

Rhythmic Gating of Hebbian Plasticity in a Modified Wilson–Cowan Model as a Mechanism for Stability–Plasticity Balance

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1 Rhythmic Gating of Hebbian Plasticity in a
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3 for Stability–Plasticity Balance

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11 **Abstract**

12 The Wilson-Cowan model is a set of two differential equations simulating the
13 dynamics of interacting populations of excitatory and inhibitory neurons. In its
14 original state, the model is applicable to epilepsy, however, this study seeks to (1)
15 provide a template of the model capable of depicting brain function in learning,
16 and effectively, memory; and (2) identify factors that are important for aiding
17 learning processes in individuals diagnosed with learning disorders. A modified
18 Wilson-Cowan model is introduced where synaptic weights evolve through Hebbian
19 plasticity rules, incorporating weight decay to prevent runaway dynamics
20 and a rhythmic modulation term to simulate gamma oscillations. Stability analysis
21 of the unforced system reveals a single, asymptotically stable fixed point.
22 When oscillatory modulation is applied, this fixed point evolves into a stable periodic
23 orbit, demonstrating that plasticity is gated to specific phases of the gamma
24 cycle, creating rhythmic "learning windows." This phase-dependent learning provides
25 a mechanistic framework to the stability-plasticity dilemma by structuring
26 synaptic modification in time, preventing chaotic growth while allowing continuous
27 learning. The findings suggest that disruptions to this rhythmic organization
28 may underlie learning deficits, and propose that interventions aimed at entraining

brain oscillations could enhance learning by restoring optimal timing for synaptic plasticity.

Keywords: Hebbian plasticity, Gamma oscillations, Synaptic stability, Wilson-Cowan Model

1 Introduction

Neurotypically, newly learned information is processed through the hippocampal complex in a process termed memory consolidation, which also plays a key role in memory recall [Cipolotti and Bird \(2006\)](#); [Ryan, Nadel, and Keil \(2001\)](#). It has commonly been postulated that memories are firstly stored through synaptic changes in the hippocampal cortex [Meeter and Murre \(2005\)](#). More specifically, changes in neural excitatory and inhibitory firing patterns facilitate the underlying processes of memory and learning. Mathematical models suggest that these processes show an initial dependence on the hippocampal complex that eventually diminishes with time [Meeter and Talamini \(2002\)](#). Several of these models suggest that the repeated reactivation of a hippocampal-neocortical representation drives the systems level consolidation process [Ellenbogen, Payne, and Stickgold \(2006\)](#); [Stickgold \(2005\)](#); [Stickgold, James, and Hobson \(2000\)](#). More recently, models simulating the consolidation process using neural networks demonstrate that such models can explain several of the main characteristics of amnesia and semantic dementia [Meeter and Murre \(2004, 2005\)](#).

Learning in the brain is primarily undermined by activity dependent changes in synaptic strength, a process generally referred to as synaptic plasticity. One of the most influential theories is Hebbian learning, which proposes that coactivation of neurons strengthens their synaptic connection [Hebb \(1949\)](#). This principle demonstrates how correlated activity leads to the reinforcement of synaptic connections, providing a cellular mechanism for associative learning. Modern interpretations expand on this by emphasizing that Hebbian plasticity is input specific and involves complex biochemical

55 activity that is often triggered by postsynaptic calcium influx, primarily through acti-
 56 vation of the NMDA receptor [Malenka and Bear \(2004\)](#). This calcium signaling acts
 57 as a coincidence detector, allowing synapses to potentiate only when presynaptic glu-
 58 tamate release coincides with postsynaptic depolarization. A fundamental challenge in
 59 neuroscience is the stability-plasticity dilemma: the brain must remain stable enough
 60 to retain established memories and skills, while simultaneously being plastic enough
 61 to encode new information. Pure Hebbian learning, however, if left uncontrolled, can
 62 lead to runaway excitation, which biological systems counterbalance through home-
 63 ostatic mechanisms like synaptic scaling and inhibitory feedback [Turrigiano \(2012\)](#).
 64 This limitation has prompted the development of hybrid models that combine Hebbian
 65 plasticity with homeostatic and inhibitory regulation to ensure stable learning in
 66 biological and artificial systems [Fox and Stryker \(2017\)](#).

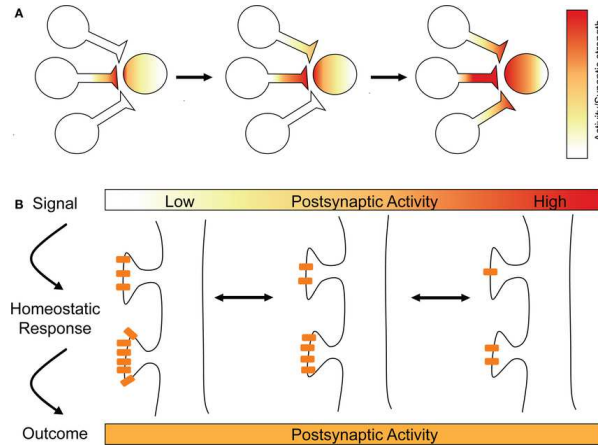


Fig. 1: Runaway Plasticity and Synaptic Scaling [Watt and Desai \(2010\)](#)

67 As shown in Part A of Figure 1, Hebbian plasticity can lead to instability, where
 68 strengthening of one synapse (through Long Term Potentiation, or LTP, shown on the
 69 left) increases the likelihood that it will further drive the postsynaptic neuron to fire.
 70 This elevated postsynaptic activity (middle) in turn makes it easier for other synapses

71 to also undergo LTP (right), potentially resulting in runaway potentiation and reduc-
72 ing the statistical likelihood of a neuron effectively storing information [Watt and Desai](#)
73 [\(2010\)](#). In Part B, synaptic scaling provides a homeostatic mechanism that responds
74 to long term changes in postsynaptic activity. When postsynaptic activity drifts out-
75 side its optimal range, synaptic scaling adjusts all synapses proportionally (illustrated
76 here by changes in AMPA receptor content) to restore balance and maintain stable
77 activity levels.

78 Of several mathematical models for brain function, one of the most popular is
79 the Wilson-Cowan model, which describes the interactions between excitatory and
80 inhibitory neural populations using coupled non-linear differential equations [Wil-](#)
81 [son and Cowan \(1972\)](#). Commonly used to simulate neural populations, the model
82 includes the macroscopic behavior of cortical tissue, including oscillatory states, multi-
83 stability, and transitions between attractor states, all vital elements to understanding
84 the underlying processes of memory encoding, retrieval, and how these processes dif-
85 fer in individuals with learning disabilities [Deco, Jirsa, and McIntosh \(2011\)](#); [Freeman](#)
86 [\(1975\)](#). While Hebbian learning describes the microscopic synaptic changes underlying
87 memory and learning, population level models like the Wilson-Cowan model provide
88 a macroscopic view, enabling simulations of dynamic, large scale neural populations.

