



# Dreaming reflects neural resynchronization after sleep-dependent neuroplastic adaptations.

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### Abstract

Synchronous and coordinated neural electrical activity is essential for proper mental functioning in the wake state. Sleep-dependent neuroplastic repair and adaptation that occur due to oxidative damage, inflammation, learning, and memory formation fostered by wake state experiences alter the finely-tuned electrical activity of neurons. Resynchronization after neuroplastic repair and adaptation, but before waking, is needed to redress such adverse effects. These circuit-specific resynchronizations restore the unique sequential activity patterns needed for the diversity of mental functions. Dreaming is the result of neural circuit activations during sleep as part of resynchronization processes. Dream content is explained by the information load of neural circuits that underwent extensive neuroplastic repair and adaptation and that were activated during resynchronization. Neural circuit activation sequences and their combinations during resynchronization determine the narratives and bizarreness of dreams.

*Keywords:* sleep, dreaming, neuroplasticity, electrical activity, resynchronization, neural circuit activation

## Dreaming Reflects Neural Resynchronization After Sleep-Dependent Neuroplastic Adaptations

Though various theories of sleep and dreaming exist, they have been found wanting (Domhoff, 2017; Frank & Heller, 2018; Hoel, 2021; Mageo, 2019; Revonsuo, 2000; Scarpelli et al., 2022; Stickgold et al., 2001). This lack of a cogent and comprehensive theory of sleep and dreaming is clearly an unsatisfactory affair that requires continuing efforts of synthesis and integration. The theory presented here is intended to further address this deficiency and it is organized as explained below.

I start by emphasizing the critical role performed by neural activities uniquely synchronized and exquisitely coordinated within specific neural pathways. Daily activities generate oxidative damage and inflammation in neurons. Such damage needs to be repaired and preempted (learning) through various neuroplastic processes. Executing neuroplastic processes is incompatible with continued wake-state mental functioning because it risks disrupting the finely-tuned electrical activities of neurons and so might impair mental functioning. A sleep state is required to permit these restorative and adaptive processes to occur. After neuroplastic modifications, neurons need to be resynchronized within circuits and circuits re-coordinated with others. Thus, inputs to a given neuron need to arrive with the proper timing and in the proper sequence for it to function correctly. Neuroplastic processes occur during NREM sleep and excessive alterations are constrained by the slow oscillations to facilitate resynchronization during REM sleep. These circuit-specific resynchronizations manifest as overall desynchronous activity. Resynchronization is coordinated by pontine centers and external stimuli are ‘simulated’ by the brainstem – thus enabling coordination with the peripheral nervous system. Resynchronization requires the activation and functional integration of neural circuits and this produces dreams. Because neural circuits are activated

during both NREM and REM sleep, but activated in different ways, dreams occur in both stages, but manifest differently. Dream content is related to events that most provoked neuroplastic processes, thus will be related to the most salient wake state experiences. The strange narratives and bizarre nature of some dreams are due to the specific sequences in which neural circuits are activated and these depend on those circuits most in need of resynchronization. I conclude with a short discussion of the adaptive value of dreams.

### **The Importance of Synchronized Neural Activity**

Mental processes are dependent on neuronal structures and activities that are highly complex, finely tuned and exquisitely synchronized (Poulet & Crochet, 2019; Puentes-Mestril et al., 2019; Zbili et al., 2020). In pyramidal neurons, for example, excitatory post synaptic potentials decay with dendrite length and the amount of decay from a given synapse is dependent on which dendrite it is located and where it is located on the dendrite (Hill et al., 2013; Spruston, 2008). Furthermore, synapses may have different morphological characteristics and function with different time courses (Jonas, 2000). The synchronized and coordinated activations of multiple synapses needed to trigger a specific action potential in a given neuron must therefore occur on millisecond time-scales (Hill et al., 2013; Spruston, 2008; Yin et al., 2018). Clearly, a disruption of intricately coordinated and synchronously ordered activities that could occur at various synapses distributed amongst complex dendritic arborizations in a neuron would impact the function of the circuit in which it serves and thus have adverse effects on downstream mental processes.

### **Neuronal Damage Occurs from Activity During Wakefulness**

Neurons have high energy demands met via oxidative phosphorylation (Hall et al., 2012) that produces reactive oxygen species (ROS). Under conditions of activity, energy demands are substantially amplified leading to elevated ROS production (Coulson et al., 2022; Qiu et al., 2020; Raefsky & Mattson, 2017). This, in turn, causes damage to lipids,

proteins and nucleic acids (Coulson et al., 2022; Schieber & Chandel, 2014). ROS can also activate the immune system leading to damaging inflammation (Besedovsky et al., 2018; Schieber & Chandel, 2014).

Learning a novel task, requiring the same neural circuitry to be activated repeatedly, therefore predisposes neurons in that circuit to oxidative damage. To counter such damage, repair processes are induced in the neuron (Cao et al., 2020; Qiu et al., 2020; Schulze, 2004; Zada et al., 2019). Furthermore, to preempt or reduce experience-dependent damaging metabolic demands, memory formation and associated processes require neuroplastic adaptations and modifications (Girardeau & Lopes-dos-Santos, 2021; Klinzing et al., 2019; Schmidt et al., 2021). These repair and modification processes are themselves energy intensive and will conflict with the high energy demands of neural functioning that occur during wakefulness. Thus, sleep deprivation, sleep apnea, and insomnia, where the wake state is prolonged (and repair impaired), aggravate oxidative stress, neuroinflammation, neurodegeneration and apoptosis (Besedovsky et al., 2018; Mahalakshmi et al., 2022; Suresh et al., 2021; Villafuerte et al., 2015; L. Wang & Aton, 2022) and degrades effective cognitive and immune function while sleep is restorative (e.g., Besedovsky et al., 2018; Klinzing et al., 2019; K. T. Li et al., 2022; Raven et al., 2019; Wunderlin et al., 2021).

### **Neuroplastic Processes Alter the Strength and Time Course of Electrical Activity**

Neural plasticity involves numerous subcellular modifications that include DNA repair (Zada et al., 2019), gene transcription (Qiu et al., 2020; Schulze, 2004), protein synthesis (Tudor et al., 2016; Yap & Greenberg, 2018), changes in dendritic spine structure (Spruston, 2008), axonal plasticity (Galliano et al., 2021; Jamann et al., 2018; Lazari et al., 2018; Leterrier, 2016), mitochondrial genesis and trafficking (Chang et al., 2006; Course & Wang, 2016; Lee et al., 2018; Lin & Sheng, 2015; Ly & Verstreken, 2006; Palikaras & Tavernarakis, 2020), myelination (Lazari et al., 2018; Pan et al., 2020; Ronzano et al., 2020; Sinclair et al.,

2017) and synaptic plasticity (Galliano et al., 2021; W. Li et al., 2017; Lieberman & Sulzer, 2020; Stampaponi Bassi et al., 2019).

