Rushworth, and A C Nobre. “Indexing the graded allocation of visuospatial attention using anticipatory alpha oscillations”. In: *J Neurophysiol* 105.3 (2011), pp. 1318–1326. DOI: 10.1152/jn.00653.2010.

- [23] François Grimbert and Olivier Faugeras. “Analysis of Jansen’s model of a single cortical column”. In: *Current Biology* 14.1 (2006), p. 34. ISSN: 09609822. DOI: 10.1016/j.cub.2003.12.020. URL: <https://hal.inria.fr/inria-00070410>.
- [24] Saskia Haegens et al. “ α -Oscillations in the monkey sensorimotor network influence discrimination performance by rhythmical inhibition of neuronal spiking.” In: *Proceedings of the National Academy of Sciences of the United States of America* 108.48 (Nov. 2011), pp. 19377–82. ISSN: 1091-6490. DOI: 10.1073/pnas.1117190108. URL: <http://www.ncbi.nlm.nih.gov/pubmed/22084106> <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC3228466>.
- [25] Saskia Haegens et al. “Laminar Profile and Physiology of the α Rhythm in Primary Visual, Auditory, and Somatosensory Regions of Neocortex.” In: *The Journal of neuroscience : the official journal of the Society for Neuroscience* 35.42 (Oct. 2015), pp. 14341–52. ISSN: 1529-2401. DOI: 10.1523/JNEUROSCI.0600-15.2015. URL: <http://www.jneurosci.org/content/35/42/14341.full.pdf> <http://www.ncbi.nlm.nih.gov/pubmed/26490871> <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC4683691>.
- [26] B Haider et al. “Neocortical network activity in vivo is generated through a dynamic balance of excitation and inhibition”. In: *J Neurosci* 26.17 (2006), pp. 4535–4545. DOI: 10.1523/JNEUROSCI.5297-05.2006.
- [27] C C Hilgetag et al. “Anatomical connectivity defines the organization of clusters of cortical areas in the macaque monkey and the cat.” In: *Philosophical transactions of the Royal Society of London. Series B, Biological sciences* 355.1393 (Jan. 2000), pp. 91–110. ISSN: 0962-8436. DOI: 10.1098/rstb.2000.0551. URL: <http://www.ncbi.nlm.nih.gov/pubmed/10703046> <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC1692723>.
- [28] Luca Iemi et al. “Spontaneous Neural Oscillations Bias Perception by Modulating Baseline Excitability.” In: *The Journal of neuroscience : the official journal of the Society for Neuroscience* 37.4 (2017), pp. 807–819. ISSN: 1529-2401. DOI: 10.1523/JNEUROSCI.1432-16.2016. URL:

- <http://www.jneurosci.org/lookup/doi/10.1523/JNEUROSCI.1432-16.2017><http://www.ncbi.nlm.nih.gov/pubmed/28123017><http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC6597018>.
- [29] Ben H Jansen and Michael E Brandt. “The effect of the phase of prestimulus alpha activity on the averaged visual evoked response”. In: *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section* 80.4 (1991), pp. 241–250. ISSN: 01685597. DOI: 10.1016/0168-5597(91)90107-9. URL: <https://linkinghub.elsevier.com/retrieve/pii/0168559791901079>.
- [30] Ben H Jansen and Vincent G Rit. “Electroencephalogram and visual evoked potential generation in a mathematical model of coupled cortical columns”. In: *Biological Cybernetics* 73.4 (1995), pp. 357–366. ISSN: 0340-1200. DOI: 10.1007/BF00199471. URL: <http://link.springer.com/10.1007/BF00199471>.
- [31] Ben H Jansen, George Zouridakis, and Michael E Brandt. “A neurophysiologically-based mathematical model of flash visual evoked potentials”. In: *Biological Cybernetics* 68.3 (1993), pp. 275–283. ISSN: 03401200. DOI: 10.1007/BF00224863. URL: <https://pubmed.ncbi.nlm.nih.gov/8452897/>.
- [32] Young Jin Jung, Kyung Hwan Kim, and Chang Hwan Im. *Mathematical issues in the inference of causal interactions among multichannel neural signals*. 2012. DOI: 10.1155/2012/472036.
- [33] Colin Kehr. “Altered excitatory-inhibitory balance in the NMDA-hypofunction model of schizophrenia”. In: *Frontiers in Molecular Neuroscience* 1 (2008), p. 6. ISSN: 1662-5099. DOI: 10.3389/neuro.02.006.2008.
- [34] R C Kelly et al. “Local field potentials indicate network state and account for neuronal response variability”. In: *J Comput Neurosci* 29.3 (2010), pp. 567–579. DOI: 10.1007/s10827-009-0208-9.
- [35] Wolfgang Klimesch, Robert Fellinger, and Roman Freunberger. “Alpha oscillations and early stages of visual encoding”. In: *Front Psychol* 2.MAY (2011), p. 118. ISSN: 1664-1078. DOI: 10.3389/fpsyg.2011.00118. URL: <http://www.ncbi.nlm.nih.gov/pubmed/21687470><http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC3108577><http://journal.frontiersin.org/article/10.3389/fpsyg.2011.00118/abstract>.