89 To connect the microscopic and macroscopic levels of brain function, this paper
90 proposes a hybrid modeling framework that integrates Hebbian learning principles
91 with a modified form of the Wilson-Cowan model. By combining these perspectives,
92 it becomes possible to study how localized changes in synaptic strength influence
93 broader patterns of neural activity, and how population level dynamics can, in turn,
94 affect synaptic structure. This interaction is crucial to understanding how repeated
95 experiences lead to long term changes in brain function and how disruptions in this
96 cycle may contribute to cognitive deficits.

97 The interaction between excitatory and inhibitory activity is a vital factor in both
 98 learning and memory. When this balance is disturbed, it could lead to neurological
 99 disorders. A growing body of research suggests that learning disabilities, including
 100 attention-deficit/hyperactivity disorder (ADHD), dyslexia, and some forms of autism
 101 spectrum disorder (ASD), may be associated with disruptions in the balance between
 102 excitatory and inhibitory (E/I) neural activity [Uhlhaas and Singer \(2010\)](#); [Yizhar et al.](#)
 103 [\(2011\)](#). In ADHD, studies have observed reduced inhibitory control in prefrontal cor-
 104 tical regions, which may impair executive function and working memory, both critical
 105 for effective learning [Castellanos and Proal \(2012\)](#). Dyslexia has been associated with
 106 atypical neural oscillations and altered E/I dynamics in language processing regions,
 107 which may contribute to difficulties in phonological decoding and reading acquisi-
 108 tion [Lehongre, Ramus, Villiermet, Schwartz, and Giraud \(2011\)](#). Similarly, in ASD,
 109 imbalances in E/I signaling, particularly in cortical microcircuits, are suggested to
 110 affect information processing and cognitive flexibility, consequently impacting learn-
 111 ing in both social and academic contexts [Rubenstein and Merzenich \(2003\)](#). While
 112 the present model does not incorporate disorder-specific mechanisms or parameters,
 113 these conditions motivate a broader investigation of how alterations in E/I dynam-
 114 ics and oscillatory structure could, in principle, influence learning-related plasticity.
 115 The model proposed in this paper serves as a theoretical framework for generating
 116 hypotheses rather than as a direct model of any specific population.

117 2 Methods

118 2.1 Model Framework and Rationale

119 This study employs a computational modeling approach to investigate dynamic inter-
 120 actions between neural population activity and synaptic plasticity in learning. The
 121 model in this study builds off the Wilson-Cowan model, which describes mean fir-
 122 ing rates of coupled excitatory (E) and inhibitory (I) neuronal populations [Wilson](#)

and Cowan (1972). The proposed model extends this framework by integrating activity dependent synaptic plasticity, where connection strengths between populations become dynamic variables evolving according to Hebbian correlation principles. This modification enables simulation of how learning at the synaptic level both influences and is influenced by macroscopic network dynamics.

The core dynamical system is defined by a set of six coupled, first-order, non-linear ordinary differential equations. Two equations govern fast-time-scale (neural) dynamics of population firing rates, and four equations govern slow-time-scale (synaptic) dynamics of connection weights. An external oscillatory forcing term is added to the synaptic equations to simulate the influence of gamma-band rhythms known to modulate plasticity Buzsáki and Wang (2012); Fries (2015).

2.2 Stability Analysis Procedures

Stability properties of the autonomous system ($\gamma = 0$) were analyzed through the following systematic procedure:

1. **Fixed Point Identification:** The equilibrium point $(E, I, w_{EE}, w_{EI}, w_{IE}, w_{II})$ was found by numerically solving the system of equations obtained by setting all time derivatives to zero. This was implemented using SciPy's fsolve function with an initial guess near the expected biological regime.
2. **Jacobian Computation:** The 6×6 Jacobian matrix $\mathbf{J}$ of the system was derived analytically. Entries representing partial derivatives of each equation with respect to each state variable were evaluated at the numerically identified fixed point.
3. **Eigenvalue Analysis:** Local stability of the fixed point was determined by calculating eigenvalues λ_i of $\mathbf{J}$ using NumPy's linalg.eig function. A fixed point is asymptotically stable if and only if $\Re(\lambda_i) < 0$ for all i .
4. **Forced System Dynamics:** The non-autonomous system ($\gamma = 0.02$) was analyzed through numerical integration. Emergence of a stable periodic orbit was confirmed

149 by observing sustained, synchronous oscillations in all state variables at the driving
 150 frequency ($\omega = 251.33$ rad/s ≈ 40 Hz) and by plotting phase portraits and Poincare
 151 sections.

152 2.3 Activation Function

153 Nonlinear activation functions $S_E(x)$ and $S_I(x)$, which map total synaptic input to
 154 firing rate, were defined as identical sigmoidal functions:

$$S(x) = \frac{1}{1 + \exp(-a(x - \theta))} \quad (1)$$

155 with gain $a = 1.5$ and threshold $\theta = -1.8$. These parameters were analytically
 156 derived by requiring the function to be consistent with fixed point firing rates (E^* ,
 157 I^*) identified for chosen external inputs (P_{e0} , P_{i0}), thus simulating model dynamics
 158 to a biologically plausible operating point.

159 3 Models and Populations

160 3.1 Wilson-Cowan Model

161 The Wilson-Cowan model simulates excitatory and inhibitory neural functioning using
 162 the following two coupled differential equations:

$$\tau_E \frac{dE}{dt} = -E + (1 - r_E E) \cdot S_E(w_{EE}E - w_{EI}I + P_E) \quad (2)$$

$$\tau_I \frac{dI}{dt} = -I + (1 - r_I I) \cdot S_I(w_{IE}E - w_{II}I + P_I) \quad (3)$$

163 Where:

Table 1: Summary of variables and parameters in the Wilson–Cowan model

Symbol	Description
$E(t)$	Mean firing rate of the excitatory neuronal population at time t
$I(t)$	Mean firing rate of the inhibitory neuronal population at time t
r_E, r_I	Refractory coefficients limiting population activity at high firing rates
$w_{EE}(t)$	Excitatory-to-excitatory synaptic weight (self-excitation)
$w_{EI}(t)$	Inhibitory-to-excitatory synaptic weight (inhibitory feedback)
$w_{IE}(t)$	Excitatory-to-inhibitory synaptic weight (feedforward excitation)
$w_{II}(t)$	Inhibitory-to-inhibitory synaptic weight (self-inhibition)
$P_E(t), P_I(t)$	External inputs to excitatory and inhibitory populations
$S_E(\cdot), S_I(\cdot)$	Nonlinear activation functions mapping synaptic input to firing rate
τ_E, τ_I	Time constants governing response speed of each population

