Published in final edited form as:
Exp Brain Res. 2012 August ; 221(2): 123–128. doi:10.1007/s00221-012-3167-8.

Alpha oscillations and the control of voluntary saccadic behavior

Jordan P. Hamm,
Department of Psychology, BioImaging Research Center, University of Georgia, Psychology Building, Athens, GA 30602-3013, USA. Department of Neuroscience, BioImaging Research Center, University of Georgia, Psychology Building, Athens, GA 30602-3013, USA

Dean Sabatinelli, and
Department of Psychology, BioImaging Research Center, University of Georgia, Psychology Building, Athens, GA 30602-3013, USA. Department of Neuroscience, BioImaging Research Center, University of Georgia, Psychology Building, Athens, GA 30602-3013, USA

Brett A. Clementz
Department of Psychology, BioImaging Research Center, University of Georgia, Psychology Building, Athens, GA 30602-3013, USA. Department of Neuroscience, BioImaging Research Center, University of Georgia, Psychology Building, Athens, GA 30602-3013, USA

Brett A. Clementz: clementz@uga.edu

Abstract

The purpose of this review is to explore the dynamic properties of alpha oscillations as biological covariates of intra- and inter-individual variance in saccadic behavior. A preponderance of research suggests that oscillatory dynamics in the alpha band co-vary with performance on a number of visuo-spatial cognitive tasks. Here we discuss a growing body of research relating these measures to saccadic behavior, focusing also on how task related and spontaneous measures of alpha oscillations may serve as potential biomarkers for ocular motor dysfunction in clinical populations.

Keywords

Alpha; Saccades; Schizophrenia; Antisaccades; Oscillations

Introduction

The generation and control of saccadic behavior is a critical skill serving diverse cognitive purposes (Hutton 2008). First, selection and foveation of behaviorally relevant visual stimuli has survival value. Second, resisting attraction to salient visual events that compete with current behavioral requirements enables goal driven behavior in complex visual environments (Munoz and Everling 2004). This latter ability requires coordination of both local and distant cortical circuitries.

In experimental settings, humans and other primates display remarkable variability in saccadic behavior even following extensive practice (Dyckman and McDowell 2005).

Correspondence to: Brett A. Clementz, clementz@uga.edu.

Conflict of interest All authors report no biomedical financial interests or potential conflicts of interest.

Distributions of saccadic latencies have large variances and are, in certain conditions, multimodal (Hamm et al. 2010). In addition, during antisaccade tasks, which probe the ability to suppress saccades to salient peripheral stimuli in favor of foveating a competing spatial location, errors persist across multiple trial repetitions (Hamm et al. 2012). Some aspect of saccadic behavior may therefore be subject to stochastic brain processes not under direct control of the actor.

A recently hypothesized source of variability in saccadic response latency and control is cortical oscillations in the alpha band (8–14 Hz) recorded by electroencephalography (EEG; Hamm et al. 2010, 2012). Cortical and thalamocortical alpha oscillations (with a period of 75–125 ms) theoretically reflect cyclic fluctuation between low (inhibited) and high excitability neuronal states (Mathewson et al. 2011; Romei et al. 2008a). Alpha oscillation characteristics (e.g., power and laterality) can be determined by task demands on the actor (Klimesch et al. 2007) but other features may be stochastic rather than under direct cognitive control (e.g., instantaneous phase; Mathewson et al. 2009). The extent to which this latter possibility accounts for saccadic performance variability requires further investigation.

One way to address this issue is to study the neural correlates of saccadic behavior among individuals with schizophrenia (SZ). SZ depart from the healthy persons on the proportion of fast reaction time (and express) saccades (Clementz 1996; Reilly et al. 2008) and antisaccade error rate (McDowell and Clementz 2001; Reilly et al. 2008). The extent to which these performance deviations in SZ can be attributed to oscillatory brain dynamics is unknown. Interestingly, though, resting state and stimulus evoked dynamics of EEG alpha oscillations are abnormal in SZ, and such abnormalities are heritable (Hong et al. 2011), show associations with candidate risk genotypes (Venables et al. 2009), and may reflect more fundamental thalomocortical disruptions associated with the disease (Hughes and Crunelli 2005; Llinas et al. 1999). In this paper, relevant saccadic neural network anatomy and the neural mechanisms and behavioral correlates of alpha oscillations will be described. A hypothesis that both controlled and stochastic properties of saccadic network alpha activity are a significant source of (1) behavioral variability in the general human population, and (2) deviance in saccadic performance parameters among SZ will be presented.