- [36] Wolfgang Klimesch, Paul Sauseng, and Simon Hanslmayr. *EEG alpha oscillations: The inhibition-timing hypothesis*. 2007. DOI: 10.1016/j.brainresrev.2006.06.003.
- [37] Itamar D Landau et al. “The Impact of Structural Heterogeneity on Excitation-Inhibition Balance in Cortical Networks”. In: *Neuron* 92.5 (2016), pp. 1106–1121. ISSN: 10974199. DOI: 10.1016/j.neuron.2016.10.027.
- [38] Katharina Limbach and Paul M Corballis. “Prestimulus alpha power influences response criterion in a detection task.” In: *Psychophysiology* 53.8 (2016), pp. 1154–64. ISSN: 1540-5958. DOI: 10.1111/psyp.12666. URL: <http://www.ncbi.nlm.nih.gov/pubmed/27144476>.
- [39] K Linkenkaer-Hansen et al. “Prestimulus oscillations enhance psychophysical performance in humans”. In: *J Neurosci* 24.45 (2004), pp. 10186–10190. DOI: 10.1523/JNEUROSCI.2584-04.2004.
- [40] M Lundqvist, P Herman, and A Lansner. “Effect of Prestimulus Alpha Power, Phase, and Synchronization on Stimulus Detection Rates in a Biophysical Attractor Network Model”. In: *Journal of Neuroscience* 33.29 (2013), pp. 11817–11824. ISSN: 0270-6474. DOI: 10.1523/JNEUROSCI.5155-12.2013. URL: <http://www.ncbi.nlm.nih.gov/pubmed/23864671><http://www.ncbi.nlm.nih.gov/pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC3722510><http://www.jneurosci.org/cgi/doi/10.1523/JNEUROSCI.5155-12.2013>.
- [41] Daniel Malagarriga, Antonio J Pons, and Alessandro E P Villa. “Complex temporal patterns processing by a neural mass model of a cortical column.” In: *Cognitive neurodynamics* 13.4 (2019), pp. 379–392. ISSN: 1871-4080. DOI: 10.1007/s11571-019-09531-2. URL: <http://www.ncbi.nlm.nih.gov/pubmed/31354883>.
- [42] Daniel Malagarriga et al. “Mesoscopic Segregation of Excitation and Inhibition in a Brain Network Model”. In: *PLoS Computational Biology* (2015). ISSN: 15537358. DOI: 10.1371/journal.pcbi.1004007.
- [43] K E Mathewson et al. “Pulsed out of awareness: EEG alpha oscillations represent a pulsed-inhibition of ongoing cortical processing”. In: *Front Psychol* 2 (2011), p. 99. DOI: 10.3389/fpsyg.2011.00099.