164 The Wilson-Cowan model works by simulating how groups of excitatory and
 165 inhibitory neurons influence each other’s average firing rates over time. Instead of
 166 simulating individual neurons, the model treats each population as a whole, using dif-
 167 ferential equations to track how their activity levels evolve. The excitatory population
 168 (E) tends to increase its own activity (via self-excitation) and can also drive activity in
 169 the inhibitory population (I). In contrast, the inhibitory population suppresses activ-
 170 ity in both itself, known as self-inhibition, and in the excitatory population. These
 171 interactions are quantified by the weights w_{EE} , w_{EI} , w_{IE} , and w_{II} , which represent
 172 the strength of the connections between populations (See Figure 2).

173 The dynamics of each population depends on a leaky integration mechanism: neu-
 174 ral activity decays over time unless driven by input. The terms $-E$ and $-I$ in the
 175 equations represent this natural decay. The remaining terms in each equation reflect
 176 input-driven activation, where the total synaptic input to a population is passed
 177 through a nonlinear activation function, typically a sigmoidal function, to determine
 178 how strongly the population responds. This nonlinearity ensures that the firing rate
 179 grows slowly at low input levels, increases rapidly around a threshold, and then satu-
 180 rates, reflecting realistic neuronal behavior. The model also includes refractory factors,
 181 $(1 - r_E E)$ and $(1 - r_I I)$, which reduce the responsiveness of neurons as their activ-
 182 ity increases, capturing the biological limitation that neurons cannot fire continuously

183 without a refractory period. External inputs (P_E , P_I) allow for the simulation of the
 184 effects of sensory stimuli or background brain activity.

The following diagram (Figure 2) shows the connectivity of neurons in a homogeneous network. The total population that is modeled comprises several billion of these networks, all of which are interlinked under the assumption that each neuron is coupled with at least one other neuron in the population.

Within this model, fixed points represent steady states of population activity. The stability of these points depends on the connection weights (w_{EE} , w_{EI} , w_{IE} , w_{II}), the refractory parameters, and the level of external input. Small changes in these parameters can shift the system from a stable equilibrium to oscillatory dynamics or even bistability, where the network can switch between two distinct states of activity.

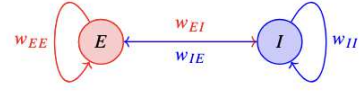


Fig. 2: Assumed Neuronal Connections

185 3.2 Modified Model

186 Although the original Wilson–Cowan model describes the dynamics of average firing
 187 rates $E(t)$ and $I(t)$ using fixed synaptic connection strengths, the modified model
 188 introduces time-dependent synaptic weights, $w_{EE}(t)$, $w_{EI}(t)$, $w_{IE}(t)$, and $w_{II}(t)$,
 189 that evolve according to Hebbian plasticity rules. These weight dynamics include

190 Hebbian terms that are proportional to presynaptic and postsynaptic activity, along
 191 with quadratic decay terms scaled by α to prevent runaway growth. In addition, a
 192 modulation term $\gamma \sin(\omega t)$ represents oscillatory input capable of inducing rhythmic
 193 fluctuations such as theta- and gamma-band activity. The activation functions $S_E(\cdot)$
 194 and $S_I(\cdot)$ remain nonlinear mappings from input to firing rate, as in the original model.
 195 The modified model is comprised of the following six coupled differential equations:

$$\frac{dE}{dt} = -E + S_E(w_{EE}(t)E - w_{EI}(t)I + P_E(t)) \quad (4)$$

$$\frac{dI}{dt} = -I + S_I(w_{IE}(t)E - w_{II}(t)I + P_I(t)) \quad (5)$$

$$\frac{dw_{EE}}{dt} = \eta (E^2 - \alpha w_{EE}^2(t)) + \gamma \sin(\omega t) \quad (6)$$

$$\frac{dw_{EI}}{dt} = \eta (EI - \alpha w_{EI}^2(t)) + \gamma \sin(\omega t) \quad (7)$$

$$\frac{dw_{IE}}{dt} = \eta (IE - \alpha w_{IE}^2(t)) + \gamma \sin(\omega t) \quad (8)$$

$$\frac{dw_{II}}{dt} = \eta (I^2 - \alpha w_{II}^2(t)) + \gamma \sin(\omega t) \quad (9)$$

196 Where, in addition to the variables from the original model, the following
 197 parameters are defined:

Table 2: Summary of parameters in the modified model

Symbol	Description
η	Learning rate (plasticity strength)
α	Weight decay factor preventing runaway growth
γ	Amplitude of sinusoidal modulation
ω	Frequency of sinusoidal modulation

198 For analytical tractability, identical plasticity rules and oscillatory modulation are
199 applied to all four synaptic weights. This symmetric formulation serves as a mini-
200 mal modeling assumption that isolates the interaction between rhythmic forcing and
201 population dynamics rather than implying biological equivalence among synapse types.

202 The synaptic weights evolve according to Hebbian correlations, bounded by
203 quadratic decay, while the oscillatory drive introduces biologically relevant rhythms
204 that can structure plasticity over time. By allowing neural activity and synaptic con-
205 nectivity to co-evolve, the model captures dynamical behaviors absent in fixed-weight
206 systems, including adaptive stabilization, rhythm-dependent learning, entrainment,
207 and resonance phenomena.

208 In biological systems, oscillations often influence plasticity indirectly through fir-
209 ing rates, intracellular signaling, or neuromodulatory processes. Here, the oscillatory
210 drive is applied directly to the weight dynamics as a proof-of-concept simplification
211 that isolates the effect of rhythmic forcing on network-level plasticity while preserving
212 analytical tractability.

213 The quadratic decay term αw^2 acts as a simple homeostatic mechanism that
214 constrains synaptic growth and stabilizes network dynamics. Although more biologi-
215 cally detailed normalization mechanisms exist, this formulation provides qualitatively
216 similar stabilizing behavior with reduced mathematical complexity, allowing the
217 stability–plasticity interaction to be demonstrated explicitly.

