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Review article

Alpha oscillations and their impairment in affective and post-traumatic stress disorders

Moranne Eidelman-Rothman (Ph.D.)[a], Jonathan Levy (Ph.D.)[a], Ruth Feldman (Ph.D.)[a,b,*]

[a] The Gonda Multidisciplinary Brain Research Center, Bar-Ilan University, Israel
[b] Department of Psychology, Bar-Ilan University, Israel

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ABSTRACT

Affective and anxiety disorders are debilitating conditions characterized by impairments in cognitive and social functioning. Elucidating their neural underpinnings may assist in improving diagnosis and developing targeted interventions. Neural oscillations are fundamental for brain functioning. Specifically, oscillations in the alpha frequency range (alpha rhythms) are prevalent in the awake, conscious brain and play an important role in supporting perceptual, cognitive, and social processes. We review studies utilizing various alpha power measurements to assess abnormalities in brain functioning in affective and anxiety disorders as well as obsessive compulsive and post-traumatic stress disorders. Despite some inconsistencies, studies demonstrate associations between aberrant alpha patterns and these disorders both in response to specific cognitive and emotional tasks and during a resting state. We conclude by discussing methodological considerations and future directions, and underscore the need for much further research on the role of alpha functionality in social contexts. As social dysfunction accompanies most psychiatric conditions, research on alpha's involvement in social processes may provide a unique window into the neural mechanisms underlying these disorders.

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1. Introduction

1.1. Neuroscience perspective on psychiatric disorders

Psychiatric disorders are debilitating mental conditions characterized by cognitive alterations, emotional difficulties, and impaired functioning, all of which introduce considerable individual suffering and a dramatic reduction in quality of life (Massion et al., 1993; Rapaport et al., 2005; Saarni et al., 2007; Zatzick

* Corresponding author at: Department of Psychology and the Gonda Brain Research Center, Bar-Ilan University, Ramat-Gan 52900, Israel.
E-mail address: feldman.ruth@gmail.com (R. Feldman).

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et al., 1997). Although therapeutic interventions (behavioral and pharmacological) exist for most disorders, such interventions are not effective for all patients, leaving many untreated (Arnone, 2005; Norrholm, 2010; Rush et al., 2006). Psychiatric disorders have long been the focus of empirical research aimed at understanding their behavioral and physiological manifestations. During the past decades, much research has been directed to elucidate the neural underpinnings of different psychiatric conditions, with the ultimate goal of identifying biomarkers that can aid in the diagnosis and development of efficient therapeutic interventions (Başar, 2013; Uhlhaas and Singer, 2012). Utilizing different noninvasive methodological approaches, neuroimaging and electrophysiological studies provided valuable insights into the functional abnormalities associated with psychiatric disorders and their relation to clinical symptoms (Drevets, 2001, 2000; Etkin and Wager, 2007; Phillips et al., 2008; Sheline, 2003). Much of this research used functional magnetic resonance imaging (fMRI), which measures brain activity with high spatial precision; however its BOLD signal is of low temporal resolution and provides an indirect measure of neuronal activity (Luck, 2014; Papanicolaou, 2009). Electroencephalography (EEG) and magnetoencephalography (MEG) techniques, on the other hand, record brain activity with high temporal resolution and their signal directly reflects electric currents and magnetic fields, respectively, which are generated by neural activity (Hämäläinen et al., 1993; Vrba and Robinson, 2001). These properties enable a noninvasive investigation of oscillatory activity and its abnormalities in psychiatric conditions – the focus of the current review.

1.2. Alpha oscillations: interpretation, measurement, and potential use as a neuromarker for psychiatric conditions

1.2.1. Neural oscillations

Neural oscillations (or "neural rhythms") are a pervasive feature of neuronal activity in the cerebral cortex (Donner and Siegel, 2011). They reflect periodic fluctuations of excitability in neural populations generated by transmembrane currents, which give rise to electric potentials known as 'local field potentials' (LFPs). The LFPs of temporally synchronized neural activities are measurable from the scalp by means of EEG and MEG recordings (Buzsáki et al., 2012; Ros et al., 2014). Neural rhythms and their behavioral correlates are highly conserved throughout mammalian evolution (Buzsáki et al., 2013; Buzsáki and Draguhn, 2004), underscoring their fundamental role in supporting brain function. Neural rhythms are thought to underpin perceptive and cognitive processes in both animals and humans (Clayton et al., 2015; Wang, 2010; Ward, 2003), supporting integrative functions such as perceptual inference, top-down attention, and decision-making (Donner and Siegel, 2011). Disruption of oscillatory behavior may therefore affect a wide range of cognitive and perceptual processes (Uhlhaas and Singer, 2010, 2006).

1.2.2. The alpha rhythm

Alpha rhythm (~8–12 Hz) is the predominant oscillation in the awake, conscious brain that functions to sustain higher intrinsic cortical functioning and serves an integrative role by synchronizing brain activity in different brain rhythms (Klimesch, 2012). Since its discovery almost a century ago (Berger, 1929), the alpha rhythm has been largely considered as an "idling rhythm". This interpretation leaned on the observation that alpha rhythm increases when subjects are not engaged in any task as well as in cortical regions that are not involved in current information processing (Pfurtscheller et al., 1996). While it is generally agreed that increased alpha is related to decreased cortical activity (Goldman et al., 2002; Laufs et al., 2003a, 2003b), technological advances in neurophysiology provide extant evidence for the functionality of the alpha rhythm, suggesting its important role in a variety of cognitive processes.

One of the most intriguing aspects of alpha rhythm's functioning is that it operates either through power (i.e., amplitude) increase or through power decrease. Klimesch et al. (2007) suggested that task-related increase in alpha power reflects top-down, inhibitory control of task irrelevant processing, while a decrease in alpha power (often termed "alpha suppression") reflects a release of functional inhibition (Klimesch et al., 2007). Others have suggested that increased alpha may reflect active (memory-related) processing (Palva and Palva, 2007). Jensen and Mazaheri, (2010) further outlined the inhibitory property of alpha oscillations in their perspective on "gating by inhibition", according to which information is routed to task-relevant brain regions by functionally blocking-off activity (through alpha power enhancement) in task-irrelevant regions. Moreover, they suggest that such alpha-mediated inhibition is required for optimal task performance. Recently, this proposal received neurophysiological support in animal research (Haegens et al., 2011). In their inquiry into the role of cortical oscillations in sustained attention, Clayton et al. (2015) proposed that in addition to the role of alpha oscillations in local cortical inhibition of task-irrelevant processes, an increase in global alpha power during tasks that require sustained attention may reflect coordinated activity in frontal and posterior brain regions associated with cognitive control. This proposal was based on studies linking global alpha power to activity in networks associated with task-related processing and sustained attention.

The role of oscillatory activity in cognitive processes has also been studied by simultaneous recording of EEG and fMRI activity (Chang et al., 2013; Sadaghiani et al., 2010; Zumer et al., 2014). For instance, Donner and Siegel (2011) systematically examined task-related oscillatory behavior and the relation between oscillatory patterns and BOLD-fMRI signal. They suggested that the link between neural oscillations and BOLD signal is not fixed, but rather process-dependent. Specifically, while the activation of local cortical regions (e.g., activation involved in the encoding of sensory information) is typically associated with decreased low-frequency activity (including alpha rhythms), thus yielding a negative correlation with BOLD signal, cortical interactions among distant brain regions, which mediate integrative functions (e.g., top-down control, decision-making), typically enhance low frequency oscillations, yielding a positive correlation with BOLD (Donner and Siegel, 2011). Together, these observations and theories reveal a complex picture of alpha functionality, which awaits further elucidation, potentially by integrating insights from research on healthy and clinical populations as well as animals.

1.2.2.1. Alpha oscillations and social processes. Cumulative evidence in social neuroscience suggests that alpha rhythm plays a key role in supporting social functioning, including the perception of biological motion (Ulloa and Pineda, 2007) and inter-individual synchronized actions (Dumas et al., 2010). We recently found that alpha rhythm is enhanced by stimuli probing social synchrony in brain regions associated with social perception and theory of mind, including the posterior superior temporal sulcus and the inferior frontal gyrus (Levy et al., 2016). Moreover, we found that oxytocin, a neuropeptide involved in social information processing, interacts with alpha activity to increase salience to specific features of individuals and contexts. This finding is congruent with previous research, indicating that oxytocin impacts alpha oscillations in the mirror neurons network (Perry et al., 2010b) and that its effects are context-bound and shaped by powerful social experiences (Bartz et al., 2011).