Saccadic control and visuo-spatial attention networks associated with alpha activity

Saccades are ultimately controlled by pontine and midbrain nuclei innervated directly from the retina and indirectly from basal ganglia, thalamic nuclei, and cerebellar structures (Leigh and Zee 2006). During saccadic planning and execution, these subcortical nodes are modulated by input from cortical structures including frontal, parietal, and supplementary eye fields (FEF, PEF, and SEF), anterior cingulate cortices (ACC), and dorsolateral prefrontal cortex (DLPFC; McDowell et al. 2008). These cortical hubs modulate and receive input from other sensory (primary visual cortex; middle occipital gyrus, MOG) and sensori-motor structures (posterior parietal cortex, PPC; precuneus) in accord with internally driven saccadic goals (Clementz et al. 2007).

The cortical saccade network is also involved in the more general task of allocating visuospatial attention (Corbetta et al. 1998). In accord with spatial and temporal expectancy, FEF and PEF modulate activity in sensory cortices, including primary visual and ventral and dorsal extrastriate cortices (for a review, see Mazer 2011), perhaps constituting a top down control function. Allocation of visuospatial attention and saccade generation can be differentiated neurophysiologically and behaviorally (Mazer 2011), but the two operations

nonetheless overlap at the mesoscopic network level (Corbetta et al. 1998) including a right hemisphere tendency (Thiebaut de Schotten et al. 2011; Hamm et al. 2010).

Recent research on the neurophysiology of brain networks highlights the significance of ongoing oscillations and oscillatory dynamics in the understanding of behavioral control. Coherent oscillations may comprise an efficient mechanism for spatially separated neural populations to achieve precise temporal synchronization, maximizing their impact on downstream and subcortical targets (Llinás et al. 2005). Larger cortical networks may synchronize via lower frequency oscillations while more focal networks entrain to faster rhythms (Kopell et al. 2000). Oscillatory coherence in low frequencies (4–12 Hz) occurs mostly between distal regions, possibly as indices of and/or mechanisms for top-down modulation (von Stein and Sarnthein 2000). Likewise, mid- (von Stein and Sarnthein 2000) and high-gamma oscillations (>60 Hz) appear to reflect highly local cortical activity (Llinás et al. 2005). The physiological reason for this relationship between physical scale and oscillatory frequency may be a function of axonal conduction delays. Specifically, the larger the range of distances involved in the network, the greater the temporal variability in signal transmission, the wider the wavelength and lower the frequency (Kopell et al. 2000).

Substantial research effort has been devoted to understanding spontaneous, induced, and task-elicited properties of alpha oscillations. Alpha oscillations in the EEG have a maximum over parietal/occipital scalp locations and were initially thought to reflect an idling state of the brain (Adrian and Matthews 1934). Oscillatory activity in the alpha range has been characterized more recently as a manifestation of cortical inhibitory processes (Klimesch et al. 2007) reflecting periodic flux between exitable and suppressed cortical states (Mathewson et al. 2011). Recent work demonstrates associations of alpha phase and amplitude dynamics with visual attention processes (Capotosto et al. 2009; Busch and VanRullen 2010). While a thorough review of the precise cognitive, behavioral, and neurophysiological properties of alpha is beyond the scope of this article (see Mathewson et al. 2011), a few aspects of alpha neurophysiology and alpha/behavior relationships are key to illustrating their relevance to saccadic behavioral control.

First, alpha oscillations may arise from diverse cortico-cortical and cortico-thalamic circuits. Cortical neurons are capable of oscillating in alpha frequencies in isolation, but circuits involving the thalamic lateral geniculate nucleus, pulvinar, and reticular nucleus, in addition to primary visual cortex, have been demonstrated to play key roles in the generation and maintenance of alpha oscillations in multiple visual cortical regions (Bollimunta et al. 2011; Hughes and Crunelli 2005; Llinás et al. 2005; Lopes Da Silva et al. 1980; Steriade et al. 1990). Second, properties of alpha oscillations correlate with both basic and higher order perceptual mechanisms. Alpha power and instantaneous phase over visual (Mathewson et al. 2009) and frontal cortices predict (Busch and VanRullen 2010) and mediate (Dugué et al. 2011) perception of transient visual events, perhaps reflecting modulation of the excitability of cortical circuits (Romei et al. 2008a). Furthermore, EEG alpha power and phase characteristics also index top-down and goal driven cortical processing as they vary with spatial (Busch and VanRullen 2010) and temporal expectancy (Rohenkohl and Nobre 2011) over visual cortices, an effect that may be controlled in part by parietal and FEF regions (Capotosto et al. 2009). Frontal and parietal modulation of visual sensory neurons in accord with internal goals is shared across attentional (Capotosto et al. 2009) and saccadic control operations (Hutton 2008). Given the theoretical relationship between anatomy and oscillatory frequency, it can be hypothesized that the alpha dynamics of visuospatial attention allocation may covary with saccadic behavior. A growing body of research on saccadic behavioral control supports this proposition.