218 4 Stability Analyses

219 4.1 Assumptions

220 Because this study employs a modified Wilson–Cowan framework, it adopts several
221 standard population-level assumptions. Neurons within each population are assumed
222 to lie in close spatial proximity, to connect randomly, and to form a sufficiently dense
223 network such that each cell receives at least one connection from within the population.

224 These assumptions permit the neglect of spatial structure and allow the system to be
225 treated as spatially homogeneous. Time is also modeled as a continuous variable.

226 For analytical tractability, synaptic weights in the model are not explicitly con-
227 strained to remain non-negative. This permits the system to be treated as a smooth
228 dynamical flow in continuous state space and facilitates linear stability analysis of the
229 full six-dimensional system. Although biological synapses are strictly non-negative in
230 strength, the present study focuses on qualitative network dynamics rather than lit-
231 eral physiological interpretation of individual weight values. Negative weights therefore
232 should not be interpreted as inhibitory synapses, but rather as mathematical artifacts
233 that allow demonstration of the system’s stability and oscillatory behavior.

234 The plasticity equations governing the four synaptic weights are structurally
235 identical, implying symmetry among excitatory–excitatory, excitatory–inhibitory,
236 inhibitory–excitatory, and inhibitory–inhibitory learning dynamics. This symmetry is
237 imposed as a simplifying assumption to reduce the effective degrees of freedom and
238 isolate the fundamental interaction between oscillatory forcing and network plasticity.
239 While biological systems exhibit heterogeneous plasticity mechanisms across synapse
240 types, the symmetric formulation enables clearer analytical interpretation of stability
241 properties and avoids additional parameter dependencies that could obscure the core
242 dynamical behavior under investigation.

243 A further simplifying assumption in the equilibrium derivation is symmetry
244 between certain synaptic weights. In particular, the equilibrium solutions for w_{EI}^* and
245 w_{IE}^* both yield

$$w_{EI}^* = w_{IE}^* = \sqrt{\frac{E^* I^*}{\alpha}}.$$

246 This equality simplifies the analytical solution but may not fully capture the distinct
247 functional roles of excitatory-to-inhibitory versus inhibitory-to-excitatory connections
248 observed in biological networks.

249 External inputs to the excitatory and inhibitory populations, $P_E(t)$ and $P_I(t)$,
 250 were held constant at baseline values ($P_{E_0} = -2.31$, $P_{I_0} = -3.81$) to represent steady-
 251 state conditions. The equilibrium and its local stability were first determined for the
 252 unforced autonomous system by setting the oscillatory modulation amplitude to zero
 253 ($\gamma = 0$).

254 The nonlinear activation functions $S_E(x)$ and $S_I(x)$ were assumed to be identical
 255 logistic sigmoid functions of the form

$$S(x) = (1 + \exp[-a(x - \theta)])^{-1},$$

256 consistent with the original Wilson–Cowan formulation. The parameters $a = 1.5$ and
 257 $\theta = -1.8$ were selected to ensure consistency with the numerically identified biolog-
 258 ically plausible fixed point of the unforced system ($E^* = 0.1263$, $I^* = 0.0874$). This
 259 was achieved by substituting the equilibrium firing rate and its corresponding net
 260 input into the logistic equation and solving for parameters satisfying

$$S_E(w_{EE}^* E^* - w_{EI}^* I^* + P_{E_0}) = E^*.$$

261 The resulting function,

$$S(x) = (1 + \exp[-1.5(x + 1.8)])^{-1},$$

262 reproduces the fixed-point firing rates.

263 For the linear stability analysis, the derivative of the activation function was
 264 required for construction of the Jacobian matrix. Differentiation of the logistic function
 265 yields

$$S'(x) = a S(x)(1 - S(x)).$$

266 Thus, S'_E and S'_I were computed directly from this analytical expression using the
267 fixed-point values and their corresponding net inputs, rather than treated as free
268 parameters.

269 The stability analysis is local in nature, relying on the Jacobian linearized about
270 the fixed point, and is therefore strictly valid only for infinitesimal perturbations. All
271 remaining parameter values were chosen to be biologically plausible and representative
272 of a stable neural regime. When oscillatory modulation was introduced ($\gamma = 0.02$),
273 the system was treated as a deterministic, non-autonomous dynamical system, and
274 the resulting periodic orbit was examined via numerical integration.

275 It is important to note that the present model is not fitted to empirical data
276 from any clinical population and does not include disorder-specific parameters. Con-
277 sequently, references to learning disorders throughout this work are intended as
278 conceptual motivation rather than as direct model validation or explanation of specific
279 clinical conditions.

280 4.2 Parameters Applied

281 The following values were substituted in the model to perform stability analyses.

Table 3: Parameter Values

Parameter	Description	Reference
w_{EE}	0.9	Nesse, Clark, and Noudoost (2024)
w_{EI}	2.0	Nesse et al. (2024)
w_{IE}	1.0	Nesse et al. (2024)
w_{II}	1.9	Nesse et al. (2024)
P_{E_0}	-2.31	Nesse et al. (2024)
P_{I_0}	-3.81	Nesse et al. (2024)
η	0.05	Kyriazos and Poga (2025)
α	0.01	Dohare et al. (2024)
γ	0.02	Nesse et al. (2024)
ω	251.33	Buzsáki and Wang (2012); Fries (2015)

The parameter values listed in Table 3 were selected to place the system in a biologically plausible dynamical regime and to illustrate the stability–plasticity interactions of interest. They are not directly derived from experimental measurements of synaptic weights or neural time constants in any specific population. Rather, these values were chosen to ensure the system exhibits stable fixed points and phase-dependent learning windows, allowing exploration of the model’s mechanistic properties.

4.3 Analytical Framework

To understand the dynamic behavior of the modified Wilson-Cowan model, a stability analysis examined both the unforced system ($\gamma = 0$) and the rhythmically modulated system ($\gamma = 0.02$). The analysis followed a systematic approach involving fixed point computation, linearization, Jacobian eigenvalue analysis, and numerical simulation of the full non-autonomous system.