Alpha activity was also shown to be involved in the perceptual processing of others' pain – the most ancient precursor of empathy that indexes the ability to increase emotional arousal and

resonate with the distress of conspecifics (Decety, 2011). A suppression of alpha power has been repeatedly demonstrated in response to observed pain in brain regions associated with first-hand experience of pain, including regions of the sensorimotor cortex (Cheng et al., 2008; Whitmarsh et al., 2011), where alpha oscillations are termed 'mu' oscillations (Kuhlman, 1978), and regions involved in social information processing, such as the superior temporal gyrus (Eidelman-Rothman et al., 2016; In Press). Such response has been shown to be affected by previous pain-related experiences in individuals exhibiting psychopathological symptoms (Eidelman-Rothman et al., 2016; In Press). Additionally, assessing patients in vegetative and minimally conscious states it was found that alpha oscillations are related in a quantitative manner to the level of consciousness expression (Babiloni et al., 2009; Fingelkurts et al., 2012).

Affective disorders, as well as obsessive compulsive and post-traumatic stress disorders, are characterized by cognitive and social impairment, which may reflect or even originate from abnormalities in alpha functioning. In light of the important role that alpha rhythm plays in both cognitive and social processing, pointing out such a plausible bio-psychiatric link could help elucidate the mechanisms underlying these disorders (Fingelkurts and Fingelkurts, 2010).

1.2.2.2. Physiological origins of the alpha rhythm. Despite scarce evidence on the neurophysiological origins of the alpha rhythm, it is generally assumed that alpha rhythm may stem from rhythmic interneuronal GABAergic feedback (Lorincz et al., 2009). Such feedback may exert alpha-mediated functional inhibition by directly silencing processing in pyramidal neurons or reducing the efficacy of excitatory input (Mann and Paulsen, 2007). Furthermore, thalamic rhythms generators may have an important role in modulating these GABAergic-generated alpha rhythms and their interactions among cortical areas (Saalmann et al., 2012).

Although studied to a much lesser extent in animal models, the robustness of alpha and its functionality in information processing and cognitive and attention-related processes have been demonstrated in other mammalian species, including non-human primates (Bollimunta et al., 2008; Haegens et al., 2011), dogs (Dumenko, 1995), cats (Chatila et al., 1992; Schürmann et al., 1998) and rats (Broussard and Givens, 2010; Wiest and Nicolelis, 2003), highlighting the evolutionary-ancient and conserved nature of the alpha rhythm. Moreover, evidence suggests an increase in alpha activity during the evolution of species (Başar, 2012; Jurko et al., 1974). Importantly, in humans, neural oscillations in general, and the alpha rhythm in particular, change substantially across development from infancy to late adolescence (Niedermeyer, 1997; Uhlhaas et al., 2009). Such changes in alpha activity have been shown to stabilize towards early adulthood and to reappear again in the elderly (Başar, 2012; Klimesch, 1999). Thus, as is the case with other frequencies (Buzsáki et al., 2013), stability of alpha rhythmic patterns is characteristic of the adult brain.

1.2.2.3. Alpha and other neural rhythms. Neural oscillations are typically analyzed in standard frequency ranges, including delta (~2–4 Hz), theta (~4–8 Hz), alpha (~8–12 Hz), beta (~12–30 Hz) and gamma (~30–100 Hz), which often occur simultaneously in the same brain state and interact with each other (Buzsáki et al., 2013; Fingelkurts et al., 2014Fingelkurts and Fingelkurts, 2014; Jensen and Colgin, 2007). In addition to the investigation of activity in a specific frequency, research into brain oscillations and cognitive processing often examine the relationship between the different frequency bands. Such research showed, for example, that decreased alpha activity during task performance is often accompanied by increased activity in the gamma band (Jerbi et al., 2009), which is thought to reflect neuronal processing (Crone et al., 2006). In addition, the ratio between theta and alpha power has been associated with attention levels (Borghini et al., 2014) and has been used as an index for attention deficits (Clarke et al., 2001; Veltmeyer et al., 2006). Studying the interplay between oscillatory activity in different frequency bands and the mechanisms by which they interact is important for understanding the role of neural oscillations in normal and disturbed information processing. This topic however, is beyond the scope of the current review.

1.2.3. Measures of alpha activity

Oscillatory activity is commonly assessed using spectral analysis methods, in which the recorded EEG and MEG signal is transformed from the temporal domain to the frequency domain by analytical techniques such as Fourier transform. The resulting power-spectrum quantifies the power of oscillatory activity in the different frequency bands (Le Van Quyen and Bragin, 2007). This measure is often referred to as quantitative EEG in the EEG literature, and has advanced the study of brain rhythms, and the characterization of the alpha rhythm in particular (Bazanova and Vernon, 2014), over the past decades.

The power of neural oscillations has been shown to be modulated by extrinsic (e.g., external stimuli) as well as intrinsic (e.g., internal mental processing) events (Cooper et al., 2003; Pfurtscheller and Lopes da Silva, 1999). As mentioned above, such modulations may include decrease or increase in power measured relative to a reference period, and are often referred to as *event-related desynchronization (ERD)* or *event-related synchronization (ERS)*, respectively (Pfurtscheller and Lopes da Silva, 1999 and see Fig. 1A). These measures reflect (de-) synchronization in the underlying neural populations (Neuper and Pfurtscheller, 2001). ERD and ERS in the alpha range have been shown to correlate with a wide range of cognitive and perceptive processes, including tactile (Gaetz and Cheyne, 2006), visual (Pfurtscheller et al., 1994) and auditory (Potes et al., 2014) processing, memory (Jensen et al., 2002; Krause et al., 2000), attention (Mazaheri et al., 2014), emotional (Onoda et al., 2007) and socio-affective stimuli processing (Perry et al., 2010a). Importantly, differences in ERD and ERS patterns have been demonstrated for different frequency bands within the alpha range. Specifically, ERD measured around 7–10 Hz, often referred to as *lower alpha or alpha 1 sub-band*, has been shown to be related to non-specific, general attentional demands including increased alertness and expectancy, while ERD measured at ~10–13 Hz, known as *upper alpha or alpha 2 sub-band*, has been related to task specific, semantic processing demands (see Klimesch, 1999 and Fig. 1B). It has been demonstrated specifically for the alpha 2 sub-band that ERD in task-related regions occurs along with ERS in task irrelevant regions (Neuper and Pfurtscheller, 2001). In addition, activity in the alpha 1 sub-band has been shown to be more strongly related to cortical activity, as revealed by simultaneous EEG and positron emission tomography (PET) recordings (Oakes et al., 2004). Thus, the modulation of alpha power is not a uniform phenomenon (Bazanova and Vernon, 2014; Klimesch et al., 2007), and specifically, the functionality of ERD in the alpha range, which is generally thought to reflect active information processing, differs across frequency ranges (Klimesch et al., 2007). In addition to its event-related modulations, pre-stimulus alpha, i.e., the alpha power measured prior to the beginning of an event, is also often assessed. It is well established that higher pre-stimulus alpha is associated with greater task-related ERD (Başar, 2012) and with enhanced cognitive performance (Klimesch, 1999) and it has been suggested to reflect a state of cognitive readiness when preparing for an upcoming task (Min and Herrmann, 2007).

Another commonly used measure of alpha power is *inter-hemispheric asymmetry*, which refers to differences in left-right brain activity (Fig. 1C). Alpha asymmetry, particularly over frontal regions, has been widely examined in research assessing individ-