Two groups have shown that trials beginning in a specific phase of an actor's occipitally (Hamm et al. 2010) or frontally (Drewes and VanRullen 2011) distributed alpha oscillations have slower saccadic reaction times in a simple prosaccade paradigm. Recently, Hamm et al. (2012) demonstrated that errors of control in an antisaccade task are more likely when a trial begins in a particular phase of ongoing alpha oscillation; this effect was localized to both occipital and dorsolateral prefrontal cortical regions. Combined with findings regarding visual target detection performance (Mathewson et al. 2009), these studies support the possibility that variability in saccadic reaction time and saccadic control capabilities are at least partially related to fluctuations of excitability in cortical regions involved in early visual registration of trial onset.

Watanabe et al. (2010) proposed that such studies (Hamm et al. 2010) do not differentiate between fixation-offset elicited EEG signals and target anticipation EEG signals because they used fixed duration "gap" paradigms in which the pre-trial fixation point disappeared prior to target onset. Phase effects, therefore, could at least partially reflect fluctuations in motor readiness rather than visual excitability. Indeed, alpha oscillations in frontal cortices have been demonstrated to relate directly to oculor motor response generation (Clementz et al. 2001). An alternative explanation of the findings reported by Hamm et al. (2012) is that the pre-stimulus fronto-occipital saccade-related alpha effects correspond to a visuospatial attentional control network (Capotosto et al. 2009), and fluctuations in the efficacy of top-down monitoring of trial onset were identified in ongoing alpha oscillations. The degree to which prestimulus alpha phase effects reflect sensory and/or motor cortical functional fluctuations could be usefully investigated by varying gap duration in future studies (Watanabe et al. 2010).

Alpha power also has been shown to vary with saccadic performance. Lateralized parietal alpha power decreases (alpha desynchronization) during a saccadic task reflects gaze-centric stimulus gating (Van Der Werf et al. 2008, 2012; Buchholz et al. 2011). Interestingly, frontally-distributed pre-trial alpha power magnitude is inversely related to saccadic control performance, whereas post-stimulus parietal alpha power is positively correlated to saccadic control (Mazaheri et al. 2011).

Measures of power and phase of ongoing oscillations are mathematically independent, but current models (e.g. Mathewson et al. 2011) suggest that alpha phase and power effects interact such that phase exerts an effect only when power is large. Alpha reflects a tonic inhibitory mechanism that varies with phase, enabling continued, but diminished, perception of unattended visualspatial locations. Busch and VanRullen (2010) reported that alpha phase effects only exist, however, when visual spatial attention is allocated, a property that is associated with reduced alpha power. While these findings highlight topics for further investigation, it is apparent that alpha phase and power reflect cognitively and perceptually relevant variance in cortical networks subserving visual perception and saccadic control. There are fewer reports of between-subject differences in alpha power/dynamics, and how such relate to saccadic performance. High resting alpha power predicts low visual cortical excitability (Romeii et al. 2008b) as well as successful visuospatial working memory performance (Klimesch et al. 2007). It could be hypothesized that high resting alpha correlates with successful saccadic control. This growing literature indicates that alpha oscillations may occur in distinct saccadic control network circuits and (1) index the state of fronto-occipital visual stimulus readiness (or receptiveness) as well as (2) serve a top-down control function for suppressing externally driven saccades in favor of internal goals. Future studies could usefully investigate the ability to modulate alpha power/dynamics to explain inter-individual variability in saccadic performance.

Abnormal alpha dynamics may underlie saccadic abnormalities in schizophrenia

Overall, variations in alpha phase and oscillatory power in occipital, parietal, and frontal cortical regions within and between subjects account for variations in saccadic behavioral control in healthy participants (HP). A consistent finding is that SZ commit more errors than HP during antisaccade tasks (McDowell and Clementz 2001). Studies investigating visuo-motor control in SZ report heightened visual orienting and (related) impaired ability to suppress competing visuospatial information (Reilly et al. 2008). Clementz et al. (2010) showed in HP that correct antisaccade task performance requires a similar suppression, yet this study employed steady-state stimuli modulated in the alpha band, which precluded the ability to study endogenous alpha activity.