At least some of these might alter neuronal properties such as synaptic contact size and morphology, glial wrapping and the density of glutamate transporters (Jonas, 2000) and thus the neuron's firing characteristics (Benna & Fusi, 2016; Fusi et al., 2005; Liu et al., 2021; Spruston, 2008; Zenke et al., 2017). Changes that occur in the firing characteristics of one or more neurons will also affect other neurons. This might alter interneuronal delay, the time taken for electrical activity in one neuron to be propagated to the next one, a critical parameter in fully connected spiking neural networks (Vibert et al., 1994) and affect long term potentiation (LTP) where LTP augmented excitation in a neuron has both individual and network level effects producing unstable circuits with excessive activity (Jamann et al., 2018; Turrigiano, 2012; Wefelmeyer et al., 2016).

### **Altered Electrical Activity Requires Resynchronization**

The firing characteristics of a neuron after damage repair or neuroplastic modification must occur with the correct timing for a neural ensemble to function effectively (Turrigiano, 2012). Neural ensembles must also have the appropriate firing characteristics after multiple neurons underwent modifications because the dynamics of neural networks depend on spiking activity that must be coordinated within specified neural circuits (Brinkman et al., 2022; Reynolds et al., 2022). Disruptions of needed synchronized inputs to a neuron could limit single-cell participation in some circuits with associated information loss (Valero et al., 2017). It may also decorrelate the correlated spiking activity required of some cortical neurons (Bartolo et al., 2020; Genç et al., 2016; Smith & Kohn, 2008) or desynchronize specific neural circuits that need to be synchronized for neural processing (Bosman et al., 2012; Poulet & Crochet, 2019) leading to impairment of perceptual functions and affecting mental health (Nobukawa et al., 2022). To restore proper mental functioning, neural activity

must be resynchronized and re-coordinated.

### **Slow Waves Constrain Distributed Neuroplastic Processes**

Important durable repair and adaptation processes occur during NREM sleep. Plasticity processes in corticothalamic circuitry are evident after prolonged and rhythmic firing during SWS (Steriade, 2000) with both depression and potentiation occurring (Brzosko et al., 2019). Synchronized firing during SWS improves memory processes (Abel et al., 2013; Girardeau & Lopes-dos-Santos, 2021; Staresina et al., 2015) suggesting that intrinsic neuronal firing patterns were occurring within bounds established by network activity. Thus, bounds appear to be determined by the slow oscillation that controls the synchronization of other sleep rhythms. It acts as a carrier wave in interconnected circuits throughout the brain and coordinates the firing characteristics of intracortical circuits, spindles in thalamocortical circuits and sharp wave ripples in hippocampocortical circuits (Crunelli & Hughes, 2010; Girardeau & Lopes-dos-Santos, 2021; Jones, 2009; Puentes-Mestril et al., 2019; Schreiner et al., 2022; Steriade & Amzica, 1998). Furthermore, by virtue of constraining neuroplastic processes, the slow oscillation might also have a homeostatic effect on neuroplastic events.

The thalamus is a relay and gating system for external stimuli (Jones, 2009). While asleep, it blocks transmission of external signals to the neocortex (Steriade, 2000). The slow oscillation arises in the thalamus (Crunelli & Hughes, 2010; Ujma et al., 2022) and it actively organizes sleep rhythms in the neocortex (Schreiner et al., 2022). As sleep deepens from stages 2 to 4, the frequency of slow oscillations increases and clear periods of delta waves emerge that are more frequent and longer (Crunelli & Hughes, 2010). Thus, it appears that upon falling asleep and the commencement of neural repair and adaptation the thalamus replaces external signals with structured slow wave activity to constrain and coordinate neuroplastic processes.

### **Simulating Wakefulness to Resynchronize Neural Activity**

After neuroplastic processes that might have altered neural synchronization, even within the bounds determined by slow waves as discussed above, functional resynchronization is required to produce the unique, event-specific sequential neural activity patterns needed for wakefulness. Ponto-geniculo-occipital (PGO) waves occur as discrete biphasic wave events occurring during REM sleep and are good candidates to orchestrate such resynchronization. They have highly synchronous effects in those neural circuits that they propagate through (Puentes-Mestril et al., 2019; Ramirez-Villegas et al., 2021). In the cortex they might present as sawtooth waves that widely activate, modulate and synchronize neuronal activity in specific circuits producing overall high spatiotemporal heterogeneity (Frauscher et al., 2020; Girardeau & Lopes-dos-Santos, 2021; Ramirez-Villegas et al., 2021). Thus, not all neural circuits are firing synchronously, but the resynchronization pertains to specific circuits – those that required neuroplastic alterations – even though those circuits and others that they need to be coordinated with might span several brain regions. These unique sequential neural activity patterns needed for a diversity of wakefulness functions produce desynchronous activity (Beaman et al., 2017; Bermudez Contreras et al., 2013; Frauscher et al., 2020).

The resynchronization processes themselves might require rapid adjustments to neuronal function. Given that synchronization implies the fine-tuning of conduction velocities and that myelination regulates conduction velocities (de Faria et al., 2021; Fields, 2015; Ronzano et al., 2020), myelin plasticity and nodes of Ranvier architecture plasticity (Rasband & Peles, 2016), with adjustments of the internodal and nodal lengths and modifications of the ion channel densities and clustering and types, are prime candidates for mediating resynchronization (Chapman & Hill, 2020; Pajevic et al., 2022).

Other potential rapid resynchronization processes are changes in the axon initial segment (Jamann et al., 2021; Leterrier et al., 2017), mitochondrial redistribution (Course & Wang, 2016; Lin & Sheng, 2015), skewed distributions of brain parameters (Buzsáki and

Mizuseki, 2014) and oligodendrocyte-mediated feedback (Pajevic et al., 2022). This feedback involves global and transient responses to local action potentials in the axons myelinated by a given oligodendrocyte. They produce rapid myelination that selectively modifies the conduction velocity of a given axon (Pajevic et al., 2022).

Resynchronization must not only occur mutually between functionally connected neural circuits of the central nervous system, but also between the central and peripheral nervous systems and in such a way as to permit accurate and sensitive interfacing with the external world. The thalamus that blocks external signals and replaces them with structured slow wave activity to constrain and coordinate neuroplastic processes during NREM sleep, during REM sleep permits signals from the brainstem to be transmitted to the cortex. These signals seem to simulate sensory, as opposed to social (Revonsuo et al., 2015), aspects of waking life, thus producing contractions of the middle ear muscles, myoclonic twitches of skeletal muscles, irregularities in respiratory and cardiac activity (Dickerson et al., 1993; Ioannides et al., 2022; Simor et al., 2018) and rapid eye movements with wakefulness-like aspects of visual processing (Andrillon et al., 2015). It is therefore of interest that both pontine waves (the rodent analogue of PGO waves) and REM sleep are regulated by the same excitatory neurons in the dorsomedial medulla (Schott et al., 2022) because the medulla plays a role in many important autonomic functions such as breathing, heart rate and blood pressure and also in the sleep wake cycle (Waldman, 2009). As a result of the involvement of the medulla via regulation of PGO waves and REM sleep, the autonomic nervous system is resynchronized with the central nervous system. All this occurs while maintaining skeletal muscle atonia (Simor et al., 2018), possibly to reduce harm by avoiding activity when external stimuli are diminished, nervous system coordination impaired and phantom simulated stimuli might be present. Thus, some processes that occur during REM sleep resynchronization seem intended to mimic those that occur during wakefulness to effect an adequate, reality-competent

resynchronization and prepare the brain for optimal activity during the wake state.