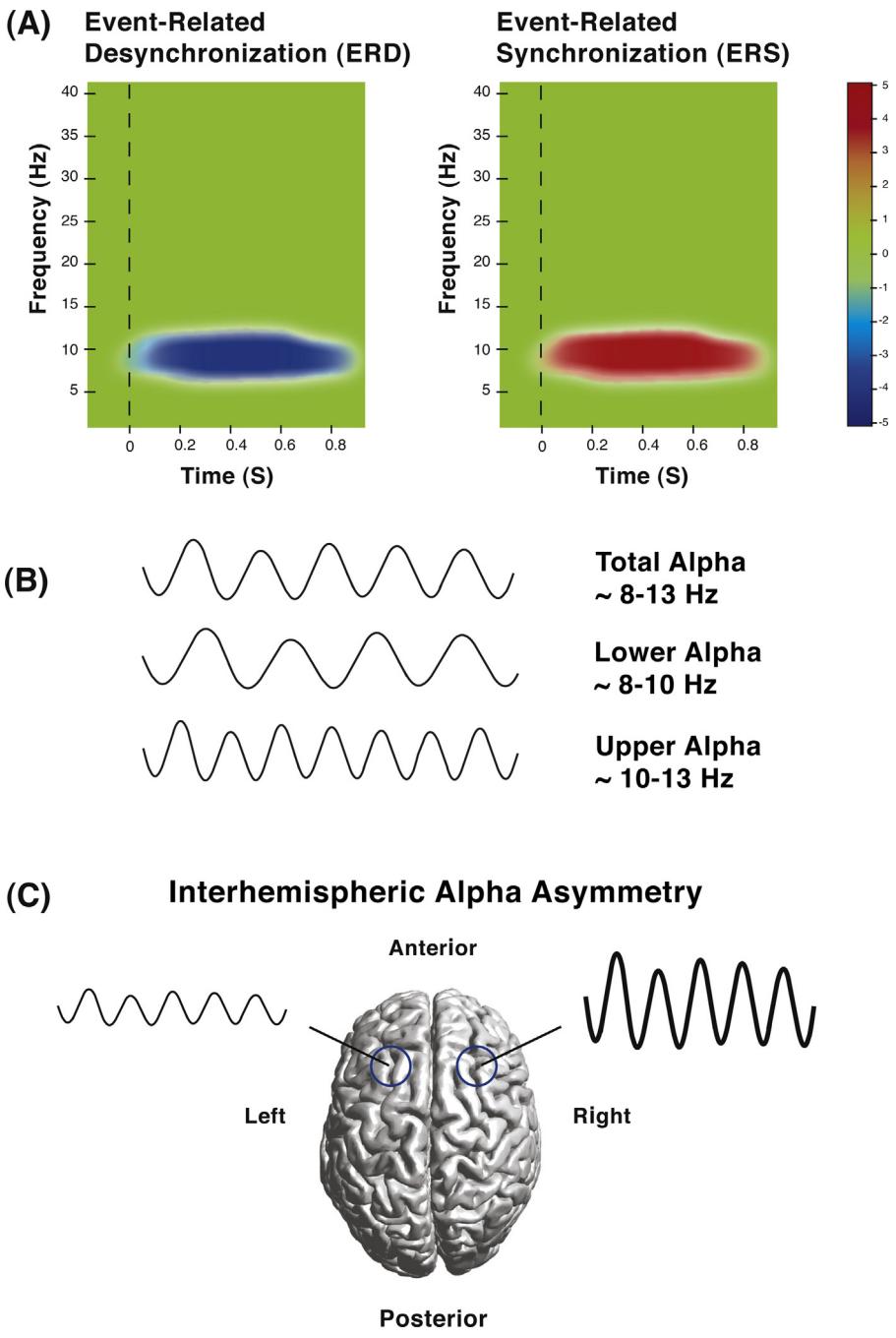

Fig. 1. Measures of Alpha Activity. (A) Time-frequency representation of oscillatory activity, demonstrating event-related desynchronization (ERD; left panel, blue shades) and event-related synchronization (ERS; right panel, red shades) in the alpha frequency band, which occurred following stimulus presentation at time zero (marked with a dashed line). (B) Total, lower and upper alpha frequency bands. (C) Illustration of one case of interhemispheric right alpha asymmetry in frontal regions. The illustration shows higher amplitude (higher activity) in the right compared to the left hemisphere. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

ual differences in personality, emotion and affective style (Coan and Allen, 2004) and has been typically interpreted in the context of theoretical models (Davidson, 1998; Heller et al., 1995; Keller et al., 2000) suggesting an association between such individual characteristics and specific patterns of alpha asymmetry (See Section 2.1 for more details). The common interpretation of alpha activity in alpha asymmetry research relies on the assumption of inversed relations between alpha power and cortical activity, such that frontal asymmetry is thought to reflect lateralization of brain activity (Meyer et al., 2015). Alpha asymmetry has been suggested to be a stable, trait-like feature (Allen et al., 2004; Jacobs and Snyder, 1996; Thibodeau et al., 2006) and was extensively examined as a biomarker for different psychiatric conditions, some of which will be presented in the following sections.

Studies assessing alpha activity often differ in their definition of the alpha range under investigation, be it a broad alpha range or alpha sub-bands. In addition, while many studies examined alpha activity in a fixed, predefined frequency range, a more recent approach includes the calculation of individualized alpha bandwidths. This approach was suggested to be advantageous over the use of a fixed alpha range (Bazanova and Vernon, 2014), since it takes into account the individual's alpha characteristics, known to

be affected by different factors such as genotype (Van Beijsterveldt and Van Baal, 2002) and age (see Section 1.2.2.2 above), and may therefore increase experimental sensitivity. Individualized alpha range is commonly determined based on the individual's alpha peak frequency (see Klimesch, 1999), among other methods (see Bazanova and Vernon, 2014), where alpha peak frequency refers to the alpha frequency bin within the alpha frequency range, showing the largest power.

Much of the research employing the aforementioned measures, including alpha ERD/ERS and asymmetry, was conducted using EEG, whereas MEG has been used much less frequently. Both EEG and MEG signals provide a direct measure of neuronal activity and are considered complementary measures (Vrba and Robinson, 2001). However, unlike the electric fields which are distorted by the changes of electric conductivity between the brain, skull, and scalp, the magnetic field measured by MEG can pass unaffected through these structures, allowing for a reliable identification of the underlying signal generators (Hari and Salmelin, 2012; Luck, 2014). As such, MEG – uniquely combining high spatial with accurate temporal resolution – may provide valuable information regarding the behavior and dynamics of alpha activity across brain regions, which may shed further light on alpha functionality in healthy and pathological populations.

1.2.4. The current review

Over the last decades, links between psychiatric conditions and abnormal oscillatory activity have been repeatedly demonstrated (Uhlhaas and Singer, 2012, 2006). Specifically, a considerable amount of evidence has been accumulated for abnormalities in activity measured in the alpha frequency band, both in association with tasks assessing cognitive and affective processes and during a resting state (Başar and Güntekin, 2012; Buzsáki and Watson, 2012; Fingelkurts and Fingelkurts, 2010). Resting state brain activity, measured in the absent of task demands is presumed to reflect internal brain dynamics and has been increasingly studied in both healthy and a wide range of clinical populations (Buckner and Vincent, 2007; Fingelkurts and Fingelkurts, 2014; Gusnard and Raichle, 2001; Woodward and Cascio, 2015).

In this review we explore oscillatory alpha activity in affective disorders, post-traumatic stress disorder (PTSD), and obsessive compulsive disorder (OCD), and seek to provide a better understanding as to what can be learned from abnormalities in alpha activity on the distinct profiles of brain functioning in each psychiatric condition. We include studies assessing resting state and those employing specific experimental tasks.

We chose to focus our inquiry on the alpha frequency band, specifically, on the well-characterized measures of alpha power mentioned above (Section 1.2.3) for which extensive research has been conducted in healthy and psychiatric populations. As previously described, the alpha rhythm is considered to have a central and important role in perceptual and cognitive processing and its disturbances may lead to abnormalities as those seen in psychiatric conditions. Further, the alpha band has been shown to have high test-retest reliability (Salinsky et al., 1991) and is therefore considered a stable individual trait (Başar, 2012) that may be potentially used as a neuromarker for clinical disorders and aid diagnosis. Finally, alpha activity has been shown to be amenable to therapeutic intervention (Kluetsch et al., 2013; Peeters et al., 2014; Ros et al., 2014), thus better understanding its disturbances in psychiatric conditions may help improve therapeutic strategies. Since developmental changes in alpha rhythm stabilize during early adulthood and reoccur in the elderly (see Section 1.2.2.2), we limited our review to studies conducted on the adult brain. It should be noted that some of the reviewed studies measured activity in other frequency ranges as well; however, we will present and discuss only findings in the alpha band.

We focus the review on the aforementioned disorders for which sufficient amount of work assessing alpha power has been accumulated. We included PTSD and OCD, which were included until recently under the umbrella of anxiety disorders (American Psychiatric Association, 1994). This has been modified in DSM-V, where these disorders fall under different categories (American Psychiatric Association, 2013). Nonetheless, as these disorders share common features with affective disorders, particularly anxiety and depression, including them in this review may provide a more comprehensive picture. Empirical findings from studies assessing alpha power will be presented and discussed followed by some methodological considerations and conclusions for future research. In some, but not in all of the studies, clinical participants showed comorbidity with other disorders and/or were receiving medication. When reviewing these studies, the presence of comorbidity or use of medication will be explicitly mentioned, otherwise, it can be assumed that participants did not have comorbid disorders and were not medicated.

2. Alpha power in affective and post-traumatic stress disorders: empirical findings

2.1. Depression

Major depression disorder (MDD) is characterized by dysphoric and irritable mood, rumination and self-referential thinking, anhedonia, a loss of motivation and interest in daily activities and impaired functioning in the social and occupational domains (American Psychiatric Association, 1994). MDD has also been shown to be associated with cognitive deficits, including impaired memory and concentration (Marazziti et al., 2010; Ravnkilde et al., 2002).