Because brain oscillations are proximal to basic genetic and neurobiological deviations (Gonzalez-Burgos and Lewis 2008), it is hypothesized that reports of neural oscillatory abnormalities represent a core deficit in SZ (Uhlhaas and Singer 2010). Although oscillatory abnormality findings span a number of frequency bands, a recent review (Moran and Hong 2011) concludes that lower frequency abnormalities (2–13 Hz) are more consistently reported than are beta (15–35 Hz) or gamma (>35 Hz) disruptions, emphasizing the role of lower frequency oscillations in organizing amplitude variation of higher frequency oscillations during healthy cognitive processes (Palva and Palva 2007). The intrinsic EEG of SZ show augmented theta/delta and reduced alpha power (e.g. Venables et al. 2009; Clementz et al. 1994; Karson et al. 1988), which correlates to psychotic symptoms (Omori et al. 1995; Merrin and Floyd 1996) and perithalamic ventricular volume (Sponheim et al. 2000), and may explain abnormalities in visual steady-state responses among SZ (Brenner et al. 2009). Stimulus elicited low frequency (delta-alpha) phase locking and single trial power is also consistently reduced in schizophrenia (Hamm et al. 2011; Clementz et al. 2004), an effect that is highly heritable (Hong et al. 2011). Thalamocortical circuits supporting EEG oscillations in the delta to alpha range rely on the same mechanisms, with metabotropic glutamate receptor 1 (mGLUR1) and muscarinic acetylcholine receptor activation determining the dominant frequency (Hughes et al. 2004, 2008; Hughes and Crunelli 2005). Importantly, thalamic aberrations have been theorized as central to SZ neuropathology and psychotic symptomology (Ferrarelli and Tononi 2011; Llinas et al. 1999). Given its known heritability and consistency across paradigms, alpha/theta/delta aberrations could be an EEG marker of thalamocortical disconnectivity among SZ (Klimesch et al. 2007).

Researchers have yet to specifically examine a link between disruptions in alpha-band measures and saccadic control in schizophrenia. Given the relationship between oscillatory frequency and spatial extent of a cortical network, a shift of intrinsic thalamocortical network oscillatory frequency could be accompanied by altered connectivity between and impaired function within the distributed cortical saccade network. Future studies investigating this link and hypothetical mechanism might initially address associations between a subject's alpha power or alpha-relative-to-theta power and antisaccade performance, or between a subject's ability to phase lock saccadic network alpha activity to expected spatiotemporal events and antisaccade performance. Second, it might be useful to demonstrate that experimenter-controlled cortical alpha dynamics have direct influence on antisaccade performance in HP or SZ. For example rhythmic transcranial magnetic stimulation can entrain intrinsic cortical alpha rhythms (Thut et al. 2011), affecting perception (Dugué et al. 2011) as well as concomitantly modifying frontal/ parietal alpha power and coherence and psychotic symptomology in SZ (Jin et al. 2006; 2011). Transcranial alternating current stimulation also has the capability of impacting perception (Kanai et al. 2008) and entraining and enhancing endogenous posterior alpha oscillations (Zaehle et al. 2010).

Of additional relevance to these propositions are findings that SZ display higher rates of express saccades than the healthy population (Clementz 1996; Reilly et al. 2008). Distributionally distinct express saccades occur at variable rates during “gap” prosaccade paradigms, and are on the order of 60 ms faster than normal saccades (Clementz 1996; Hamm et al. 2010). Hamm et al. (2010) showed that lack of an occipitally-distributed phase locking of alpha oscillations to a pretrial central fixation point predicted the occurrence of express saccades through a series of neural events that included early activation of occipital and parietal saccade-related regions. SZ may display higher rates of express saccades, therefore, because they do not synchronize endogenous alpha oscillations to external stimuli, further suggesting that alpha-generating neurophysiology is accountable for SZ saccadic deviations.

Antipsychotic medications currently used to treat SZ symptoms have little to no effect on alpha band power deviations in SZ (Takahashi et al. 2010). Interestingly, drugs targeting the mGLUR system ameliorate psychotic symptoms in SZ (Javitt 2010). Clinical trials for such compounds may usefully measure alpha activity as a biomarker of efficacy. Administering polyphenols to human participants may also improve memory-guided saccade production while decreasing alpha amplitude in temporal-parietal regions (Cimrová et al. 2011). Although the neuro-pharmacodynamics of such compounds are not well-characterized, this study demonstrated that pharmacological manipulations can act on saccade behavior and alpha oscillations in the absence of changes in other frequency bands or event related potentials. This finding also emphasizes the possible relevance of alpha oscillations for supporting saccade behavior.