### Dreaming

Objectively, the ‘simulation’ of predominantly sensory-related input during REM sleep to resynchronize specific neural circuits produces measurable brain activity that resembles wakefulness. Subjectively, the same brain activity that resembles wakefulness produces dreams. The transition from resembling wakefulness to wakefulness itself depends on the locus coeruleus (LC). During wakefulness, LC activity is high, but variable. Sparse LC activity with high noradrenaline levels that occur during NREM sleep (Osorio-Forero et al., 2022) coordinates sleep spindles, themselves constrained by slow oscillations. Thus, LC activity appears involved in constraining neuroplastic events to be compatible with sensory-related activity from the brainstem and facilitate brain-body coordination (Osorio-Forero et al., 2022; Totah et al., 2018). Though LC activity and NA levels during NREM sleep seem to promote brain-body coordination in preparation for the waking state, during REM sleep the LC is quiescent (Osorio-Forero et al., 2022) to avoid wakefulness while circuit-specific resynchronization is in progress.

### Sleep Architecture

The first NREM episode might be relatively long as many neuroplastic processes occur. In contrast, the first REM episode during which circuit-specific resynchronization is attempted might reveal shortcomings in those neuroplastic processes. Such obstacles might result in limited synchrony required between specific neural circuits and involve a limited number of neural circuits. This results in truncated REM sleep followed by further NREM sleep to address these shortcomings possibly via the perineuronal net (Bozzelli et al., 2018). The perineuronal net is a lattice-like structure present in several brain regions that include the cortex and hippocampus. It appears to be involved in synchronous neuronal population discharges that occur with gamma oscillations and sharp-wave ripples (Bozzelli et al., 2018).

After the NREM remedial neuroplastic processes have been completed, a subsequent REM episode follows consisting of more extensive resynchronization and coordination of specific neurons within neural circuits as well as involving more neural circuits with which they are functionally integrated. Further shortcomings revealed are addressed in further NREM episodes. These will generally decline in duration or frequency (Abel et al., 2013; Simor et al., 2018) as mending and adaptation needs reduce. Conversely, REM episodes will increase in duration or frequency (Abel et al., 2013; Simor et al., 2018) as specific neurons within neural circuits become more comprehensively resynchronized and more and more neural circuits are integrated in the resynchronization procedure to produce the unique sequential neural activity patterns for event-specific mental functioning. This should result in highly-specific patterns of both locally synchronous brain activity and ones that extend over larger brain regions. Patterns of short-range synchrony are indeed observed during phasic REM periods while patterns of long-range synchrony occur during tonic REM periods (Simor et al., 2018). Furthermore, external information processing appears to be modest during phasic REM periods with cortical activity mostly detached from sensory input. In contrast, during tonic REM sleep, a wake-like state is achieved where alertness and environmental processing appear to be partially maintained (Simor et al., 2018). This is congruent with the notion that wakefulness is simulated during resynchronization to prepare for the wake state.

### **Dream Content**

Neural circuits that required more neuroplastic adaptations due to specific waking activities that precipitated this need are likely to require more extensive resynchronization. The information load of these activated circuits are thus more likely to feature in dreams. Thus, waking experiences that placed a particular neuroplastic burden on the nervous system, due to its frequency, intensity or emotional valence, are likely to feature prominently in dream content (Nemeth & Fazekas, 2018; Vallat et al., 2017) whereas waking experiences of a more

general and diffuse character might not. Dream content will generally feature events related to the preceding day – the day residue effect – but further resynchronization and reintegration of neuroplastically adapted neural circuits might also occur on subsequent days and account for the dream lag effect (Carr & Solomonova, 2019; Picard-Deland & Nielsen, 2022). Moreover, any neural circuit that requires neuroplastic adaptation needs to be resynchronized with – at least – some other downstream neural circuits with which it interfaces even if these neural circuits themselves did not require neuroplastic adaptations. Consequently, dream content might also reflect activation of these latter neural circuits. This establishes, for example, the concordance observed between head, limb and eye movements during REM sleep (Leclair-Visonneau et al., 2010).

During resynchronization, neural circuits are activated in sequences and with frequencies that are deemed dependent on the types and extent of neuroplastic adaptations effected. These sequences and their frequencies predominantly generate the narratives of dream episodes. The weirdness of dreams (Carr & Solomonova, 2019; Scarpelli et al., 2022) also derives from the resynchronization and integration demands of neural circuits that are determined by neuroplastic alterations, but predominantly from the combinations of neural circuits that are ‘tested’ together. Even though aspects of simulation occur during resynchronization, resynchronization is not likely to result in an activation of neural circuits in the same coordinated manner as experienced in the waking state and provoked by external stimuli because the activation of neural circuits is dependent on the extent of neuroplastic alterations.

### Dream Length

Because REM episodes will increase in duration or frequency (Abel et al., 2013; Carr & Solomonova, 2019; Simor et al., 2018), scope arises for more dreams or dreams of longer duration. Indeed, this is expected as specific neural circuits need to become more

comprehensively resynchronized and functionally integrated with more downstream neural circuits.

### NREM Dreaming

Dreaming can also occur in NREM sleep (Carr & Solomonova, 2019; Nemeth & Fazekas, 2018) because neural circuit activation, thus dreaming as a consequence of such activation, also occurs during NREM sleep. However, the activation of intracortical circuits, spindles in thalamocortical circuits and sharp wave ripples in hippocampocortical circuits (Crunelli & Hughes, 2010; Girardeau & Lopes-dos-Santos, 2021; Jones, 2009; Puentes-Mestril et al., 2019; Schreiner et al., 2022; Steriade & Amzica, 1998) are coordinated by the slow oscillation, not by PGO waves. Thus, the activation of neural circuits occurs in a different manner from that in REM sleep and different dream attributes could reasonably be expected. Though NREM dreams do indeed appear to be qualitatively different from REM dreams, being shorter, more mundane and more thought-like (Carr & Solomonova, 2019; Nemeth & Fazekas, 2018), it is yet unclear how to provide a more detailed mechanistic explanation accounting for these specific attributes.