Abnormalities in alpha activity have been repeatedly demonstrated in depressed individuals. Extant research has focused on individual differences in EEG alpha asymmetry patterns, following Davidson's conceptual model which suggested that individual differences in asymmetry patterns may be associated with a tendency towards certain affective styles and may be related to the individual's susceptibility to develop depression (Davidson, 1998; Fingelkurts and Fingelkurts, 2015; Thibodeau et al., 2006). Specifically, it has been suggested that relatively higher left compared to right frontal activity is associated with behavioral approach whereas relatively higher right than left frontal activity is related to behavioral withdrawal (Coan and Allen, 2004; Davidson et al., 1990). As such, individuals showing decreased left frontal activity or enhanced right frontal activity are more likely to experience feelings of sadness and anhedonia or to exhibit behavioral inhibition and withdrawal (Sutton and Davidson, 1997), all of which are known to be associated with depression, in addition to other psychiatric conditions. In addition to the frontal asymmetry patterns suggested by this model – often referred to as the approach-withdrawal model – depression has been linked to alterations in posterior asymmetry (Henriques and Davidson, 1990). Specifically, it has been hypothesized by the valance-arousal model that depression would be related to decreased activity in right posterior regions, involved in the modulation of behavioral arousal (Heller, 1993). Research findings and methodological considerations related to alpha symmetry and depression have been previously reviewed (Rotenberg, 2008, 2004) and a recent review has similarly addressed these topics (Jesulola et al., 2015). Thus, some research on alpha asymmetry will be reviewed and summarized in this section along with other findings on depression-related alpha abnormalities.

2.1.1. Resting state studies

Evidence from several resting state EEG studies provide support for the hypothesized association between depressions and relatively lower right, or greater left frontal alpha power, which was interpreted as increased right or decreased left cortical activity. Increased left-sided anterior, as well as enhanced right posterior alpha (8–13 Hz) activity was found in 6 individuals with a history of depression, compared with 8 healthy, never-depressed controls (Henriques and Davidson, 1990). Similarly, left frontal asymmetry was observed in 15 depressed individuals (men and women) compared with 13 matched controls; however, no differences in posterior asymmetry were found (Henriques and Davidson, 1991). Some of the patients in the latter sample were medicated; yet, the results were not affected by medication status.

Results from other studies demonstrating similar alterations in resting state alpha (8–13 Hz) asymmetry include decreased right relative to left frontal alpha activity in a group of 6 individuals with MDD (mixed for gender), but not in 7 healthy matched controls (Putnam and McSweeney, 2008); increased left relative to right frontal alpha in 15 men and women with MDD compared to 15 controls (Kemp et al., 2010) and in 20 medicated women with recurrent MDD compared with 40 healthy controls (Chang et al., 2012).

Such asymmetry patterns were also demonstrated for alpha sub-bands, for example, increased left relative to right frontal alpha 1 (8–10.5 Hz) and alpha 2 (10.5–12.5 Hz) activity in 20 medicated males and females with MDD compared with 19 healthy controls (Cantisani et al., 2015), and increased left relative to right mid-frontal alpha 2 (10.5–13 Hz) activity (but not alpha 1(8–10.5 Hz) or total alpha (8–13 Hz) activity) in a mixed gender group of 53 individuals with MDD (some with comorbid anxiety), compared to 43 healthy controls (Jaworska et al., 2012).

Two of these studies (Jaworska et al., 2012; Kemp et al., 2010) also examined group differences in alpha asymmetry in posterior regions (parieto-temporal and parietal, respectively), however, no significant group differences were found. In addition, both studies examined alterations in alpha power and found enhanced power in individuals with MDD. Specifically, Kemp et al. (2010) found increased global alpha (8–13 Hz) power and Jaworska et al. (2012) found increased alpha in frontal and parietal regions, including the alpha 1 (8–10.5 Hz) alpha 2 and total alpha (8–13 Hz) ranges. The finding of depression-related enhanced resting state alpha power is consistent with previous research: Fingelkurts et al. (2006) found increased occipital and parietal alpha (9–13 Hz) power in a mixed gender group of 12 MDD patients (some with comorbid panic attacks and anxiety disorder) compared to 10 healthy controls. Increased alpha (7.5–14 Hz) power was also reported in right posterior temporal-occipital regions in 111 individuals with depressive symptoms (diagnosed with MDD and other conditions such as dysthymia, some were medicated) compared to 526 non-depressed controls (Grin-Yatsenko et al., 2010).

While all the above mentioned alpha asymmetry studies were conducted during rest, it has been suggested that alpha asymmetry patterns may be different when measured in a specific emotional context and during a resting state. This concept has been outlined by Coan et al. (2006), who proposed "the capability model of frontal EEG asymmetry", according to which individual differences may be more pronounced during emotional activation than during rest. In support, Stewart et al. (2014) compared frontal alpha (8–13 Hz) asymmetry scores during a resting state and during the performance of emotional task, the Directed Facial Action task (Coan et al., 2001), in which participants are asked to perform facial emotional expressions of approach or withdrawal valance. Participants were 143 depressed males and females with current or past MDD and 163 healthy controls. Depressed individuals showed relative higher left than right frontal alpha activity during the approach and withdrawal conditions but not during rest. Importantly, when an EEG reference-free measure was applied – current source density analysis – alpha asymmetry was significantly different in MDD participants across all conditions; approach, withdrawal, and rest. The authors suggest that current source density analysis may provide a more robust indication of the individual's stable trait frontal EEG asymmetry, possibly as it reflects predominantly frontal sources (Hagemann, 2004; Kayser and Tenke, 2006).

In contrast to the aforementioned research, several studies failed to find associations between frontal alpha asymmetry patterns and depression. Bruder et al. (1997) studied 44 males and females with MDD and 26 healthy controls. Of the MDD patients, 19 had comorbidity with various anxiety disorders. While depressed patients with anxiety showed decreased alpha (7.8–12.5 Hz) in right compared to left anterior and posterior sites, depressed patients without anxiety disorder did not differ from controls in anterior alpha asymmetry. However, they showed different posterior asymmetry with increased alpha power in right relative to left posterior sites compared to controls, which was suggested to reflect hypo-activity in right parietal regions, as previously reported in depressed individuals. A lack of frontal alpha (8–13 Hz) asymmetry was similarly demonstrated in a group of 52 males and females with subclinical depression, but also in a matched group of 52 subclinically depressed individuals with comorbid anxiety, compared to 52 healthy controls (Mathersul et al., 2008). In addition, unlike Bruder et al. (1997) no differences in posterior (parieto-temporal) alpha asymmetry patterns were found in depressed individuals with and without comorbidity, relative to controls. When assessing alterations in alpha power, however, both groups of depressed individuals showed decreased alpha power in bilateral frontal and right parieto-temporal regions, the latter associated with higher levels of depression. It was consequently suggested that higher right parieto-temporal cortical activity may be associated with higher levels of negative mood. Segrave et al. (2011) investigated whether examination of individualized alpha bandwidths and alpha sub-bands (see Section 1.2.3) increases the sensitivity of the anterior alpha asymmetry measure in MDD. They tested alpha asymmetry in individualized lower and upper alpha frequency ranges, as well as in a fixed and broad alpha range (8–13 Hz), but found no differences in alpha asymmetry between 16 female with MDD and 18 healthy controls. Similarly, Gold et al. (2013) found no difference in frontal alpha (8–12 Hz) asymmetry measured in 79 depressed individuals compared to normative population database of 678 individuals. Importantly, most participants in the study were females, had comorbid anxiety, and used medication.