Conclusion

Cortical oscillations in the alpha band impact the control of saccadic behavior, are dynamically modulated during saccadic tasks, and generally constitute a neurophysiological signature of activity in the distributed saccadic cortical network. Given the relevance of alpha dynamics for saccadic control, and the demonstrated abnormalities in alpha activity and saccadic control in persons with schizophrenia, it is hypothesized that alpha oscillations will prove a useful and sensitive biomarker for this disease and ocular motor endophenotypes. This hypothesis may be pertinent to pharmacological developments aimed at neurochemically modulating the alpha relevant metabotropic glutamate system and may hold key insights into psychosis etiology.

Acknowledgments

This work was supported by Grants from the National institutes of Health (R01s MH076998 and MH082514).

References

Adrian ED, Matthews BHC. The Berger rhythm: potential changes from the occipital lobes in man. Brain. 1934; 4:355–385.

Bollimunta A, Mo J, Schroeder CE, Ding M. Neuronal mechanisms and attentional modulation of corticothalamic α oscillations. J Neurosci. 2011; 31(13):4935–4943.10.1523/ JNEUROSCI. 5580-10.2011 [PubMed: 21451032]

Brenner CA, Krishnan GP, Vohs JL, Ahn WY, Hetrick WP, Morzorati SL, O’Donnell BF. Steady state responses: electrophysiological assessment of sensory function in schizophrenia. Schizophr Bull. 2009; 35(6):1065–1077. [PubMed: 19726534]

Buchholz VN, Jensen O, Medendorp WP. Multiple reference frames in cortical oscillatory activity during tactile remapping for saccades. J Neurosci. 2011; 31(46):16864–16871.10.1523/ JNEUROSCI.3404-11.2011 [PubMed: 22090512]

Busch NA, VanRullen R. Spontaneous EEG oscillations reveal periodic sampling of visual attention. PNAS USA. 2010; 107(37): 16048–16053.10.1073/pnas.1004801107 [PubMed: 20805482]

Capotosto P, Babiloni C, Romani GL, Corbetta M. Frontoparietal cortex controls spatial attention through modulation of anticipatory alpha rhythms. J Neurosci. 2009; 29(18):5863–5872.10.1523/JNEUROSCI.0539-09.2009 [PubMed: 19420253]

Cimrová B, Budáč S, Melicherová U, Jergelova M, Jagla F. Electrophysiological evidence of the effect of natural polyphenols upon the human higher brain functions. Neuro Endocrinol Lett. 2011; 32(4): 464–468. [PubMed: 21876511]

Clementz BA. The ability to produce express saccades as a function of gap interval among schizophrenia patients. Exp Brain Res. 1996; 111:121–130. [PubMed: 8891642]

Clementz BA, Sponheim SR, Iacono WG, Beiser M. Resting EEG in first-episode schizophrenia patients, bipolar psychosis patients, and their first-degree relatives. Psychophysiology. 1994; 31(5): 486–494.10.1111/j.1469-8986.1994.tb01052.x [PubMed: 7972603]

Clementz BA, McDowell JE, Stewart SE. Timing and magnitude of frontal activity differentiates refixation and anti-saccade performance. NeuroReport. 2001; 12(9):1863–1868.10.1097/00001756-200107030-00020 [PubMed: 11435913]

Clementz BA, Keil A, Kissler J. Aberrant brain dynamics in schizophrenia: delayed buildup and prolonged decay of the visual steady-state response. Brain Res Cogn Brain Res. 2004; 18(2):121–129.10.1016/j.cogbrainres.2003.09.007 [PubMed: 14736571]

Clementz BA, Brahmbhatt SB, McDowell JE, Brown R, Sweeney JA. When does the brain inform the eyes whether and where to move? An EEG study in humans. Cereb Cortex. 2007; 17(11):2634–2643.10.1093/cercor/bhl171 [PubMed: 17283204]

Clementz BA, Gao Y, McDowell JE, Moratti S, Keedy SK, Sweeney JA. Top-down control of visual sensory processing during an ocular motor response inhibition task. Psychophysiology. 2010; 47(6):1011–1018.10.1111/j.1469-8986.2010.01026.x [PubMed: 20477977]