### Moods and Emotions

Moods and emotions are associated with the limbic system and play a central role in dreaming (Simor et al., 2018; Stickgold et al., 2001). They are highly prominent in REM dreaming (Carr & Solomonova, 2019; Stickgold et al., 2001) but appear less so in NREM dreaming (Carr & Solomonova, 2019; Scarpelli et al., 2022). Parts of the limbic system, including the amygdala and limbic forebrain, will also need to be resynchronized and functionally reintegrated with other neural circuits because they might also undergo neuroplastic adaptations, especially after exposure to adverse and traumatic events (Scarpelli et al., 2022). Thus, in earlier REM episodes, resynchronization will involve the activation of fewer associated neural circuits while in later episodes, resynchronization will be more

extensive with a wider range of participating neural circuits being concurrently activated with unique sequential patterns. Moods and emotions might appear as more isolated dream experiences initially and, as resynchronization progresses, will occur along with other dream facets to produce dreams where emotions are more integrated with complex narratives (Carr & Solomonova, 2019; Simor et al., 2018). Furthermore, they might also occur during NREM episodes (Moyne et al., 2022) and be qualitatively different (Carr & Solomonova, 2019) as explained above. Even if no part of the limbic system required neuroplastic adaptations, other circuits that did so need to be synchronized with them and that would result in moods and emotions featuring in the concomitant dreams. The excessively strong activation of limbic system neural circuits during a resynchronization episode might explain the occurrence of some nightmares.

### **Vividness and Complexity**

The vividness and complexity of dreams (Carr & Solomonova, 2019; Nemeth & Fazekas, 2018; Simor et al., 2018) are potentially accounted for by three factors. The first is that the intensity of dream experiences is likely related to the intensity or extent of neural repair or neural adaptions required – generally as a result of experiences. Such provoked changes are expected to produce robust neuroplastic alterations within the constraints imposed by SWS that might yet produce strong, even though moderated, excitation of neural circuits during the resynchronization process. Homeostatic processes and neuromodulation moderate excessive excitation of neural circuitry, including on short timescales (Brzosko et al., 2019; Turrigiano, 2012; Wefelmeyer et al., 2016; Zenke et al., 2017). The pruning of neural spines during REM sleep (W. Li et al., 2017) might be a further mechanism for moderating excitation intensity. Increased vividness and bodily sensations also occur when sensory stimulation is applied during sleep stages (Scarpelli et al., 2022), thus simulated stimuli deriving from the brainstem might contribute to the vividness of dreams. Finally,

besides intensity moderation that might occur, as more neural circuits are involved and gradually become more synchronized and functionally coordinated dreams also become more coherent and attain a greater ‘focus’ of vividness and complexity.

### **Discussion**

Dreams might have adaptive value in several respects. The resynchronization and coordination of neural circuits have an obvious benefit in restoring and improving mental function. It is also likely that dreaming would eliminate parasitic modes of excitation (Crick & Mitchison, 1983) that might otherwise occur in poorly synchronized and poorly coordinated circuits. Furthermore, dreaming consists of the activation of different neural circuits in a sequence and manner dependent on the degree and extent of neuroplastic adaptations needed in various neural circuits – and these are in turn precipitated by experiences during wakefulness. Thus, unusual sequences of activation might occur. Memories due to such unusual activations, whether explicitly formed and accessible through dream recall or implicitly formed due to a resynchronization sequence, might provide unexpected insights or solutions to problems. This is compatible with dreams having a biological function (Revonsuo, 2000). It is precisely those circuits most challenged during wakefulness that will undergo the most extensive neuroplastic adaptations and be most extensively resynchronized in a particular pattern. Such sequences of activation might well represent available solutions to the presented challenges in terms of extant neural circuits and their knowledge loading.

### **References**

- Abel, T., Havekes, R., Saletin, J. M., & Walker, M. P. (2013). Sleep, plasticity and memory from molecules to whole-brain networks. *Current Biology*, 23(17), R774–R788.  
<https://doi.org/10.1016/j.cub.2013.07.025>

- Andrillon, T., Nir, Y., Cirelli, C., Tononi, G., & Fried, I. (2015). Single-neuron activity and eye movements during human REM sleep and awake vision. *Nature Communications*, 6(1), Article 7884. <https://doi.org/10.1038/ncomms8884>
- Bartolo, R., Saunders, R. C., Mitz, A. R., & Averbeck, B. B. (2020). Information-Limiting Correlations in Large Neural Populations. *The Journal of Neuroscience*, 40(8), 1668–1678. <https://doi.org/10.1523/JNEUROSCI.2072-19.2019>
- Beaman, C. B., Eagleman, S. L., & Dragoi, V. (2017). Sensory coding accuracy and perceptual performance are improved during the desynchronized cortical state. *Nature communications*, 8(1), Article 1308. <https://doi.org/10.1038/s41467-017-01030-4>
- Benna, M. K., & Fusi, S. (2016). Computational principles of synaptic memory consolidation. *Nature Neuroscience*, 19(12), 1697–1706. <https://doi.org/10.1038/nn.4401>
- Bermudez Contreras, E. J., Schjetnan, A. G. P., Muhammad, A., Bartho, P., McNaughton, B. L., Kolb, B., ... & Luczak, A. (2013). Formation and reverberation of sequential neural activity patterns evoked by sensory stimulation are enhanced during cortical desynchronization. *Neuron*, 79(3), 555–566.  
<https://doi.org/10.1016/j.neuron.2013.06.013>
- Besedovsky, L., Lange, T., & Haack, M. (2019). The sleep-immune crosstalk in health and disease. *Physiological reviews*, 99(3), 1325–1380.  
<https://doi.org/10.1152/physrev.00010.2018>
- Bosman, C. A., Schoffelen, J.-M., Brunet, N., Oostenveld, R., Bastos, A. M., Womelsdorf, T., Rubehn, B., Stieglitz, T., De Weerd, P., & Fries, P. (2012). Attentional stimulus selection through selective synchronization between monkey visual areas. *Neuron*, 75(5), 875–888. <https://doi.org/10.1016/j.neuron.2012.06.037>
- Bozzelli, L. P., Alaiyed, S., Kim, E., Villapol, S., & Conant, K. (2018). Proteolytic remodeling of perineuronal nets: Effects on synaptic plasticity and neuronal

population dynamics. *Neural Plasticity*, 2018, Article 5735789.

<https://doi.org/10.1155/2018/5735789>

Brinkman, B. A. W., Yan, H., Maffei, A., Park, I. M., Fontanini, A., Wang, J., & La Camera,

G. (2022). Metastable dynamics of neural circuits and networks. *Applied Physics*

*Reviews*, 9(1), Article 011313. <https://doi.org/10.1063/5.0062603>

Brzosko, Z., Mierau, S. B., & Paulsen, O. (2019). Neuromodulation of spike-timing-

dependent plasticity: Past, present, and future. *Neuron*, 103(4), 563–581.