2.1.2. Task-related studies

A number of studies employed working memory (WM) tasks to assess alpha abnormalities in depression during active cognitive processing. These studies have focused on the parieto-occipital cortex, in which a significant WM-related modulation of alpha activity has been observed (Jensen et al., 2002; Michels et al., 2008). Segrave et al. (2010) recorded EEG from 15 females with MDD (some medicated), and 15 healthy matched controls while performing a WM task (modified Sternberg WM task; Sternberg, 1966) were participants need to judge, following a retention period, whether a certain letter was presented as part of a letter sequence that was previously presented. ERS was calculated for individualized upper alpha bandwidth (see Section 1.2.3) during the retention phase, in which WM load is high. MDD participants did not differ from controls in task performance, however they displayed greater alpha ERS than controls over left parieto-occipital cortex. This was suggested to reflect an increased inhibition of competing, irrelevant material during WM processing in depressed subjects, or a compensatory mechanism in which additional neural resources are invested in order to achieve performance accuracy. In a later

study by the same group, ERD in individualized lower alpha 1 frequency band was measured during the retention phase of a similar WM task, which additionally included distracting emotional stimuli (positive, negative and neutral facial expression), as background images (Segrave et al., 2012). Participants were 15 women with MDD and 16 healthy matched controls. MDD and controls did not differ in task performance. However, control subjects showed greater ERD over the posterior parietal cortex when positive distracters were presented, whereas depressed participants displayed greater ERD during the presentation of negative distracters, with no differences between medicated and unmediated MDD participants. Considering the association between alpha 1 activity and non-specific attention, results were suggested to reflect an attentional bias for positive stimuli in healthy controls which is attenuated in depressed individuals. Contrary to the findings by Segrave et al. (2010), Bailey et al. (2014) found WM performance impairments and decreased parietal-occipital upper alpha (10–12.5 Hz) ERS during the retention phase of a modified Sternberg task, in 17 men and women with MDD (some medicated and some with comorbid anxiety) and 31 healthy controls. Following the association between parietal-occipital upper alpha activity and inhibition of non-relevant information (Jensen et al., 2002), the authors proposed the results to suggest difficulty in inhibiting negative ruminations during task performance in depressed individuals.

2.1.3. Summary epression

The reviewed literature demonstrates depression-related abnormalities in alpha activity, tested mainly during a resting state and to a lesser extent, during task performance. Resting-state studies show that frontal alpha asymmetry patterns found in depressed individuals generally support the suggested link between depression and enhanced left or attenuated right alpha power (i.e., increased right, or decreased left, frontal activity; Cantisani et al., 2015; Chang et al., 2012; Henriques and Davidson, 1991, 1990; Jaworska et al., 2012; Kemp et al., 2010; Putnam and McSweeney, 2008) although, some studies failed to find such an association (Bruder et al., 1997; Gold et al., 2013; Mathersul et al., 2008; Segrave et al., 2011). Regarding posterior alpha asymmetry, some studies provided support for the suggested association between increased right posterior alpha (i.e., decreased cortical activity) and depression (Bruder et al., 1997; Henriques and Davidson, 1990) while others did not (Jaworska et al., 2012; Kemp et al., 2010; Mathersul et al., 2008). In addition to alpha asymmetry abnormalities, depressed individuals demonstrated enhancement of resting state alpha power in anterior and posterior regions (Fingelkurts et al., 2006; Grin-Yatsenko et al., 2010; Jaworska et al., 2012; Kemp et al., 2010). Finally, two studies included an examination of alpha patterns in depressed individuals with and without anxiety comorbidity. In one study, different patterns of alpha activity were found in each group compared with controls (Bruder et al., 1997) however, in the other study no such differences were observed (Mathersul et al., 2008). Studies employing WM tasks have pointed at alpha activity alterations in MDD, including greater ERD in response to negative distractors (Segrave et al., 2012) and greater parieto-occipital ERS during memory retention (Segrave et al., 2010), although the opposite trend of alpha ERS was also reported (Bailey et al., 2014).

2.2. Bipolar disorder

Bipolar disorder (BD), or manic-depression illness, is characterized by extreme variations in mood and motivation. It is distinct from unipolar disorder (depression) described above by the occurrence of manic episodes, characterized by increased arousal and energy, and by high-risk, pleasure seeking impulsive behavior (American Psychiatric Association, 1994; Belmaker, 2004). There are different subtypes of BD, including bipolar disorder 1, bipolar disorder 2 and cyclothymic disorder, all involve a certain degree of mania and depression episodes (Hirschfeld et al., 2002). BD is associated with cognitive processing deficits (Clark et al., 2002; Martínez-Arán et al., 2004; Robinson et al., 2006), including deficits in attention, memory and emotional processing. Although studied to a much lesser extent than unipolar depression, research has shown that BD is associated with disturbed resting state as well as task-related alpha activity anomalies (see also in Degabriele and Lagopoulos, 2009).

2.2.1. Resting state studies

Resting state studies have mostly yielded inconsistent results related to alpha asymmetry and alpha power alterations. Frontal alpha asymmetry was tested in 4 bipolar women compared with 4 matched controls and greater left compared to right alpha (8–13 Hz) power was found among bipolar patients (Allen et al., 1993). In a larger study, Clementz et al. (1994) found decreased resting state EEG alpha (8–13 Hz) across central recording sites and decreased right sided alpha, in a group of 31 first episode bipolar patients with psychotic characteristics, compared with 113 healthy controls, with some patients being medicated but no medication effect. El-Badri et al. (2001) demonstrated EEG impairments in bipolar patients currently in their euthymic phase. Specifically, the authors found a widely distributed increase in resting state EEG alpha (8–14 Hz) in a mixed-gender group of 29 euthymic patients with bipolar 1 disorder and 26 healthy controls, again with no effect of medication. Başar et al. (2012) studied EEG alpha (8–13 Hz) activity during rest in 18 euthymic bipolar patients (including patients with bipolar 1 and bipolar 2 disorders), who showed reduced alpha power in bilateral occipital regions compared to 18 healthy matched controls. In this study, no lateralization in alpha asymmetry power was found.

2.2.2. Task-related studies

Differences in alpha were also found during task performance in bipolar patients. Harmon-Jones et al. (2008) studied a group of 41 patients, including bipolar and cyclothymic disorders compared to 53 healthy matched controls, with some patients medicated and some showing comorbidity with anxiety disorders, substance use, and eating disorder. EEG was measured while participants were preparing to perform a cognitive task (anagrams of scrambled words) in various degree of difficulty. In order to examine responses to elicited motivation, participants were informed that potential reward or punishment will be given depending on the solution. Compared with controls, individuals in the bipolar group showed higher right relative to left frontal alpha (8–13 Hz) power only in preparation to the hard task with a potential reward, but not a potential punishment. Such alpha asymmetry pattern was related to self-report behavioral activation. Lee et al. (2010) investigated oscillatory activity patterns in bipolar disorder vs. unipolar depressive disorder using MEG. Three groups of men and women where included: 20 MDD patients, 20 medicated BD patients, and 20 healthy controls. During the MEG scan, participants performed an implicit emotional task in which they had to judge the gender of images displaying different facial expressions. Brain responses to angry expression, which were high in emotional salience and arousal, were compared. Bipolar and MDD patients showed distinct alterations in alpha (8–12 Hz) activity compared with controls in several brain regions. BD was associated with increased alpha in regions related to affective evaluation and emotion detection, including the right inferior frontal gyrus and left insula, which was suggested to reflect increased sensitivity to emotionally salient information. MDD patients showed increased as well as decreased alpha compared to controls in different sets of brain regions, including the left superior temporal gyrus, cuneus, and right precentral

gyrus (increase) and the right middle temporal gyrus (decrease). The results were suggested to indicate that bipolar and unipolar disorders are associated with distinct neuropathological patterns.

2.2.3. Summary bipolar disorder

Alpha alterations were found in different bipolar populations and different phases of the disorder (e.g., euthymia, mania), mostly during rest. Results suggest decreased (Başar et al., 2012; Clementz et al., 1994) but also increased alpha power (El-Badri et al., 2001) in BD, with the latter also being evident during task performance (Lee et al., 2010). Some demonstrated resting state alpha asymmetry with greater left relative to right alpha (Allen et al., 1993) but others did not (Başar et al., 2012). The direction of alpha asymmetry during task performance was opposite to the one observed during rest (Allen et al., 1993), namely, greater right relative to left activity (Harmon-Jones et al., 2008).

2.3. Anxiety disorders: general anxiety, social anxiety, and panic disorder

Anxiety is characterized by excessive and often uncontrolled feeling of worry and fear which affect occupational, interpersonal and emotional domains. The disorder is associated with physical and cognitive symptoms, such as restlessness and impaired concentration (American Psychiatric Association, 1994), and with behavioral inhibition and increase in attention (Eysenck et al., 2007). Social anxiety (or social phobia) is specifically characterized by extreme fear and avoidance of social and performance situations. Panic disorder (PD) is characterized by sudden and repeated episodes of intense fear (panic attacks) followed by relatively prolonged periods of anticipatory anxiety (American Psychiatric Association, 1994). Alpha abnormalities have been demonstrated in the different types of anxiety disorders, during rest and during the provocation of anxiety.