295 For the unforced system, first, the equilibrium points were identified by setting all
 296 time derivatives to zero:

$$\begin{aligned}
 0 &= -E^* + S_E (w_{EE}^* \cdot E^* - w_{EI}^* \cdot I^* + P_{E_0}) \\
 0 &= -I^* + S_I (w_{IE}^* \cdot E^* - w_{II}^* \cdot I^* + P_{I_0}) \\
 0 &= \eta (E^* \cdot E^* - \alpha (w_{EE}^*)^2) \\
 0 &= \eta (E^* \cdot I^* - \alpha (w_{EI}^*)^2) \\
 0 &= \eta (I^* \cdot E^* - \alpha (w_{IE}^*)^2) \\
 0 &= \eta (I^* \cdot I^* - \alpha (w_{II}^*)^2)
 \end{aligned}$$

297 The plasticity equations yield analytical expressions for the equilibrium weights.
 298 Solving the quadratic forms:

$$\begin{aligned}
 E^* \cdot E^* - \alpha (w_{EE}^*)^2 &= 0 \Rightarrow w_{EE}^* = \frac{E^*}{\sqrt{\alpha}} \\
 E^* \cdot I^* - \alpha (w_{EI}^*)^2 &= 0 \Rightarrow w_{EI}^* = \sqrt{\frac{E^* I^*}{\alpha}} \\
 I^* \cdot E^* - \alpha (w_{IE}^*)^2 &= 0 \Rightarrow w_{IE}^* = \sqrt{\frac{I^* E^*}{\alpha}} \\
 I^* \cdot I^* - \alpha (w_{II}^*)^2 &= 0 \Rightarrow w_{II}^* = \frac{I^*}{\sqrt{\alpha}}
 \end{aligned}$$

299 Substituting these into the neural activity equations gives the following:

$$\begin{aligned}
 E^* &= S_E \left(\frac{(E^*)^2}{\sqrt{\alpha}} - \sqrt{\frac{E^* I^*}{\alpha}} \cdot I^* + P_{e_0} \right) \\
 I^* &= S_I \left(\sqrt{\frac{I^* E^*}{\alpha}} \cdot E^* - \frac{(I^*)^2}{\sqrt{\alpha}} + P_{i_0} \right)
 \end{aligned}$$

300 Simplifying the input terms:

$$\begin{aligned}\text{Input to } E^* &: \frac{1}{\sqrt{\alpha}} \left((E^*)^2 - (E^* I^*)^{1/2} \cdot I^* \right) + P_{e_0} \\ \text{Input to } I^* &: \frac{1}{\sqrt{\alpha}} \left((E^* I^*)^{1/2} \cdot E^* - (I^*)^2 \right) + P_{i_0}\end{aligned}$$

301 Solving this coupled nonlinear system numerically revealed a single biologically
302 plausible fixed point at:

$$E^* = 0.1263$$

$$I^* = 0.0874$$

$$w_{EE}^* = 1.2630$$

$$w_{EI}^* = 1.0506$$

$$w_{IE}^* = 1.0506$$

$$w_{II}^* = 0.8740$$

303 4.4 Linear Stability Analysis

304 Next, the 6×6 Jacobian matrix $\mathbf{J}$ was evaluated at the fixed point to analyze local sta-
305 bility. The system state vector is $\mathbf{X} = [E, I, w_{EE}, w_{EI}, w_{IE}, w_{II}]^T$, and the Jacobian
306 elements are partial derivatives of each equation with respect to each state variable.

307 The complete Jacobian matrix at the fixed point is:

$$\mathbf{J} = \begin{bmatrix} -1 + S'_E w_{EE}^* & -S'_E w_{EI}^* & S'_E E^* & -S'_E I^* & 0 & 0 \\ S'_I w_{IE}^* & -1 - S'_I w_{II}^* & 0 & 0 & S'_I E^* & -S'_I I^* \\ 2\eta E^* & 0 & -2\eta\alpha w_{EE}^* & 0 & 0 & 0 \\ \eta I^* & \eta E^* & 0 & -2\eta\alpha w_{EI}^* & 0 & 0 \\ \eta I^* & \eta E^* & 0 & 0 & -2\eta\alpha w_{IE}^* & 0 \\ 0 & 2\eta I^* & 0 & 0 & 0 & -2\eta\alpha w_{II}^* \end{bmatrix}$$

308 The eigenvalues of $\mathbf{J}$ are as follows:

$$\lambda_1 = -0.675240$$

$$\lambda_2 = -1.000007$$

$$\lambda_3 = -0.000889$$

$$\lambda_4 = -0.000889$$

$$\lambda_5 = -0.000881$$

$$\lambda_6 = -0.001051$$

309 These eigenvalues were obtained numerically by evaluating the Jacobian at the
 310 fixed point using the parameter values listed in Table 3. The Jacobian was derived
 311 analytically, and its eigenvalues were computed using standard numerical linear algebra
 312 routines. Due to the dimensionality and nonlinearity of the system, closed-form
 313 expressions for the eigenvalues are not available, and the stability analysis is therefore
 314 local and parameter-dependent. All code related to evaluating Jacobian matrices and
 315 eigenvalues is provided under the data availability statement.

316 Since all eigenvalues satisfy $\Re(\lambda_i) < 0$ for $i = 1, \dots, 6$, the fixed point is asymptoti-
 317 cally stable, implying that small perturbations around equilibrium decay exponentially
 318 and the system returns to the fixed point. The two most negative eigenvalues, λ_1 and

λ_2 , correspond to fast stable modes associated primarily with the neural activity variables E and I , governing the rapid relaxation of population firing rates. In contrast, the remaining eigenvalues (λ_3 – λ_6) are clustered close to zero, indicating the presence of slow modes. These slow eigenvalues are associated with the synaptic plasticity dynamics and define a slow manifold on which the synaptic weights evolve gradually toward equilibrium. The slowest timescale is determined by the least negative eigenvalue, $\lambda_5 = -0.000881$, implying that convergence of the synaptic variables occurs over a much longer temporal horizon compared to the neural activity. This strong timescale separation reflects a biologically realistic system in which neural firing adapts quickly, while synaptic weights change slowly through plasticity. Finally, all eigenvalues are real, indicating that the local dynamics near the fixed point are entirely relaxational, with no intrinsic oscillatory components.

The absence of oscillatory eigenmodes in the Jacobian spectrum implies that no intrinsic oscillations occur in the vicinity of the fixed point for the chosen parameter regime. This conclusion is strictly local and applies only to small perturbations near equilibrium. Due to the nonlinear and high-dimensional nature of the system, other parameter regimes or larger perturbations may admit oscillatory dynamics, bifurcations, or other complex attractors that are not captured by linearization. The present analysis therefore characterizes local stability rather than the global dynamical behavior of the system.