- [44] K E Mathewson et al. “To see or not to see: prestimulus alpha phase predicts visual awareness”. In: *J Neurosci* 29.9 (2009), pp. 2725–2732. ISSN: 1529-2401. DOI: 10.1523/JNEUROSCI.3963-08.2009. URL: <http://www.ncbi.nlm.nih.gov/pubmed/19261866>.
- [45] Carrie J McAdams and John H R Maunsell. “Effects of attention on orientation-tuning functions of single neurons in macaque cortical area V4”. In: *Journal of Neuroscience* (1999). ISSN: 02706474. DOI: 10.1523/jneurosci.19-01-00431.1999.
- [46] T Moore and M Fallah. “Microstimulation of the frontal eye field and its effects on covert spatial attention”. In: *J Neurophysiol* 91.1 (2004), pp. 152–162. DOI: 10.1152/jn.00741.2002.
- [47] Young MP et al. “Non-metric multidimensional scaling in the analysis of neuroanatomical connection data and the organization of the primate cortical visual system”. In: *Philosophical transactions of the Royal Society of London. Series B, Biological sciences* 348.1325 (1995), pp. 281–308. ISSN: 0962-8436. DOI: 10.1098/RSTB.1995.0069. URL: <https://pubmed.ncbi.nlm.nih.gov/8577827/>.
- [48] B K Murphy and K D Miller. “Balanced amplification: a new mechanism of selective amplification of neural activity patterns”. In: *Neuron* 61.4 (2009), pp. 635–648. DOI: 10.1016/j.neuron.2009.02.005.
- [49] John D Murray et al. “Linking Microcircuit Dysfunction to Cognitive Impairment: Effects of Disinhibition Associated with Schizophrenia in a Cortical Working Memory Model”. In: *Cerebral Cortex* 24.4 (2014), pp. 859–872. ISSN: 1047-3211. DOI: 10.1093/cercor/bhs370. URL: <https://academic.oup.com/cercor/article-lookup/doi/10.1093/cercor/bhs370>.
- [50] Astrid A Prinz, Dirk Bucher, and Eve Marder. “Similar network activity from disparate circuit parameters.” In: *Nature neuroscience* 7.12 (Dec. 2004), pp. 1345–52. ISSN: 1097-6256. DOI: 10.1038/nn1352. URL: <http://www.ncbi.nlm.nih.gov/pubmed/15558066>.

- [51] Rajasimhan Rajagovindan and Mingzhou Ding. “From prestimulus alpha oscillation to visual-evoked response: an inverted-U function and its attentional modulation.” In: *Journal of cognitive neuroscience* 23.6 (2011), pp. 1379–1394. ISSN: 0898-929X. DOI: 10.1162/jocn.2010.21478.
- [52] Gustavo Rohenkohl and Anna C Nobre. “ α oscillations related to anticipatory attention follow temporal expectations.” In: *The Journal of neuroscience : the official journal of the Society for Neuroscience* 31.40 (Oct. 2011), pp. 14076–84. ISSN: 1529-2401. DOI: 10.1523/JNEUROSCI.3387-11.2011. URL: <http://www.ncbi.nlm.nih.gov/pubmed/21976492><http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC4235253>.
- [53] A van Rotterdam et al. “A model of the spatial-temporal characteristics of the alpha rhythm”. In: *Bulletin of Mathematical Biology* 44.2 (1982), pp. 283–305. ISSN: 0092-8240. DOI: 10.1007/BF02463252. URL: <http://link.springer.com/10.1007/BF02463252>.
- [54] J L R Rubenstein and M M Merzenich. *Model of autism: Increased ratio of excitation/inhibition in key neural systems*. 2003. DOI: 10.1034/j.1601-183X.2003.00037.x.
- [55] Jason Samaha, Luca Iemi, and Bradley R Postle. “Prestimulus alpha-band power biases visual discrimination confidence, but not accuracy”. In: *Consciousness and Cognition* 54 (2017), pp. 47–55. ISSN: 10902376. DOI: 10.1016/j.concog.2017.02.005.
- [56] Jason Samaha et al. “Top-down control of the phase of alpha-band oscillations as a mechanism for temporal prediction.” In: *Proceedings of the National Academy of Sciences of the United States of America* 112.27 (2015), pp. 8439–8444. ISSN: 1091-6490. DOI: 10.1073/pnas.1503686112. URL: <http://www.ncbi.nlm.nih.gov/pubmed/26100913><http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC4500260>.
- [57] T O Sharpee, K D Miller, and M P Stryker. “On the importance of static nonlinearity in estimating spatiotemporal neural filters with natural stimuli.” In: *J Neurophysiol* 99.5 (2008), pp. 2496–2509. URL:

- <http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?cmd=prlinks&dbfrom=pubmed&retmode=ref&id=18353910>.
- [58] W L Shew et al. “Information capacity and transmission are maximized in balanced cortical networks with neuronal avalanches”. In: *J Neurosci* 31.1 (2011), pp. 55–63. DOI: 10.1523/JNEUROSCI.4637-10.2011.
- [59] F H da Silva et al. “Model of brain rhythmic activity - The alpha-rhythm of the thalamus”. In: *Kybernetik* 15.1 (1974), pp. 27–37. ISSN: 03401200. DOI: 10.1007/BF00270757.
- [60] R M Smeal, G B Ermentrout, and J A White. “Phase-response curves and synchronized neural networks”. In: *Philos Trans R Soc Lond B Biol Sci* 365.1551 (2010), pp. 2407–2422. DOI: 10.1098/rstb.2009.0292.
- [61] Roberto C Sotero et al. “Realistically coupled neural mass models can generate EEG rhythms”. In: *Neural Computation* (2007). ISSN: 1530888X. DOI: 10.1162/neco.2007.19.2.478.
- [62] Eelke Spaak et al. “Layer-specific entrainment of γ -band neural activity by the α rhythm in monkey visual cortex.” In: *Current biology : CB* 22.24 (Dec. 2012), pp. 2313–8. ISSN: 1879-0445. DOI: 10.1016/j.cub.2012.10.020. URL: <http://www.ncbi.nlm.nih.gov/pubmed/23159599><http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC3528834>.
- [63] P Suffczynski et al. “Computational model of thalamo-cortical networks: dynamical control of alpha rhythms in relation to focal attention”. In: *Int J Psychophysiol* 43.1 (2001), pp. 25–40. DOI: 10.1016/S0167-8760(01)00177-5.
- [64] G Thut et al. “Alpha-band electroencephalographic activity over occipital cortex indexes visuospatial attention bias and predicts visual target detection”. In: *J Neurosci* 26.37 (2006), pp. 9494–9502. DOI: 10.1523/JNEUROSCI.0875-06.2006.
- [65] Gina G. Turrigiano et al. *Rapid and Active Stabilization of Visual Cortical Firing Rates Across Light-Dark Transitions*. 2019. DOI: 10.1101/542670.
- [66] Fabrice Wendling et al. “Epileptic fast activity can be explained by a model of impaired GABAergic dendritic inhibition”. In: *European Journal of Neuroscience*

- 15.9 (2002), pp. 1499–1508. ISSN: 0953816X. DOI: 1225
10.1046/j.1460-9568.2002.01985.x. 1226
- [67] Hugh R Wilson and Jack D Cowan. “Excitatory and inhibitory interactions in 1227
localized populations of model neurons.” In: *Biophysical journal* 12.1 (1972), 1228
pp. 1–24. ISSN: 0006-3495. DOI: 10.1016/S0006-3495(72)86068-5. URL: 1229
<http://www.ncbi.nlm.nih.gov/pubmed/4332108> 1230
<http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC1484078>. 1231
- [68] Mingshan Xue, Bassam V Atallah, and Massimo Scanziani. “Equalizing 1232
excitation-inhibition ratios across visual cortical neurons”. In: *Nature* 511.7511 1233
(2014), pp. 596–600. ISSN: 14764687. DOI: 10.1038/nature13321. 1234
- [69] Ofer Yizhar et al. “Neocortical excitation/inhibition balance in information 1235
processing and social dysfunction”. In: *Nature* 477.7363 (2011), pp. 171–178. ISSN: 1236
00280836. DOI: 10.1038/nature10360. URL: 1237
<https://www.nature.com/articles/nature10360>. 1238
- [70] Edward Zagher, John D Murray, and David A McCormick. “Simulating Cortical 1239
Feedback Modulation as Changes in Excitation and Inhibition in a Cortical 1240
Circuit Model”. In: *eneuro* 3.4 (2016), pp. 208–216. ISSN: 2373-2822. DOI: 1241
10.1523/ENEURO.0208-16.2016. URL: 1242
<http://eneuro.org/lookup/doi/10.1523/ENEURO.0208-16.2016>. 1243