2.3.1. General anxiety

Several studies have pointed at association between general anxiety and increased EEG alpha power. Saletu-Zyhlarz et al. (1997) found higher resting state alpha in 44 patients with generalized anxiety disorder (GAD) compared with 44 controls. A later series of studies conducted by Knyazev et al., with non-clinical anxious participants showed associations between increased resting state alpha power and anxiety (Knyazev et al., 2003, 2002), which was enhanced by the exposure to experimentally induced anxiety (Knyazev et al., 2005, 2004). For example, in a group of 30 male participants, EEG data was examined in several experimental conditions, including resting state and presentation of an unexpected loud alarm sound (Knyazev et al., 2004). Individually adjusted lower and upper alpha sub-bands (see Section 1.2.3) in a range of 7.5–12.5 Hz, were analyzed. In both experimental conditions, higher alpha power across all cortical sites was associated with higher levels of state and trait anxiety. When inspecting differences between low and high anxious individuals, the most pronounced difference was associated with the unexpected arousing alarm sound, where low-anxiety individuals responded with enhanced upper alpha and high anxiety individuals with enhanced lower alpha power. Importantly, these responses were recorded a number of minutes after the provoking event, probably following an event related desynchronization. Based on the specific functions attributed to the lower and upper alpha sub-bands (Klimesch, 1999) these results were suggested to reflect an increase in specific attention in low anxious individuals compared with increased unspecific attention in high anxious individuals. Overall, the enhanced alpha activity observed in this study was suggested to reflect higher vigilance and readiness towards anticipated threat (see Section 4 for further discussion). In a further analysis of the same sample, Knyazev et al. (2006) found that high anxious subjects had higher alpha power during periods of expectation for a perceptual event which included auditory presentation of recorded neutral words while subject remained with their eyes closed, as well as higher magnitude of alpha desynchronization in response to this event. Particularly, upper alpha ERD was most prominent in task-irrelevant posterior regions, as well as the higher pre-stimulus alpha observed in this frequency band. This was suggested to be linked with anxious visual imagery and preparedness for the processing of visual information (see Section 4 for a discussion). Based on their observation of enhanced alpha power in reference periods along with enhanced alpha ERD, the authors suggested that higher alpha power reflect higher readiness of the alpha response system, which is associated with enhance alpha reactivity (see Section 4 for further discussion).

A few studies examined anxiety-related alpha asymmetry patterns. Mathersul et al. (2008) investigated the relation between anxiety (and depression, see Section 2.1 above) and resting state frontal and parieto-temporal alpha (8–13 Hz) asymmetry in a mixed gender group of 52 sub-threshold, nonclinical participants compared with 52 healthy controls. The Depression Anxiety Stress Scales was used to subtype individuals into anxious apprehension and anxious arousal groups, hypothesized to differ on frontal and parieto-temporal activity (Heller et al., 1997). The anxious group showed enhanced left relative to right frontal alpha (8–13 Hz) activity compared to controls. Among sub-types, the anxious apprehension group showed higher right relative to left frontal alpha, while the anxious arousal group showed enhanced left relative to right frontal alpha. In addition, the anxious apprehension group had higher left relative to right parieto-temporal alpha. These asymmetry patterns were suggested to support an updated valence-arousal model (Heller and Nitscke, 1998), according to which anxious arousal is associated with higher relative right frontal activity, whereas anxious apprehension is associated with higher left frontal activity. The latter is thought to indicates increased approach tendencies (Davidson, 1998), but also heightened tendency to worry and heightened rumination, due to the association of the left hemisphere with language function (Heller et al., 1997).

Guided by the capability model (Coan et al., 2006), Crost et al. (2008) tested whether anxiety-related frontal alpha asymmetry is affected by context. One hundred and six nonclinical males with high and low-level self-reported anxiety were confronted with negative or positive personality feedback, presented in a private or a public context. In the private context, they read the self-related feedback while being alone, whereas in the public context they read the feedback in the presence of another person who could also read the same feedback. Analysis was performed for lower (8–10.25 Hz) upper (10.5–12.75 Hz) and total (8–12.75 Hz) alpha bands. Consistent with the hypothesized relations between personality and frontal asymmetry, effects of anxiety on alpha asymmetry were found only when the negative feedback was presented in public, expressed as greater relative left lower-alpha frontal alpha activity (enhanced right cortical activity) in high compared to low anxious participants. No differences in frontal asymmetry were found during rest.

2.3.2. Social anxiety

Beaton et al. (2008) studied frontal alpha (8–13 Hz) asymmetry patterns during rest and during anxiety provocation (i.e., anticipation of a public speech task) in a nonclinical mixed gender sample of 24 individuals with high social anxiety and 25 with low social anxiety. No group differences in alpha asymmetry were found during rest, and unlike the finding of Crost et al. (2008) obtained with generally anxious subjects, no effect of context on alpha asymmetry was found. However, in line with Crost et al. (2008), context-

dependent alpha asymmetry was demonstrated in an earlier study conducted with a clinical group of socially anxious individuals (Davidson et al., 2000). This study examined frontal asymmetry patterns associated with anticipatory anxiety related to public speaking, conducted with 18 individuals diagnosed with social phobia and 10 healthy controls. Phobics were screened to have a specific fear of speaking in front of small groups. The alpha asymmetry analysis was focused on the alpha 1 (8–10 Hz) sub-band, based on a previous observation by this group that affective constructs are more consistently related to low-frequency alpha power than high-frequency alpha (Goncharova and Davidson, 1995). While no group differences in asymmetry were found at rest, during a waiting period to make a public speech, social phobic individuals showed decreased right anterior-temporal and lateral-prefrontal alpha activity compared with controls. It was consequently suggested that increased right-sided cortical activity is induced by the provocation of anxiety. Finally, in a study conducted with clinical population, social anxiety-related alterations in alpha power were examined, however, no differences in resting state alpha 1 (7.6–10.4 Hz) and alpha 2 (10.6–13.0 Hz) were found between 25 patients with social phobia compared with 25 healthy matched controls (Sachs et al., 2004).

2.3.3. Panic disorder

Only a few EEG studies examined alpha disturbances in PD, utilizing resting state as well as task activation paradigms, and yielded inconsistent results. Newman et al. (1992) found a global reduction in slow (8.6–10.5 Hz) and fast (10.9–12.5 Hz) resting state alpha power in 7 patients with PD compared to 7 controls, however, no group differences in alpha response were found following an anxiogenic intervention (i.e., caffeine challenge), which induced a decrease in slow alpha in both groups. Following observations of relaxation-induced panic attacks, Knott et al. (1997) recorded EEG from 37 males and females diagnosed with PD and from 20 healthy matched controls, during a relaxation paradigm which included audio delivered muscle relaxation instructions, and during an audio recording of a neutral story. Panic-related symptoms and anxiety levels were also assessed, however, no relaxation-induced alterations in panic symptoms, anxiety and alpha (8–12 Hz) power were found in individuals with PD.

Two studies demonstrated alterations in alpha power as well as alpha asymmetry patterns in PD. In a study of 23 panic disorder patients, several of which were medicated and had comorbid GAD and social phobia, and in 25 controls, frontal and parietal activity was measured during rest and during the presentation of various visual stimuli (Wiedemann et al., 1999). During rest and when viewing panic-relevant or anxiety-related stimuli, but not during neutral stimuli, individuals with PD showed frontal alpha (8–13 Hz) asymmetry, with reduced right relative to left frontal alpha. This was suggested to indicate an enhanced activation of the avoidance-withdrawal system in situations associated with negative valance and that individuals with PD experience rest periods as more aversive and anxiety inducing. These results also support the concept of context-dependent personality-related alpha asymmetry. Additional findings showed lower parietal alpha in response to panic-related vs. neutral pictures in individuals with PD, but not in controls. More recently, resting state EEG alpha asymmetry and alpha power were studied in 52 males and females with PD and comorbid disorders, including OCD, PTSD, GAD, and MDD, compared to 104 healthy controls (Wise et al., 2011). Alpha activity was analyzed in the alpha 1 (8–11 Hz) and alpha 2 (11–13 Hz) frequency ranges. Compared to controls, individuals with PD showed decreased alpha 1 activity at all recorded sites and a frontal asymmetry pattern of greater left sided alpha 1, reflecting increased right frontal activity. These findings were not impacted by medication or comorbid disorders. Based on the notion that alpha 1 desynchronization is associated with increased alertness and expectancy (Klimesch, 1996; Klimesch et al., 1998), it was suggested that patients with PD compared to healthy controls were alert during the distraction-free resting state period.

Finally, Locatelli et al. (1993) investigated resting state alpha activity in PD patients compared to controls, as well as in different subtypes of PD. No group differences in alpha 1 (8–10 Hz) or alpha 2 (10–12 Hz) power were found between a mixed gender group of 37 PD patients (some with agoraphobia) and 30 controls. However, distinct alpha 2 patterns were demonstrated in PD patients with and without depersonalization and/or derealization (PD+ and PD- respectively) following an odor stimulation task. Specifically, while PD- patients showed increased right but decreased left temporal alpha 2 during odor activation, PD+ patients showed decreased alpha 2 in both hemispheres. The odor task was used to activate temporo-limbic regions in which dysfunction has been associated with the presence of depersonalization and derealization aspects of PD, and results were suggested to demonstrate biological heterogeneity in the two PD subtypes.