<https://doi.org/10.1016/j.neuron.2019.05.041>

Buzsáki, G., & Mizuseki, K. (2014). The log-dynamic brain: how skewed distributions affect

network operations. *Nature Reviews Neuroscience*, 15(4), 264–278.

<https://doi.org/10.1038/nrn3687>

Cao, J., Herman, A. B., West, G. B., Poe, G., & Savage, V. M. (2020). Unraveling why we

sleep: Quantitative analysis reveals abrupt transition from neural reorganization to

repair in early development. *Science advances*, 6(38), Article eaba0398.

<https://doi.org/10.1126/sciadv.aba0398>

Carr, M., & Solomonova, E. (2019). Dream recall and content in different sleep stages. In K.

Valli & R. Hoss (Eds.), *Dreams: Understanding biology, psychology, and culture*

(pp.188–195). Greenwood Publishing Group.

Chang, D. T. W., Honick, A. S., & Reynolds, I. J. (2006). Mitochondrial trafficking to

synapses in cultured primary cortical neurons. *Journal of Neuroscience*, 26(26), 7035–

7045. <https://doi.org/10.1523/JNEUROSCI.1012-06.2006>

Chapman, T. W., & Hill, R. A. (2020). Myelin plasticity in adulthood and aging.

*Neuroscience Letters*, 715, Article 134645.

<https://doi.org/10.1016/j.neulet.2019.134645>

- Coulson, R. L., Mourrain, P., & Wang, G. X. (2022). Sleep deficiency as a driver of cellular stress and damage in neurological disorders. *Sleep Medicine Reviews*, 63, Article 101616. <https://doi.org/10.1016/j.smrv.2022.101616>
- Course, M. M., & Wang, X. (2016). Transporting mitochondria in neurons. *F1000Research*, 5, F1000 Faculty Rev-1735. <https://doi.org/10.12688/f1000research.7864.1>
- Crick, F., & Mitchison, G. (1983). The function of dream sleep. *Nature*, 304, 111–114.
- Crunelli, V., & Hughes, S. W. (2010). The slow (<1 Hz) rhythm of non-REM sleep: A dialogue between three cardinal oscillators. *Nature Neuroscience*, 13(1), 9–17. <https://doi.org/10.1038/nn.2445>
- de Faria, O., Pivonkova, H., Varga, B., Timmeler, S., Evans, K. A., & Káradóttir, R. T. (2021). Periods of synchronized myelin changes shape brain function and plasticity. *Nature Neuroscience*, 24(11), 1508–1521. <https://doi.org/10.1038/s41593-021-00917-2>
- Dickerson, L. W., Huang, A. H., Thumher, M. M., Nearing, B. D., & Verrier, R. L. (1993). Relationship between coronary hemodynamic changes and the phasic events of rapid eye movement sleep. *Sleep*, 16(6), 550–557.
- Domhoff, G. W. (2017). The invasion of the concept snatchers: The origins, distortions, and future of the continuity hypothesis. *Dreaming*, 27(1), 14–39. <https://doi.org/10.1037/drm0000047>
- Fields, R. D. (2015). A new mechanism of nervous system plasticity: Activity-dependent myelination. *Nature Reviews Neuroscience*, 16(12), 756–767. <https://doi.org/10.1038/nrn4023>
- Frank, M. G., & Heller, H. C. (2018). The Function(s) of Sleep. In H.-P. Landolt & D.-J. Dijk (Eds.), *Sleep-Wake Neurobiology and Pharmacology* (Vol. 253, pp. 3–34). Springer International Publishing. [https://doi.org/10.1007/164\\_2018\\_140](https://doi.org/10.1007/164_2018_140)

- Frauscher, B., von Ellenrieder, N., Dolezalova, I., Bouhadoun, S., Gotman, J., & Peter-Derex, L. (2020). Rapid eye movement sleep sawtooth waves are associated with widespread cortical activations. *The Journal of Neuroscience*, 40(46), 8900–8912.  
<https://doi.org/10.1523/JNEUROSCI.1586-20.2020>
- Fusi, S., Drew, P. J., & Abbott, L. F. (2005). Cascade models of synaptically stored memories. *Neuron*, 45(4), 599–611. <https://doi.org/10.1016/j.neuron.2005.02.001>
- Galliano, E., Hahn, C., Browne, L. P., R. Villamayor, P., Tufo, C., Crespo, A., & Grubb, M. S. (2021). Brief sensory deprivation triggers cell type-specific structural and functional plasticity in olfactory bulb neurons. *The Journal of Neuroscience*, 41(10), 2135–2151. <https://doi.org/10.1523/JNEUROSCI.1606-20.2020>
- Genç, E., Schölvinck, M. L., Bergmann, J., Singer, W., & Kohler, A. (2016). Functional connectivity patterns of visual cortex reflect its anatomical organization. *Cerebral Cortex*, 26(9), 3719–3731. <https://doi.org/10.1093/cercor/bhv175>
- Girardeau, G., & Lopes-dos-Santos, V. (2021). Brain neural patterns and the memory function of sleep. *Science*, 374(6567), 560–564.  
<https://doi.org/10.1126/science.abi8370>
- Hall, C. N., Klein-Flugge, M. C., Howarth, C., & Attwell, D. (2012). Oxidative phosphorylation, not glycolysis, powers presynaptic and postsynaptic mechanisms underlying brain information processing. *Journal of Neuroscience*, 32(26), 8940–8951. <https://doi.org/10.1523/JNEUROSCI.0026-12.2012>
- Hill, D. N., Varga, Z., Jia, H., Sakmann, B., & Konnerth, A. (2013). Multibranch activity in basal and tuft dendrites during firing of layer 5 cortical neurons in vivo. *Proceedings of the National Academy of Sciences*, 110(33), 13618–13623.  
<https://doi.org/10.1073/pnas.1312599110>