Corbetta M, Akbudak E, Conturo TE, et al. A common network of functional areas for attention and eye movements. Neuron. 1998; 21(4):761–773.10.1016/S0896-6273(00)80593-0 [PubMed: 9808463]

Drewes J, VanRullen R. This is the rhythm of your eyes: the phase of ongoing electroencephalogram oscillations modulates saccadic reaction time. J Neurosci. 2011; 31:4698–4708.10.1523/JNEUROSCI.4795-10.2011 [PubMed: 21430168]

Dugué L, Marque P, VanRullen R. The phase of ongoing oscillations mediates the causal relation between brain excitation and visual perception. J Neurosci. 2011; 31(33):11889–11893.10.1523/JNEUROSCI.1161-11.2011 [PubMed: 21849549]

Dyckman KA, McDowell JE. Behavioral plasticity of antisaccade performance following daily practice. Exp Brain Res. 2005; 162(1):63–69.10.1007/s00221-004-2105-9 [PubMed: 15551081]

Ferrarelli F, Tononi G. The thalamic reticular nucleus and schizophrenia. Schizophr Bull. 2011; 37(2): 306–315. [PubMed: 21131368]

Gonzalez-Burgos G, Lewis DA. GABA neurons and the mechanisms of network oscillations: implications for understanding cortical dysfunction in schizophrenia. Schizophr Bull. 2008; 34:944–961.10.1093/schbul/sbn070 [PubMed: 18586694]

Hamm JP, Dyckman KA, Ethridge LE, McDowell JE, Clementz BA. Preparatory activations across a distributed cortical network determine production of express saccades in humans. J Neurosci. 2010; 30:7350–7357.10.1523/JNEUROSCI.0785-10. 2010 [PubMed: 20505102]

Hamm JP, Gilmore CS, Picchetti NA, Sponheim SR, Clementz BA. Abnormalities of neuronal oscillations and temporal integration to low- and high-frequency auditory stimulation in schizophrenia. Biol Psychiatry. 2011; 69:989–996.10.1016/j.biopsych. 2010.11.021 [PubMed: 21216392]

Hamm JP, Dyckman KA, McDowell JE, Clementz BA. Pre-cue fronto-occipital alpha phase and distributed cortical oscillations predict failures of cognitive control. J Neurosci. 2012; 32:7034–7041.10.1523/JNEUROSCI.5198-11.2012 [PubMed: 22593071]

Hong LE, Summerfelt A, Mitchell BD, O'Donnell P, Thaker GK. A shared low-frequency oscillatory rhythm abnormality in resting and sensory gating in schizophrenia. Clin Neurophysiol. 2011; 123:285–292.10.1016/j.clinph.2011.07.025 [PubMed: 21862398]

Hughes SW, Crunelli V. Thalamic mechanisms of EEG alpha rhythms and their pathological implications. Neuroscientist. 2005; 11(4):357–372. [PubMed: 16061522]

Hughes SW, Lörincz M, Cope DW, Blethyn KL, Kékesi KA, Parri HR, Juhász G, Crunelli V. Synchronized oscillations at alpha and theta frequencies in the lateral geniculate nucleus. Neuron. 2004; 42(2):253–268. [PubMed: 15091341]

Hughes SW, Errington A, Lorincz ML, Kékesi KA, Juhász G, Orbán G, Cope DW, Crunelli V. Novel modes of rhythmic burst firing at cognitively-relevant frequencies in thalamocortical neurons. Brain Res. 2008; 1235:12–20. [PubMed: 18602904]

Hutton SB. Cognitive control of saccadic eye movements. Brain Cogn. 2008; 68:327–340.10.1016/j.bandc.2008.08.021 [PubMed: 19028265]

Javitt DC. Glutamatergic theories of schizophrenia. Isr J Psychiatry Relat Sci. 2010; 47(1):4–16. [PubMed: 20686195]

Jin Y, Potkin SG, Kemp AS, Huerta ST, Alva G, Thai TM, Carreon D, Bunney WE Jr. Therapeutic effects of individualized alpha frequency transcranial magnetic stimulation (alphaTMS) on the negative symptoms of schizophrenia. Schizophr Bull. 2006; 32(3):556–561.10.1093/schbul/sbj020 [PubMed: 16254067]

Jin Y, Kemp AS, Huang Y, Thai TM, Liu Z, Xu W, He H, Potkin SG. Alpha EEG guided TMS in schizophrenia. Brain Stimul. 201110.1016/j.brs.2011.09.005