2.3.4. Summary anxiety

Alterations in alpha activity are found in anxious individuals, whose nature showed variation within and across the different disorders. Consisted with the observation that GAD is underresearched (Dugas et al., 2010; MacNamara and Proudfit, 2014), we found only one study assessing alpha abnormalities in this disorder (Saletu-Zyhlarz et al., 1997). With regards to alpha power alterations, anxious individuals (GAD patients and nonclinical individuals) show enhanced alpha power (Knyazev et al., 2004, 2003, 2002; Saletu-Zyhlarz et al., 1997); no alterations were found in social phobia (Sachs et al., 2004); and decreased alpha (Newman et al., 1992; Wise et al., 2011) or no power alterations (Knott et al., 1997) were found in panic disorder. Examination of alpha asymmetry patterns generally revealed higher left relative to right frontal alpha activity (Crost et al., 2008; Davidson et al., 2000; Mathersul et al., 2008; Wiedemann et al., 1999; Wise et al., 2011), suggesting enhanced right frontal activity. However, in some cases this pattern differed by anxiety (Mathersul et al., 2008) or PD (Locatelli et al., 1993) subtype and was sensitive to the context in which asymmetry was measured (Crost et al., 2008; Davidson et al., 2000; Wiedemann et al., 1999), and in others, no alpha asymmetry was found (Beaton et al., 2008). Alpha asymmetry in posterior regions was reported in one study (Mathersul et al., 2008) and varied according to anxiety subtype.

2.4. Post-traumatic stress disorder

Post-Traumatic Stress Disorder (PTSD) may develop in individuals who were exposed to a traumatic event that involves risk to physical or emotional integrity, such as combat, natural disaster, car accident, physical assault, or sexual abuse. PTSD is characterized by the co-occurrence of four symptom clusters, including re-experiencing, avoidance, negative alterations in mood and cognition, and alterations in arousal and reactivity symptoms (American Psychiatric Association, 2013). Studies have repeatedly demonstrated dysfunction in working memory (Elzinga and Bremner, 2002), attention, and cognitive control (Banich et al., 2009) in patients with PTSD.

A number of EEG and MEG studies investigated alterations in alpha power and alpha asymmetry patterns in PTSD, mostly during rest but also during cognitive-emotional processing. Results from EEG studies, which were conducted nearly exclusively during a resting state, will be presented followed by more recent findings from MEG research on PTSD.

2.4.1. EEG studies on PTSD

Begić et al. (2001) were among the firsts to use quantitative EEG (see Section 1.2.3) to investigate oscillatory abnormalities in PTSD. They recorded resting state brain activity from 18 male veterans with combat related PTSD and 20 healthy non-veterans controls, and found no differences between groups in alpha 1 (7.5–9.5 Hz) or alpha 2 (10–13 Hz) power. A later study of the same group using a larger sample of 79 male individuals with combat-related PTSD compared to 37 combat veterans without PTSD found that PTSD veterans had decreased alpha 1 (7.5–9.5 Hz) power over frontal, central, and occipital regions, and this decrease was more pronounced over the left hemisphere, with no differences in alpha 2 activity (Jokic-begic and Begic, 2003). As activity in alpha 1 has been associated with processes related to attention, the alteration in alpha 1 was suggested to be related to the attentional deficits characteristic of PTSD (the observation that the direction of alteration was decreased power was not discussed). Similarly, Veltmeyer et al. (2006) found reduced resting state alpha 1 (8–11 Hz) power in frontal, central and parietal regions in a mixed gender group of 34 patients with PTSD due to a variety of traumatic events (not elaborated) compared to 136 trauma-unexposed controls. In addition, a regional effect was found in alpha 2 (11–13 Hz), showing a reduction in power primarily at midline cortical sites, which correlated with hyperarousal symptoms. Results were suggested to indicate increased cortical arousal in PTSD. Importantly, some of the members in the PTSD group were medicated and this reduced the decreased alpha 1(8–11 Hz) effect to trend. Finally, no significant lateralization effects were found.

More recent resting state studies failed to find PTSD-related alterations in alpha power, and, consistent with Veltmeyer et al. (2006), most studies have failed to observe alpha asymmetry differences between individuals with PTSD and controls. Imperatori et al. (2014) studied 17 men and women with PTSD due to various traumatic events (e.g., gun aggression, family murdered, rape, car accident, fire victim and physical aggression) and 17 healthy matched controls, and found no group differences in alpha (8–12.5 Hz) power. Similarly no differences in alpha power (frequency range not specified) were found between individuals with PTSD (mixed gender; some medicated) associated with the Chernobyl disaster compared to 22 healthy unexposed controls (Loganovsky and Zdanevich, 2013) and between 59 combat male veterans with PTSD and 27 veterans with no PTSD diagnosis, some of which were medicated and showed depression symptoms (Wahbeh and Oken, 2013). The latter study also did not find significant effects for frontal alpha (8–13 Hz) asymmetry. Shankman et al. (2008) studied alpha asymmetry in a mixed gender group of 32 patients with PTSD (some medicated) due to various traumatic events (mainly motor vehicle accidents, being threatened with a weapon and physical assault). A control group of 42 trauma-unexposed individuals was chosen to be maximally different from the PTSD group with respect to several personality traits (i.e., neuroticism, extraversion, stress and anxiety). This specific strategy in selecting a control group was guided by the proposition that brain activity asymmetries reflect trait-like characteristics (Davidson, 1998; Heller and Nitscke, 1998; see Sections 1.2.3 and 2.1 for theory). However, no group differences were found in alpha (8–13 Hz) asymmetry (frontal, central, or posterior) as well as no correlation between asymmetry and behavioral measures related to personality and symptom severity. Similarly, no differences in fronto-central alpha (8–13 Hz) asymmetry were found between 48 men and women with PTSD (type of trauma, comorbidity and medication status not specified) compared with normative controls (Gordon et al., 2010).

Two resting state studies found correlations between behavioral clinical measures and alpha asymmetry in PTSD, although they did not demonstrate group differences in alpha asymmetry. Specifically, Kemp et al. (2010) found no group differences in frontal or posterior alpha asymmetry (8–13 Hz) measured in a mixed gender group of 14 individuals with PTSD (cause not specified; some were medicated) and 15 controls. In addition, groups did not differ in right parieto-temporal alpha power, a region examined based on its suggested association with anxious arousal (Heller et al., 1997; see Section 2.1). However, a correlation was found between decreased right-sided alpha (indicating greater right-lateralized frontal activity) and higher PTSD symptom severity, which was retained after controlling for depression symptom severity. An earlier study examined the relationship between PTSD symptoms and alpha (8–13 Hz) asymmetry in 50 female nurse veterans of the Vietnam War with and without PTSD related to their military service (Metzger et al., 2004). Comorbidity with other psychopathologies was identified in some of the participants, including phobia and OCD, and high rates of MDD, particularly in participants with PTSD. PTSD arousal symptoms as well as depressive symptoms were associated with decreased right relative to left alpha activity in parietal regions (i.e., interpreted as greater right-sided parietal activation). This study did not assess group differences in alpha asymmetry.

Finally, Rabe et al. (2006) examined alpha asymmetry during a resting state, but also during the presentation of emotional pictures, in 43 males and females survivors of motor vehicle accidents, including 22 survivors with PTSD, 21 with sub-syndromal PTSD, and 21 without PTSD. In addition, a group of 23 healthy individuals with no history of severe accidents was included as control. Participants with PTSD and sub-syndromal PTSD showed decreased right-sided anterior and posterior alpha (8–13 Hz), which was evident only during the presentation of trauma-related pictures, but not during rest and other emotional conditions, and was correlated with PTSD symptoms. It is noteworthy that PTSD and sub-syndromal PTSD patients demonstrated comparable symptom severity in the re-experiencing, avoidance, and hyperarousal dimensions, all which were associated with the observed pattern of alpha asymmetry. Some of the participants had comorbidity with MDD, however this did not affect the results. It was consequently suggested that PTSD due to motor vehicle accidents is associated with alteration in alpha asymmetry patterns specifically in the context of traumatic stimuli, and that enhanced right cortical activation (reflected by decreased right sided alpha) may be associated with anxious arousal, as suggested previously (Heller et al., 1997).