- Hoel, E. (2021). The overfitted brain: Dreams evolved to assist generalization. *Patterns*, 2(5), Article 100244. <https://doi.org/10.1016/j.patter.2021.100244>
- Ioannides, A. A., Orphanides, G. A., & Liu, L. (2022). Rhythmicity in heart rate and its surges usher a special period of sleep, a likely home for PGO waves. *Current Research in Physiology*, 5, 118–141. <https://doi.org/10.1016/j.crphys.2022.02.003>
- Jamann, N., Dannehl, D., Lehmann, N., Wagener, R., Thielemann, C., Schultz, C., Staiger, J., Kole, M. H. P., & Engelhardt, M. (2021). Sensory input drives rapid homeostatic scaling of the axon initial segment in mouse barrel cortex. *Nature Communications*, 12(1), Article 23. <https://doi.org/10.1038/s41467-020-20232-x>
- Jamann, N., Jordan, M., & Engelhardt, M. (2018). Activity-dependent axonal plasticity in sensory systems. *Neuroscience*, 368, 268–282.  
<https://doi.org/10.1016/j.neuroscience.2017.07.035>
- Jonas, P. (2000). The time course of signaling at central glutamatergic synapses. *Physiology*, 15(2), 83–89. <https://doi.org/10.1152/physiologyonline.2000.15.2.83>
- Jones, E. G. (2009). Synchrony in the interconnected circuitry of the thalamus and cerebral cortex. *Annals of the New York Academy of Sciences*, 1157(1), 10–23.  
<https://doi.org/10.1111/j.1749-6632.2009.04534.x>
- Klinzing, J. G., Niethard, N., & Born, J. (2019). Mechanisms of systems memory consolidation during sleep. *Nature Neuroscience*, 22(10), 1598–1610.  
<https://doi.org/10.1038/s41593-019-0467-3>
- Lazari, A., Koudelka, S., & Sampaio-Baptista, C. (2018). Experience-related reductions of myelin and axon diameter in adulthood. *Journal of Neurophysiology*, 120(4), 1772–1775. <https://doi.org/10.1152/jn.00070.2018>
- Leclair-Visonneau, L., Oudiette, D., Gaymard, B., Leu-Semenescu, S., & Arnulf, I. (2010). Do the eyes scan dream images during rapid eye movement sleep? Evidence from the

- rapid eye movement sleep behaviour disorder model. *Brain*, 133(6), 1737–1746.  
<https://doi.org/10.1093/brain/awq110>
- Lee, A., Hirabayashi, Y., Kwon, S.-K., Lewis, T. L., & Polleux, F. (2018). Emerging roles of mitochondria in synaptic transmission and neurodegeneration. *Current Opinion in Physiology*, 3, 82–93. <https://doi.org/10.1016/j.cophys.2018.03.009>
- Leterrier, C. (2016). The axon initial segment, 50 years later: A nexus for neuronal organization and function. *Current Topics in Membranes* 77, 185–233.  
<https://doi.org/10.1016/bs.ctm.2015.10.005>
- Leterrier, C., Clerc, N., Rueda-Boroni, F., Montersino, A., Dargent, B., & Castets, F. (2017). Ankyrin G membrane partners drive the establishment and maintenance of the axon initial segment. *Frontiers in Cellular Neuroscience*, 11, Article 6.  
<https://doi.org/10.3389/fncel.2017.00006>
- Li, K. T., He, X., Zhou, G., Yang, J., Li, T., Hu, H., Ji, D., Zhou, C., & Ma, H. (2022). Rational designing of oscillatory rhythmicity for memory rescue in plasticity-impaired learning networks. *Cell Reports*, 39(2), Article 110678.  
<https://doi.org/10.1016/j.celrep.2022.110678>
- Li, W., Ma, L., Yang, G., & Gan, W.-B. (2017). REM sleep selectively prunes and maintains new synapses in development and learning. *Nature Neuroscience*, 20(3), 427–437.  
<https://doi.org/10.1038/nn.4479>
- Lieberman, O. J., & Sulzer, D. (2020). The Synaptic Autophagy Cycle. *Journal of Molecular Biology*, 432(8), 2589–2604. <https://doi.org/10.1016/j.jmb.2019.12.028>
- Lin, M.-Y., & Sheng, Z.-H. (2015). Regulation of mitochondrial transport in neurons. *Experimental Cell Research*, 334(1), 35–44.  
<https://doi.org/10.1016/j.yexcr.2015.01.004>

- Liu, W., Liu, Q., Crozier, R. A., & Davis, R. L. (2021). Analog transmission of action potential fine structure in spiral ganglion axons. *Journal of Neurophysiology*, 126(3), 888–905. <https://doi.org/10.1152/jn.00237.2021>
- Ly, C. V., & Verstreken, P. (2006). Mitochondria at the synapse. *The Neuroscientist*, 12(4), 291–299. <https://doi.org/10.1177/1073858406287661>
- Mageo, J. (2019). Mimesis versus simulation: Contemporary dream theory and the nature of dream mentation. *Dreaming*, 29(4), 370–387. <https://doi.org/10.1037/drm0000120>
- Mahalakshmi, A. M., Lokesh, P., Hediyal, T. A., Kalyan, M., Vichitra, C., Essa, M. M., Qoronfleh, M. W., Pandi-Perumal, S. R., & Chidambaram, S. B. (2022). Impact of sleep deprivation on major neuroinflammatory signal transduction pathways. *Sleep and Vigilance*, 6(1), 101–114. <https://doi.org/10.1007/s41782-022-00203-6>
- Moyné, M., Legendre, G., Arnal, L., Kumar, S., Sterpenich, V., Seeck, M., Grandjean, D., Schwartz, S., Vuilleumier, P., & Domínguez-Borràs, J. (2022). Brain reactivity to emotion persists in NREM sleep and is associated with individual dream recall. *Cerebral Cortex Communications*, 3(1), Article tgac003. <https://doi.org/10.1093/texcom/tgac003>
- Nemeth, G., & Fazekas, P. (2018). Beyond the REM--NREM dichotomy: a multidimensional approach to understanding dreaming. *Journal of Consciousness Studies*, 25(11–12), 13–33.
- Nobukawa, S., Wagatsuma, N., Ikeda, T., Hasegawa, C., Kikuchi, M., & Takahashi, T. (2022). Effect of steady-state response versus excitatory/inhibitory balance on spiking synchronization in neural networks with log-normal synaptic weight distribution. *Cognitive Neurodynamics*, 16(4), 871–885. <https://doi.org/10.1007/s11571-021-09757-z>

Osorio-Forero, A., Cherrad, N., Banterle, L., Fernandez, L. M. J., & Lüthi, A. (2022). When the locus coeruleus speaks up in sleep: Recent insights, emerging perspectives.

*International Journal of Molecular Sciences*, 23(9), Article 5028.

<https://doi.org/10.3390/ijms23095028>

Pajevic, S., Plenz, D., Bassar, P. J., & Fields, R. D. (2022). *Oligodendrocyte-mediated myelin plasticity and its role in neural synchronization*. BioRxiv, 2022–07.

<https://doi.org/10.1101/2022.07.03.498570>

Palikaras, K., & Tavernarakis, N. (2020). Regulation and roles of mitophagy at synapses.

*Mechanisms of Ageing and Development*, 187, Article 111216.