Kanai R, Chaieb L, Antal A, Walsh V, Paulus W. Frequency-dependent electrical stimulation of the visual cortex. Curr Biol. 2008; 18:1839–1843. [PubMed: 19026538]

Karson CN, Coppola R, Daniel DG, Weinberger DR. Computerized EEG in schizophrenia. Schizophr Bull. 1988; 14(2):193–197. [PubMed: 3201177]

Klimesch W, Sauseng P, Hanslmayr S. EEG alpha oscillations: the inhibition-timing hypothesis. Brain Res Rev. 2007; 53(1):63–88.10.1016/j.brainresrev.2006.06.003 [PubMed: 16887192]

Kopell N, Ermentrout GB, Whittington MA, Traub RD. Gamma rhythms and beta rhythms have different synchronization properties. PNAS. 2000; 97:1867–1872.10.1073/pnas.97.4.1867 [PubMed: 10677548]

Leigh, RJ.; Zee, DS. The neurology of eye movements. Vol. 4. Oxford UP; Oxford: 2006.

Llinas RR, Ribary U, Jeanmonod D, Kronberg E, Mitra PP. Thalamocortical dysrhythmia: a neurological and neuropsychiatric syndrome characterized by magnetoencephalography. Proc Natl Acad Sci U S A. 1999; 96(26):15222–15227. [PubMed: 10611366]

Llinás R, Urbano FJ, Leznik E, Ramírez RR, van Marle HJ. Rhythmic and dysrhythmic thalamocortical dynamics: GABA systems and the edge effect. Trends Neurosci. 2005; 28(6):325–333.10.1016/j.tins.2005.04.006 [PubMed: 15927689]

Lopes Da Silva FH, Vos JE, Mooibroek J, Van Rotterdam A. Relative contributions of intracortical and thalamocortical processes in the generation of alpha rhythms, revealed by partial coherence analysis. Electroencephalogr Clin Neurophysiol. 1980; 50:449–456.10.1016/0013-4694(80)90011-5 [PubMed: 6160987]

Mathewson KE, Gratton G, Fabiani M, Beck DM, Ro T. To see or not to see: prestimulus alpha phase predicts visual awareness. J Neurosci. 2009; 29:2725–2732.10.1523/JNEUROSCI.3963-08.2009 [PubMed: 19261866]

Mathewson KE, Lleras A, Beck DM, Fabiani M, Ro T, Gratton G. Pulsed out of awareness: EEG alpha oscillations represent a pulsed-inhibition of ongoing cortical processing. Front Psychol. 2011; 2:99.10.3389/fpsyg.2011.000999 [PubMed: 21779257]

Mazaheri A, DiQuattro NE, Bengson J, Geng JJ. Pre-stimulus activity predicts the winner of top-down vs. bottom-up attentional selection. PLoS One. 2011; 6(2):e16243.10.1371/journal. pone.0016243 [PubMed: 21386896]

Mazer JA. Spatial attention, feature-based attention, and saccades: three sides of one coin? Biol Psychiatry. 2011; 69(12):1147–1152.10.1016/j.biopsych.2011.03.014 [PubMed: 21529782]

McDowell JE, Clementz BA. Behavioral and brain imaging studies of saccadic performance in schizophrenia. Biol Psychol. 2001; 57(1–3):5–22. [PubMed: 11454432]

McDowell JE, Dyckman KA, Austin BP, Clementz BA. Neurophysiology and neuroanatomy of reflexive and volitional saccades: evidence from studies of humans. Brain Cogn. 2008; 68:255–270.10.1016/j.bandc.2008.08.016 [PubMed: 18835656]

Merrin EL, Floyd TC. Negative symptoms and EEG alpha in schizophrenia: a replication. Schizophr Res. 1996; 19:151–161. [PubMed: 8789913]

Moran LV, Hong LE. High vs low frequency neural oscillations in schizophrenia. Schizophr Bull. 2011; 37:659–663.10.1093/schbul/sbr056 [PubMed: 21653278]

Munoz DP, Everling S. Look away: the anti-saccade task and the voluntary control of eye movement. Nat Rev Neurosci. 2004; 5(3):218–228.10.1038/nrn1345 [PubMed: 14976521]

Omori M, Koshino Y, Murata T, Murata I, Nishio M, Sakamoto K, Horie T, Isaki K. Quantitative EEG in never-treated schizophrenic patients. Biol Psychiatry. 1995; 38(5):305–309. [PubMed: 7495924]