4.5 Modulated Dynamics

Introducing the oscillatory forcing term ($\gamma = 0.02$, $\omega = 251.33$ rad/s) transforms the system into a non-autonomous dynamical system:

$$\begin{aligned}
\frac{dw_{EE}}{dt} &= \eta (E \cdot E - \alpha w_{EE}^2) + \gamma \sin(\omega t) \\
\frac{dw_{EI}}{dt} &= \eta (E \cdot I - \alpha w_{EI}^2) + \gamma \sin(\omega t) \\
\frac{dw_{IE}}{dt} &= \eta (I \cdot E - \alpha w_{IE}^2) + \gamma \sin(\omega t) \\
\frac{dw_{II}}{dt} &= \eta (I \cdot I - \alpha w_{II}^2) + \gamma \sin(\omega t)
\end{aligned}$$

Numerical integration of this forced system revealed that the stable fixed point of the autonomous system evolves into a stable periodic orbit. This behavior is consistent with the system undergoing a transition where the external rhythmic input entrains the slow synaptic weight dynamics, causing all state variables to oscillate synchronously at the driving gamma frequency. This observed transition to a periodic orbit is therefore a consequence of the external rhythmic forcing, not an intrinsic bifurcation of the autonomous system. The autonomous system's fixed point remains stable; the oscillation is a direct response to the continuous external drive.

The system exhibits phase-dependent dynamics where the effective learning rate becomes:

$$\eta \left(1 + \frac{\gamma}{\eta (E \cdot E - \alpha w_{EE}^2)} \sin(\omega t) \right)$$

for non-zero Hebbian terms, creating temporal windows for enhanced plasticity. All state variables oscillate rhythmically at the driving frequency, ω , with amplitudes modulated by intrinsic network dynamics. The model demonstrates that learning is enhanced during specific phases of the oscillation while being suppressed during others. This creates rhythmic "learning windows" that repeat at approximately 40 Hz (gamma frequency), as defined by the modulation frequency $\omega = 251.33$ rad/s. During each gamma cycle, the actual learning rate modulates around the baseline $\eta = 0.05$, creating optimal temporal windows for synaptic modification.

360 The expression for the time-dependent effective learning rate should be interpreted
 361 heuristically rather than as a formal mathematical derivation. The oscillatory modula-
 362 tion introduces both additive and multiplicative influences on the plasticity dynamics,
 363 and the effective learning rate formulation serves as an intuitive interpretation of how
 364 rhythmic forcing gates synaptic modification over time. This representation is not
 365 intended as a rigorous reduction of the governing equations, but rather as a concep-
 366 tual tool for understanding phase-dependent learning effects observed in numerical
 367 simulations.

368 4.6 Simulated Plots

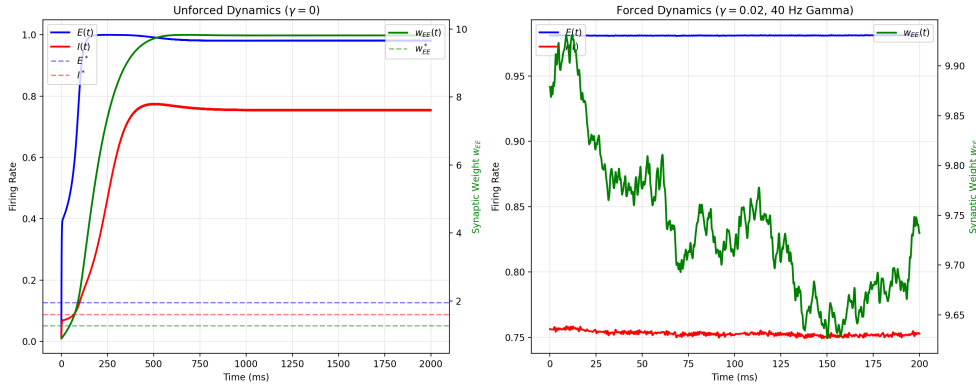


Fig. 3: Numerical simulation of the modified Wilson-Cowan model.

On the left, the unforced dynamics ($\gamma = 0$) are simulated. The system converges to a stable fixed point, with the synaptic weight, w_{EE} , showing slow, asymptotic convergence. On the right, the forced dynamics ($\gamma = 0.02$) are simulated. The system exhibits a stable periodic orbit, with all variables oscillating at the gamma frequency (40 Hz), demonstrating rhythmic entrainment. Time series show excitatory firing rate, $E(t)$ (blue), inhibitory firing rate, $I(t)$ (red), and excitatory synaptic weight, $w_{EE}(t)$ (green, right axis).

Simulation Results: Unforced and Forced Dynamics

Figure 3 illustrates the temporal evolution of the excitatory and inhibitory populations ($E(t)$ and $I(t)$) and the corresponding synaptic weight, $w_{EE}(t)$, under both unforced and rhythmically forced conditions. In the unforced case ($\gamma = 0$), the system exhibits intrinsic stability, with both $E(t)$ and $I(t)$ rapidly converging from their initial values to steady-state equilibrium points. This behavior confirms the asymptotic stability predicted by the eigenvalue analysis in Section 4.4, where all eigenvalues of the Jacobian equate to negative real parts. The synaptic weight, $w_{EE}(t)$, in contrast, evolves on a slower timescale, gradually approaching its equilibrium value, w_{EE}^* . This separation of timescales between fast neural dynamics and slow synaptic adaptation reflects the system's ability to maintain stability while permitting gradual learning through plasticity. The results confirm that, in the absence of rhythmic modulation, the modified model's network settles into a single asymptotically stable fixed point.

When rhythmic modulation is introduced ($\gamma = 0.02$), the system transitions from a stable fixed point to a stable periodic orbit. The oscillatory input at the gamma frequency (approximately 40 Hz) entrains both neural activity and synaptic dynamics, leading to rhythmic fluctuations in $E(t)$, $I(t)$, and $w_{EE}(t)$. These oscillations demonstrate that plasticity is temporally gated by the phase of the external rhythm: synaptic modification is enhanced during specific phases of the gamma cycle and suppressed

395 during others. This phase-dependent organization of plasticity creates rhythmic "learn-
396 ing windows" that structure synaptic change in time, providing a mechanistic solution
397 to the stability-plasticity dilemma. In effect, the network learns continuously yet
398 remains dynamically stable, as oscillatory entrainment constrains synaptic growth and
399 coordinates neural activity across populations.

400 5 Discussion

401 The phase-dependent learning demonstrated by this model implements a temporal
402 Hebbian principle, where the efficacy of synaptic modification depends on the precise
403 timing of neural activity relative to an ongoing gamma oscillation (around 40 Hz). This
404 rhythmic organization creates discrete "learning windows" that repeat each gamma
405 cycle, structuring synaptic change in time rather than allowing it to occur continuously.
406 This mechanism provides an elegant solution to the stability-plasticity dilemma: the
407 network can incorporate new information during specific phases while maintaining
408 operational stability during others.