<https://doi.org/10.1016/j.mad.2020.111216>

Pan, S., Mayoral, S. R., Choi, H. S., Chan, J. R., & Kheirbek, M. A. (2020). Preservation of a remote fear memory requires new myelin formation. *Nature Neuroscience*, 23(4),

487–499. <https://doi.org/10.1038/s41593-019-0582-1>

Picard-Deland, C., & Nielsen, T. (2022). Targeted memory reactivation has a sleep stage-specific delayed effect on dream content. *Journal of Sleep Research*, 31(1). Article e13391. <https://doi.org/10.1111/jsr.13391>

Poulet, J. F. A., & Crochet, S. (2019). The cortical states of wakefulness. *Frontiers in Systems Neuroscience*, 12, Article 64. <https://doi.org/10.3389/fnsys.2018.00064>

Puentes-Mestril, C., Roach, J., Niethard, N., Zochowski, M., & Aton, S. J. (2019). How rhythms of the sleeping brain tune memory and synaptic plasticity. *Sleep*, 42(7), Article zsz095. <https://doi.org/10.1093/sleep/zsz095>

Qiu, J., Dando, O., Febery, J. A., Fowler, J. H., Chandran, S., & Hardingham, G. E. (2020). Neuronal activity and its role in controlling antioxidant genes. *International Journal of Molecular Sciences*, 21(6), Article 1933. <https://doi.org/10.3390/ijms21061933>

- Raefsky, S. M., & Mattson, M. P. (2017). Adaptive responses of neuronal mitochondria to bioenergetic challenges: Roles in neuroplasticity and disease resistance. *Free Radical Biology and Medicine*, 102, 203–216.  
<https://doi.org/10.1016/j.freeradbiomed.2016.11.045>
- Ramirez-Villegas, J. F., Besserve, M., Murayama, Y., Evrard, H. C., Oeltermann, A., & Logothetis, N. K. (2021). Coupling of hippocampal theta and ripples with pontogeniculoccipital waves. *Nature*, 589(7840), 96–102.  
<https://doi.org/10.1038/s41586-020-2914-4>
- Rasband, M. N., & Peles, E. (2016). The nodes of Ranvier: Molecular assembly and maintenance. *Cold Spring Harbor Perspectives in Biology*, 8(3), Article a020495.  
<https://doi.org/10.1101/cshperspect.a020495>
- Raven, F., Meerlo, P., Van der Zee, E. A., Abel, T., & Havekes, R. (2019). A brief period of sleep deprivation causes spine loss in the dentate gyrus of mice. *Neurobiology of Learning and Memory*, 160, 83–90. <https://doi.org/10.1016/j.nlm.2018.03.018>
- Revonsuo, A. (2000). The reinterpretation of dreams: An evolutionary hypothesis of the function of dreaming. *Behavioral and brain sciences*, 23(6), 877–901.
- Revonsuo, A., Tuominen, J., & Valli, K. (2015). The avatars in the machine: Dreaming as a simulation of social reality. In T. K. Metzinger & J. M. Windt (Eds.) *Open MIND*. MIND Group.
- Reynolds, J. N. J., Avvisati, R., Dodson, P. D., Fisher, S. D., Oswald, M. J., Wickens, J. R., & Zhang, Y.-F. (2022). Coincidence of cholinergic pauses, dopaminergic activation and depolarisation of spiny projection neurons drives synaptic plasticity in the striatum. *Nature Communications*, 13(1), Article 1296. <https://doi.org/10.1038/s41467-022-28950-0>

- Ronzano, R., Thetiot, M., Lubetzki, C., & Desmazieres, A. (2020). Myelin plasticity and repair: Neuro-glial choir sets the tuning. *Frontiers in Cellular Neuroscience*, 14, Article 42. <https://doi.org/10.3389/fncel.2020.00042>
- Scarpelli, S., Alfonsi, V., Gorgoni, M., & De Gennaro, L. (2022). What about dreams? State of the art and open questions. *Journal of Sleep Research*, 31(4) Article e13609. <https://doi.org/10.1111/jsr.13609>
- Schieber, M., & Chandel, N. S. (2014). ROS function in redox signaling and oxidative stress. *Current Biology*, 24(10), R453–R462. <https://doi.org/10.1016/j.cub.2014.03.034>
- Schmidt, S., Gull, S., Herrmann, K.-H., Boehme, M., Irintchev, A., Urbach, A., Reichenbach, J. R., Klingner, C. M., Gaser, C., & Witte, O. W. (2021). Experience-dependent structural plasticity in the adult brain: How the learning brain grows. *NeuroImage*, 225, Article 117502. <https://doi.org/10.1016/j.neuroimage.2020.117502>
- Schott, A., Baik, J., Chung, S., & Weber, F. (2022). *A medullary hub for controlling REM sleep and pontine waves*. ResearchSquare <https://doi.org/10.21203/rs.3.rs-1534766/v1>
- Schreiner, T., Kaufmann, E., Noachtar, S., Mehrkens, J. H., & Staudigl, T. (2022). The human thalamus orchestrates neocortical oscillations during NREM sleep. *Nature Communications*, 13(1), 5231. <https://doi.org/10.1038/s41467-022-32840-w>
- Schulze, G. (2004). Sleep protects excitatory cortical circuits against oxidative damage. *Medical Hypotheses*, 63(2), 203–207. <https://doi.org/10.1016/j.mehy.2004.02.040>
- Simor, P., Gombos, F., Blaskovich, B., & Bódizs, R. (2018). Long-range alpha and beta and short-range gamma EEG synchronization distinguishes phasic and tonic REM periods. *Sleep*, 41(3), Article zsx210. <https://doi.org/10.1093/sleep/zsx210>
- Sinclair, J. L., Fischl, M. J., Alexandrova, O., Heß, M., Grothe, B., Leibold, C., & Kopp-Scheinpflug, C. (2017). Sound-evoked activity influences myelination of brainstem

- axons in the trapezoid body. *The Journal of Neuroscience*, 37(34), 8239–8255.  
<https://doi.org/10.1523/JNEUROSCI.3728-16.2017>
- Smith, M. A., & Kohn, A. (2008). Spatial and temporal scales of neuronal correlation in primary visual cortex. *Journal of Neuroscience*, 28(48), 12591–12603.  
<https://doi.org/10.1523/JNEUROSCI.2929-08.2008>
- Spruston, N. (2008). Pyramidal neurons: Dendritic structure and synaptic integration. *Nature Reviews Neuroscience*, 9(3), 206–221. <https://doi.org/10.1038/nrn2286>
- Stampanoni Bassi, M., Iezzi, E., Gilio, L., Centonze, D., & Buttari, F. (2019). Synaptic plasticity shapes brain connectivity: Implications for network topology. *International Journal of Molecular Sciences*, 20(24), Article 6193.  
<https://doi.org/10.3390/ijms20246193>
- Staresina, B. P., Bergmann, T. O., Bonnefond, M., Van Der Meij, R., Jensen, O., Deuker, L., ... & Fell, J. (2015). Hierarchical nesting of slow oscillations, spindles and ripples in the human hippocampus during sleep. *Nature neuroscience*, 18(11), 1679–1686.  
<https://doi.org/10.1038/nn.4119>
- Steriade, M. (2000). Corticothalamic resonance, states of vigilance and mentation. *Neuroscience*, 101(2), 243–276. [https://doi.org/10.1016/S0306-4522\(00\)00353-5](https://doi.org/10.1016/S0306-4522(00)00353-5)
- Steriade, M., & Amzica, F. (1998). Slow sleep oscillation, rhythmic K-complexes, and their paroxysmal developments. *Journal of Sleep Research*, 7(S1), 30–35.  
<https://doi.org/10.1046/j.1365-2869.7.s1.4.x>
- Stickgold, R., Hobson, J. A., Fosse, R., & Fosse, M. (2001). Sleep, learning, and dreams: Off-line memory reprocessing. *Science*, 294(5544), 1052–1057.  
<https://doi.org/10.1126/science.1063530>