Palva S, Palva JM. New vistas for alpha-frequency band oscillations. Trends Neurosci. 2007; 30(4): 150–158. [Epub 2007 Feb 20]. [PubMed: 17307258]

Reilly JL, Harris MS, Khine TT, Keshavan MS, Sweeney JA. Reduced attentional engagement contributes to deficits in prefrontal inhibitory control in schizophrenia. Biol Psychiatry. 2008; 63(8):776–783.10.1016/j.biopsych.2007.11.009 [PubMed: 18191110]

Rohenkohl G, Nobre AC. Alpha oscillations related to anticipatory attention follow temporal expectations. J Neurosci. 2011; 31(40):14076–14084.10.1523/JNEUROSCI.3387-11.2011 [PubMed: 21976492]

Romei V, Brodbeck V, Michel C, Amedi A, Pascual-Leone A, Thut G. Spontaneous fluctuations in posterior alpha-band EEG activity reflect variability in excitability of human visual areas. Cereb Cortex. 2008a; 18(9):2010–2018. [PubMed: 18093905]

Romei V, Rihs T, Brodbeck V, Thut G. Resting electroencephalogram alpha-power over posterior sites indexes baseline visual cortex excitability. Neuroreport. 2008b; 19(2):203–208.10.1097/WNR. 0b013e3282f454c4 [PubMed: 18185109]

Sponheim SR, Clementz BA, Iacono WG, Beiser M. Clinical and biological concomitants of resting state EEG power abnormalities in schizophrenia. Biol Psychiatry. 2000; 48:1088–1097. [PubMed: 11094142]

Steriade M, Gloor P, Llinas R, Lopes Da Silva F, Mesulam M. Basic mechanisms of cerebral rhythmic activities. Electroencephalogr Clin Neurophysiol. 1990; 76:481–508.10.1016/0013-4694 (90)90001-Z [PubMed: 1701118]

Takahashi T, Cho RY, Mizuno T, et al. Antipsychotics reverse abnormal EEG complexity in drug-naive schizophrenia: a multiscale entropy analysis. Neuroimage. 2010; 51(1):173–182.10.1016/ j.neuroimage.2010.02.009 [PubMed: 20149880]

Thiebaut de Schotten M, Dell'Acqua F, Forkel SJ, et al. A lateralized brain network for visuospatial attention. Nat Neurosci. 2011; 14(10):1245–1246.10.1038/nn.2905 [PubMed: 21926985]

Thut G, Veniero D, Romei V, Miniussi C, Schyns P, Gross J. Rhythmic TMS causes local entrainment of natural oscillatory signatures. Curr Biol. 2011; 21:1176–1185.10.1016/j.cub.2011. 05.049 [PubMed: 21723129]

Uhlhaas PJ, Singer W. Abnormal neural oscillations and synchrony in schizophrenia. Nat Rev Neurosci. 2010; 11(2):100–113.10.1038/nrn2774 [PubMed: 20087360]

Van Der Werf J, Jensen O, Fries P, Medendorp WP. Gamma-band activity in human posterior parietal cortex encodes the motor goal during delayed prosaccades and antisaccades. J Neurosci. 2008; 28(34):8397–8405.10.1523/JNEUROSCI.0630-08.2008 [PubMed: 18716198]

Van Der Werf J, Buchholz VN, Jensen O, Medendorp WP. Reorganization of oscillatory activity in human parietal cortex during spatial updating. Cereb Cortex. 201210.1093/cercor/bhr387

Venables NC, Bernat EM, Sponheim SR. Genetic and disorder-specific aspects of resting state EEG abnormalities in schizophrenia. Schizophr Bull. 2009; 35(4):826–839.10.1093/schbul/sbn021 [PubMed: 18381357]

Von Stein A, Sarnthein J. Different frequencies for different scales of cortical integration: from local gamma to long range alpha/theta synchronization. Int J Psychophysiol. 2000; 38(3):301–313.10.1016/S0167-8760(00)00172-0 [PubMed: 11102669]

Watanabe M, Hirai M, Marino RA, Cameron IG. Occipital-parietal network prepares reflexive saccades. J Neurosci. 2010; 30(42):13917–13918.10.1523/JNEUROSCI.3884-10.2010 [PubMed: 20962213]

Zaehle T, Rach S, Herrmann CS. Transcranial alternating current stimulation enhances individual alpha activity in human EEG. PLoS One. 2010; 5(11):e13766. [PubMed: 21072168]