409 The model naturally implements three distinct temporal scales that work in
410 conjunction: fast ($\sim$ ms) neural firing dynamics, medium ($\sim$ 25ms) gamma rhythm
411 oscillations, and slow ($\sim$ seconds) synaptic plasticity dynamics. This multi-scale tempo-
412 ral architecture enables the network to process information rapidly, organize learning
413 rhythmically, and accumulate experience gradually. Although all connection types are
414 modulated by the same oscillatory rhythm, they may be differentially affected depend-
415 ing on the precise timing of pre- and post-synaptic activity relative to the oscillation
416 phase, introducing an additional layer of specificity in synaptic modification.

417 The findings align with and extend previous work on oscillatory influences
418 on plasticity. The observed phase-dependent learning windows are consistent with
419 experimental evidence showing gamma-phase dependent plasticity in cortical and hip-
420 pocampal circuits [Huerta and Lisman \(1993\)](#), theta-gamma coupling during memory

421 tasks [Huerta and Lisman \(1995\)](#), and phase-specific stimulation effects on learning.
422 The model provides a mechanistic explanation for how such rhythmic coordina-
423 tion could emerge from the interaction between population dynamics and synaptic
424 plasticity rules.

425 The key insight from this analysis is that learning is not a continuous process,
426 but occurs in rhythmic bursts synchronized to brain oscillations. This represents a
427 fundamental organizational principle that prevents chaotic synaptic growth through
428 temporal structuring, coordinates distributed learning across multiple brain regions,
429 and allows flexible network reorganization while maintaining operational stability.

430 Several testable predictions emerge from this framework: (1) stimulation at differ-
431 ent gamma phases should produce qualitatively different learning outcomes, (2) the
432 optimal timing for inducing plasticity depends critically on the oscillation phase, and
433 (3) learning should be maximal when inputs arrive during the "preferred phase" of the
434 plasticity rhythm. These predictions could be tested using phase-specific transcranial
435 stimulation (e.g., tACS) or rhythmic sensory stimulation paradigms.

436 From a clinical perspective, these findings generate hypotheses regarding how dis-
437 ruptions in rhythmic coordination might contribute to learning difficulties. The model
438 suggests that interventions aimed at entraining oscillatory patterns could potentially
439 enhance learning by restoring optimal timing for synaptic plasticity. However, it is
440 crucial to emphasize that the present model does not provide direct evidence for
441 clinical efficacy; establishing such relevance would require empirical validation in bio-
442 logical systems and human populations. The model serves primarily as a theoretical
443 framework for understanding rhythm-plasticity interactions rather than as a clinical
444 prediction tool.

6 Conclusion

This study has developed and analyzed a hybrid computational framework that integrates the macroscopic dynamics of the Wilson-Cowan model with the microscopic principles of Hebbian synaptic plasticity. By introducing time-dependent synaptic weights governed by correlation-based rules and modulated by oscillatory input, the model bridges a gap between population-level neural activity and synaptic change. The stability analysis confirms that the unforced system possesses a stable fixed point, ensuring baseline operational stability. More significantly, the introduction of rhythmic modulation transforms this equilibrium into a stable periodic orbit, revealing that synaptic plasticity is structured into discrete, phase-dependent "learning windows."

The key theoretical advance of this model is its demonstration that gamma frequency oscillations can temporally organize synaptic modification, providing a mechanistic solution to the stability plasticity dilemma. By gating plasticity to specific phases of an oscillation, the network can incorporate new information continuously without succumbing to runaway excitation or catastrophic interference. This phase dependent learning implements a temporal Hebbian rule, where the precise timing of pre- and postsynaptic activity relative to the ongoing brain rhythm determines the magnitude and direction of synaptic change.

The model suggests that, in principle, disruptions in rhythmic coordination could influence learning-related plasticity in addition to static E/I imbalances. Whether such mechanisms play a causal role in specific conditions such as ADHD, dyslexia, or ASD remains an open empirical question beyond the scope of the present model. Consequently, interventions aimed at entraining oscillatory patterns, such as rhythmic sensory stimulation or transcranial alternating current stimulation, represent promising directions for future experimental investigation. However, establishing any clinical efficacy would require empirical validation in biological systems and human populations.

472 In summary, this model posits that learning is an inherently rhythmic process.
473 The interplay between fast neural firing, medium speed network oscillations, and slow
474 synaptic plasticity creates a multi-scale temporal framework that is fundamental to
475 stable, effective learning in biological systems. By formalizing this principle, the model
476 provides a tool for future explorations into the dynamical origins of cognitive deficits
477 and the development of temporally precise, biologically informed interventions. Over-
478 all, the present work should be interpreted as a theoretical and mechanistic model,
479 intended to generate hypotheses about rhythm-plasticity interactions rather than to
480 provide direct clinical predictions.

481 **7 Data Availability Statement**

482 All code for this paper is available at https://github.com/AmitChalmeti/Modeling_Hebbian_Learning.

483 **8 Equations**

484 Not applicable

485 **9 Tables**

486 Not applicable

487 **10 Figures**

488 Not applicable

489 **11 Algorithms, Program codes and Listings**

490 Not applicable

491 12 Cross referencing

492 Not applicable

493 13 Examples for theorem like environments

494 Not applicable

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498 Declarations

499 All authors certify that they have no affiliations or involvement in any organization
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502 Appendix A Section title of first appendix

503 Not Applicable

504 References

505 Buzsáki, G., & Wang, X.-J. (2012). Mechanisms of gamma oscillations. *Annual*
506 *Review of Neuroscience*, 35(1), 203–225, [https://doi.org/10.1146/annurev](https://doi.org/10.1146/annurev-neuro-062111-150444)
507 [-neuro-062111-150444](https://doi.org/10.1146/annurev-neuro-062111-150444)
508

509 Castellanos, F.X., & Proal, E. (2012). Large-scale brain systems in adhd: beyond the
510 prefrontal–striatal model. *Trends in Cognitive Sciences*, 16(1), 17–26, [https://](https://doi.org/10.1016/j.tics.2011.11.007)
511 doi.org/10.1016/j.tics.2011.11.007

512

513 Cipolotti, L., & Bird, C.M. (2006). Amnesia and the hippocampus. *Current opinion in*
514 *neurology*, 19(6), 693–598, <https://doi.org/10.1097/01.wco.0000247608.42320>
515 [.f9](#)

516

517 Deco, G., Jirsa, V.K., McIntosh, A.R. (2011). Emerging concepts for the dynamical
518 organization of resting-state activity in the brain. *Nature Reviews Neuroscience*,
519 12(1), 43–56,