- Suresh, K., Shankar, V., & Dayanand, C. (2021). Impact of REM sleep deprivation and sleep recovery on circulatory neuroinflammatory markers. *Sleep Science*, 14(1), Article 64. <https://doi.org/10.5935/1984-0063.20190157>
- Sweatt, J. D., & Hawkins, K. E. (2016). The molecular neurobiology of the sleep-deprived, fuzzy brain. *Science Signaling*, 9(425), Article fs7-fs7. <https://doi.org/10.1126/scisignal.aaf6196>
- Totah, N. K., Neves, R. M., Panzeri, S., Logothetis, N. K., & Eschenko, O. (2018). The locus coeruleus is a complex and differentiated neuromodulatory system. *Neuron*, 99(5), 1055–1068. <https://doi.org/10.1016/j.neuron.2018.07.037>
- Tudor, J. C., Davis, E. J., Peixoto, L., Wimmer, M. E., van Tilborg, E., Park, A. J., Poplawski, S. G., Chung, C. W., Havekes, R., Huang, J., Gatti, E., Pierre, P., & Abel, T. (2016). Sleep deprivation impairs memory by attenuating mTORC1-dependent protein synthesis. *Science Signaling*, 9(425), Article ra41-ra41. <https://doi.org/10.1126/scisignal.aad4949>
- Turrigiano, G. (2012). Homeostatic synaptic plasticity: Local and global mechanisms for stabilizing neuronal function. *Cold Spring Harbor Perspectives in Biology*, 4(1), a005736–a005736. <https://doi.org/10.1101/cshperspect.a005736>
- Ujma, P. P., Szalárdy, O., Fabó, D., Erőss, L., & Bódizs, R. (2022). Thalamic activity during scalp slow waves in humans. *NeuroImage*, 257, Article 119325. <https://doi.org/10.1016/j.neuroimage.2022.119325>
- Valero, M., Averkin, R. G., Fernandez-Lamo, I., Aguilar, J., Lopez-Pigozzi, D., Brotons-Mas, J. R., Cid, E., Tamas, G., & Menendez de la Prida, L. (2017). Mechanisms for selective single-cell reactivation during offline sharp-wave ripples and their distortion by fast ripples. *Neuron*, 94(6), 1234-1247. <https://doi.org/10.1016/j.neuron.2017.05.032>

- Vallat, R., Lajnef, T., Eichenlaub, J.-B., Berthomier, C., Jerbi, K., Morlet, D., & Ruby, P. M. (2017). Increased evoked potentials to arousing auditory stimuli during sleep: Implication for the understanding of dream recall. *Frontiers in Human Neuroscience*, 11, Article 132. <https://doi.org/10.3389/fnhum.2017.00132>
- Vibert, J.-F., Pakdaman, K., & Azmy, N. (1994). Interneural delay modification synchronizes biologically plausible neural networks. *Neural Networks*, 7(4), 589–607. [https://doi.org/10.1016/0893-6080\(94\)90039-6](https://doi.org/10.1016/0893-6080(94)90039-6)
- Villafuerte, G., Miguel-Puga, A., Murillo Rodríguez, E., Machado, S., Manjarrez, E., & Arias-Carrión, O. (2015). Sleep deprivation and oxidative stress in animal models: a systematic review. *Oxidative medicine and cellular longevity*, 2015, Article 234952. <https://doi.org/10.1155/2015/234952>
- Waldman, S. D. (2009). The medulla oblongata. In S. D. Waldman (Ed.), *Pain Review* (p. 208). W.B. Saunders. <https://doi.org/10.1016/B978-1-4160-5893-9.00120-9>
- Wang, L., & Aton, S. J. (2022). Perspective – ultrastructural analyses reflect the effects of sleep and sleep loss on neuronal cell biology. *Sleep*, 45(5), Article zsac047. <https://doi.org/10.1093/sleep/zsac047>
- Wefelmeyer, W., Puhl, C. J., & Burrone, J. (2016). Homeostatic plasticity of subcellular neuronal structures: From inputs to outputs. *Trends in Neurosciences*, 39(10), 656–667. <https://doi.org/10.1016/j.tins.2016.08.004>
- Wunderlin, M., Züst, M. A., Hertenstein, E., Fehér, K. D., Schneider, C. L., Klöppel, S., & Nissen, C. (2021). Modulating overnight memory consolidation by acoustic stimulation during slow-wave sleep: A systematic review and meta-analysis. *Sleep*, 44(7), Article zsaa296. <https://doi.org/10.1093/sleep/zsaa296>

Yap, E.-L., & Greenberg, M. E. (2018). Activity-regulated transcription: Bridging the gap between neural activity and behavior. *Neuron*, 100(2), 330–348.

<https://doi.org/10.1016/j.neuron.2018.10.013>

Yin, L., Zheng, R., Ke, W., He, Q., Zhang, Y., Li, J., Wang, B., Mi, Z., Long, Y., Rasch, M.

J., Li, T., Luan, G., & Shu, Y. (2018). Autapses enhance bursting and coincidence detection in neocortical pyramidal cells. *Nature Communications*, 9(1), Article 4890.

<https://doi.org/10.1038/s41467-018-07317-4>

Zada, D., Bronshtein, I., Lerer-Goldshtain, T., Garini, Y., & Appelbaum, L. (2019). Sleep increases chromosome dynamics to enable reduction of accumulating DNA damage in single neurons. *Nature Communications*, 10(1), Article 895.

<https://doi.org/10.1038/s41467-019-08806-w>

Zbili, M., Rama, S., Yger, P., Inglebert, Y., Boumedine-Guignon, N., Fronzaroli-Molinier, L., Brette, R., Russier, M., & Debanne, D. (2020). Axonal Na<sup>+</sup> channels detect and transmit levels of input synchrony in local brain circuits. *Science Advances*, 6(19), Article eaay4313. <https://doi.org/10.1126/sciadv.aay4313>

Zenke, F., Gerstner, W., & Ganguli, S. (2017). The temporal paradox of Hebbian learning and homeostatic plasticity. *Current Opinion in Neurobiology*, 43, 166–176.

<https://doi.org/10.1016/j.conb.2017.03.015>