520

521 Dohare, S., Hernandez-Garcia, J.F., Lan, Q., Rahman, P., Mahmood, A.R., Sutton,
522 R.S., et al. (2024). Loss of plasticity in deep continual learning. *Nature*,
523 632, 768–774, <https://doi.org/10.1038/s41586-024-07711-7> Retrieved from
524 <https://doi.org/10.1038/s41586-024-07711-7>

525

526 Ellenbogen, J.M., Payne, J.D., Stickgold, R. (2006). The role of sleep in declarative
527 memory consolidation: passive, permissive, active or none? *Current opinion in*
528 *neurobiology*, 16(6), 716—722, <https://doi.org/10.1016/j.conb.2006.10.006>

529

530 Fox, K., & Stryker, M. (2017). Integrating hebbian and homeostatic plasticity:
531 Introduction. *Philosophical Transactions of the Royal Society B: Biological Sci-*
532 *ences*, 372(20160413), , <https://doi.org/10.1098/rstb.2016.0413> Retrieved from
533 <https://royalsocietypublishing.org/doi/full/10.1098/rstb.2016.0413> (Introduc-
534 tion to the themed issue “Integrating Hebbian and homeostatic plasticity”)

535

536 Freeman, W.J. (1975). Mass action in the nervous system. *Academic Press*, ,
537

538 Fries, P. (2015). Rhythms for cognition: Communication through coherence. *Neuron*,
539 88(1), 220–235, <https://doi.org/10.1016/j.neuron.2015.09.034> Retrieved from
540 <https://pmc.ncbi.nlm.nih.gov/articles/PMC4605134/>
541

542 Hebb, D.O. (1949). *The organization of behavior: A neuropsychological theory*. Wiley.

543 Huerta, P., & Lisman, J. (1993). Heightened synaptic plasticity of hippocampal ca1
544 neurons during a cholinergically induced rhythmic state. *Nature*, 364(6439),
545 723–725, <https://doi.org/10.1038/364723a0>
546

547 Huerta, P., & Lisman, J. (1995). Bidirectional synaptic plasticity induced by a single
548 burst during cholinergic oscillation in ca1 in vitro. *Neuron*, 15(5), 1053–1063,
549 [https://doi.org/10.1016/0896-6273\(95\)90094-2](https://doi.org/10.1016/0896-6273(95)90094-2)
550

551 Kyriazos, T., & Poga, M. (2025). Balancing accuracy and recall in hebbian and
552 quantum-inspired learning models. *The Open Bioinformatics Journal*, 18,
553 e18750362380098, <https://doi.org/10.2174/0118750362380098250416071630>
554

555 Lehongre, K., Ramus, F., Villiermet, N., Schwartz, J.-L., Giraud, A.-L. (2011). Altered
556 low-gamma sampling in auditory cortex accounts for the three main facets of
557 dyslexia. *Neuron*, 72(6), 1080–1090, <https://doi.org/10.1016/j.neuron.2011.11>
558 .002
559

- 560 Malenka, R.C., & Bear, M.F. (2004). Synaptic plasticity and ampa receptor trafficking.
561 *Annual Review of Neuroscience*, 27, 351–379,
562
- 563 Meeter, M., & Murre, J.M. (2004). Simulating episodic memory deficits in semantic
564 dementia with the tracelink model. *Memory (Hove, England)*, 12(3), 272–287,
565 <https://doi.org/10.1080/09658210244000658>
566
- 567 Meeter, M., & Murre, J.M. (2005). Tracelink: A model of consolidation and amnesia.
568 *Cognitive neuropsychology*, 22(5), 559–587,
569
- 570 Meeter, M., & Talamini, L.M. (2002). A computational approach to memory deficits
571 in schizophrenia. *Neurocomputing*, ,
572
- 573 Nesse, W.H., Clark, K.L., Noudoost, B. (2024). Information representation in an
574 oscillating neural field model modulated by working memory signals. *Frontiers*
575 *in Computational Neuroscience*, 17, 1253234, [https://doi.org/10.3389/fncom](https://doi.org/10.3389/fncom.2023.1253234)
576 [.2023.1253234](https://doi.org/10.3389/fncom.2023.1253234)
577
- 578 Rubenstein, J.L.R., & Merzenich, M.M. (2003). Model of autism: increased ratio of
579 excitation/inhibition in key neural systems. *Genes, Brain and Behavior*, 2(5),
580 255–267, <https://doi.org/10.1046/j.1601-183X.2003.00037.x>
581
- 582 Ryan, L., Nadel, L., Keil, K. (2001). Hippocampal complex and retrieval of recent
583 and very remote autobiographical memories: evidence from functional magnetic

584 resonance imaging in neurologically intact people. *Hippocampus*, 11(6), 707—
585 714, <https://doi.org/10.1002/hipo.1086>
586

587 Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature*, 437(7063),
588 1272—1278, <https://doi.org/10.1038/nature04286>
589

590 Stickgold, R., James, L., Hobson, J.A. (2000). Visual discrimination learning requires
591 sleep after training. *Nature neuroscience*, 3(12), 1237—1238, [https://doi.org/](https://doi.org/10.1038/81756)
592 [10.1038/81756](https://doi.org/10.1038/81756)
593

594 Turrigiano, G. (2012). Homeostatic synaptic plasticity: local and global mechanisms
595 for stabilizing neuronal function. *Cold Spring Harbor Perspectives in Biology*,
596 4(1), a005736,
597

598 Uhlhaas, P.J., & Singer, W. (2010). Abnormal neural oscillations and synchrony in
599 schizophrenia. *Nature Reviews Neuroscience*, 11, 100–113, [https://doi.org/](https://doi.org/10.1038/nrn2774)
600 [10.1038/nrn2774](https://doi.org/10.1038/nrn2774)
601

602 Watt, A., & Desai, N. (2010, 06). Homeostatic plasticity and stdp: Keeping a neuron's
603 cool in a fluctuating world. *Frontiers in synaptic neuroscience*, 2, 5, [https://](https://doi.org/10.3389/fnsyn.2010.00005)
604 doi.org/10.3389/fnsyn.2010.00005
605

606 Wilson, H.R., & Cowan, J.D. (1972). Excitatory and inhibitory interactions in
607 localized populations of model neurons. *Biophysical Journal*, 12(1), 1–24,
608

609 Yizhar, O., Fenno, L., Prigge, M., Schneider, F., Davidson, T.J., O'Shea, D.J., ...
610 Deisseroth, K. (2011). Neocortical excitation/inhibition balance in information
611 processing and social dysfunction. *Nature*, 477, 171–178,
612