2.4.2. MEG studies on PTSD

A few studies examined alpha activity in PTSD using MEG. Huang et al. (2014) recorded resting state activity from a group of 25 participants (24 males) with PTSD and partial PTSD due to traumatic events associated with their military activity. Thirty healthy volunteers, including active-duty military personnel as well as civilians, were included as controls. Decreased alpha (8–12 Hz) activity was found in the PTSD group compared to controls in a number of brain regions, including the dorsolateral prefrontal cortex (dlPFC), a region associated with executive functioning, and the precuneous. The latter is a part of the default-mode network (DMN), a network shown to be active during rest and to be associated with processes related to self-consciousness and self-referential thoughts (Gusnard and Raichle, 2001). In the dlPFC, the decreased alpha power was more pronounced in the right hemisphere. Decreased resting state alpha activity in the precuneous was correlated with PTSD symptom severity. In a recent study, resting state brain activity was measured in a group of 26 male veterans who were exposed to a combat-related trauma, including individuals with PTSD and partial PTSD diagnosis showing similar levels of symptom severity, and in a group of 14 trauma-unexposed healthy controls. Some of the participants had comorbidity with other psychopathologies, including depression, phobia, panic attacks and OCD. In the

trauma exposed group, increased alpha (8–13 Hz) activity was found compared to controls in regions of the left dlPFC associated with cognitive functions and working memory. The enhanced dlPFC alpha was interpreted as decreased activity in this region, and was correlated with higher number of re-experiencing symptoms (Eidelman-Rothman et al., 2015). This finding was suggested to be associated with the difficulty to suppress unwanted memories in PTSD, as was previously proposed (Anderson et al., 2004; Yan et al., 2013). In the same experimental group, alpha (8–12 Hz) ERD was examined in response to socio-affective stimuli showing others in painful vs. non-painful conditions. Compared to controls, who responded with greater ERD to painful vs. non-painful stimuli in regions of the sensorimotor cortex, posterior cingulate cortex, superior temporal gyrus and fusiform gyrus, combat-exposed individuals showed no differentiation in response in the posterior cingulate cortex, a region associated with pain perception, as well as in the detection of potential threat. This was attributed to the previous exposure of combat veterans to severe pain of others during their combat experience (Eidelman-Rothman et al., 2016; In Press). Finally, Badura-Brack et al. (2015) studied a group of 16 combat male veterans with PTSD and comorbidity with other conditions including depression and anxiety disorders, and 14 veterans without PTSD. MEG was recorded while a non-threatening tactile stimulation was applied to the participants' right hand. Group differences in alpha activity (8–14 Hz) were found in the early processing of the stimulation (0–125 ms following stimulation), in regions of the left somatosensory, the left superior parietal and the right prefrontal cortices, where the PTSD patients exhibited decreased alpha and controls exhibited increased alpha activity, relative to a baseline period. Based on previous works with healthy participants showing increased oscillatory activity in response to similar stimuli, the findings in individuals with PTSD were suggested to indicate reduced responsiveness to a non-threatening stimulation and to reflect impairments in sensory, motor and attentional processing in PTSD.

2.4.3. Summary PTSD

Most of the reviewed studies of PTSD-related alpha abnormalities employed a resting state paradigm rather than specific tasks. In most cases, an association between resting state alpha asymmetry and PTSD was not found (Gordon et al., 2010; Kemp et al., 2010; Rabe et al., 2006; Shankman et al., 2008; Veltmeyer et al., 2006; Wahbeh and Oken, 2013), although one study observed such an association when tested in a specific context (i.e., trauma-related stimuli; Rabe et al., 2006). A correlation between resting state alpha asymmetry (i.e. decreased right-sided alpha) and PTSD symptoms was also observed (Kemp et al., 2010; Metzger et al., 2004) although no group differences were demonstrated. Findings related to alterations in resting-state alpha power were less consistent, showing PTSD-associated alpha decrease (Huang et al., 2014; Jokic-begic and Begic, 2003; Veltmeyer et al., 2006) increase (Eidelman-Rothman et al., 2015) or no differences in alpha power compared to controls (Begić et al., 2001; Imperatori et al., 2014; Kemp et al., 2010; Loganovsky and Zdanevich, 2013; Wahbeh and Oken, 2013). Finally, atypical alpha ERD was demonstrated in response to sensory stimulation (Badura-Brack et al., 2015) and to socio-affective stimuli (Eidelman-Rothman et al., 2016; In Press).

2.5. Obsessive compulsive disorder

Obsessive Compulsive Disorders (OCD) is characterized by unwanted, intrusive, and distressing recurrent thoughts and impulses which are often associated with compulsive behaviors that are repetitive, time consuming, and often ritualized (American Psychiatric Association, 1994; Chamberlain et al., 2005). OCD patients usually have deficits in inhibitory regulation, as well as in cognitive functions including altered executive control and memory impairments (Olley et al., 2007).

Abnormalities in alpha activity in OCD were examined during resting state and during the performance of tasks related to memory and behavioral inhibition.

2.5.1. Resting state studies

Two resting state studies demonstrated an association between OCD and decreased alpha. Locatelli et al. (1996) examined resting state temporal alpha activity, including the alpha 1 (8–9.9 Hz) and alpha 2 (10–11.9 Hz) sub-bands, and found decreased alpha 2 (10–11.9 Hz) power in a group of 37 males and females with OCD compared with 30 healthy controls. Karadag et al. (2003) studied 32 males and females with heterogeneous characteristics of OCD (some with comorbid depression), and 31 healthy controls. Reduced alpha (8–13 Hz) was found in fronto-temporal regions in the "doubting" OCD subtype and in parieto-occipital regions in the "checking" subtype. When examined according to symptom severity, patients with higher level of symptom severity showed alpha reduction similar to the doubting subtype. In contrast, OCD patients with comorbid depression showed increased rather than decreased alpha power over several brain regions, compared to non-depressed patients. Finally, no differences in alpha asymmetry were found between OCD patients and controls. However, within the OCD group, the "doubting" subtype as well as patients with comorbid depression showed decreased left relative to right alpha activity in frontal, central and occipital sites. These results demonstrate distinct alpha alterations in patients with different OCD characteristics.

Two later studies investigated the relations between alpha power measured during a resting state, and the performance of patients with OCD in behavioral tasks associated with cognitive functioning. Bucci et al. (2004) analyzed alpha activity in the alpha 1 (7.7–9.5 Hz) and alpha 2 (9.7–12.5 Hz) frequency bands in 32 males and females with OCD and 32 healthy controls. In line with the above mentioned findings, OCD patients showed decreased alpha 1 power compared to controls in anterior and posterior regions, which was retained also after the exclusion of OCD patients with high depressive symptoms. Reduced alpha 1 power, particularly in frontal regions, was correlated with slower performance in tasks assessing executive functioning. Based on the relation between alpha 1 and attentional processes (see Section 1.2.3) and on the hypothesized role of frontal dysfunction in OCD, results were suggested to reflect an association between enhanced frontal activation and hyperactivity of attentional and executive control processes in OCD patients. Shin et al. (2004) found a correlation between resting state alpha (7.5–12.5 Hz) and task performance in a visuospatial memory task in a mixed gender group of 23 OCD patients. The direction of this correlation, however, differed by hemisphere: decreased performance was associated with decreased left frontal alpha while better performance was associated with decreased right frontal alpha. It was consequently suggested that impaired visuospatial memory performance is related to prefrontal executive functioning deficits, and that right cortical hyperfrontality reflects a compensatory mechanism for left cortical hyper-frontality. Importantly, this study did not include a healthy control group thus it cannot be determine whether the results are specific to OCD.

2.5.2. Task-related studies

OCD-related alpha abnormalities were demonstrated also during the performance of working memory tasks. Ciesielski et al. (2007) used MEG to investigate prefrontal and parieto-occipital alpha (8–13 Hz) modulations during the performance of a visual-spatial delayed matching-to-sample memory task in 8 OCD patients and 8 healthy matched controls. Two version of the task were used,

one with and the other without a distractor presented soon following the encoding phase (i.e., stimulus presentation). The latter was included to increase task difficulty for OCD patients, known to exhibit unselective processing of task-relevant and task-irrelevant stimuli. Individuals with OCD showed reduced alpha ERD compared to controls when the task included a distractor. Specifically, a decrease in prefrontal alpha ERD was found during the memory retention phase (in which information is actively maintained) and in prefrontal and parieto-occipital regions during the retrieval phase (no effect in the memory encoding phase). In addition, OCD patients had lower pre-stimulus alpha (measured in the period precipitating the encoding phase) in the two task conditions (with and without distractor), unlike controls who showed enhanced pre-stimulus alpha during the more difficult task (with distractor), presumably reflecting enhanced cognitive readiness to the upcoming task. Together, these results were suggested to reflect a persistent desynchronization in OCD possibly due to intrinsic events, and reduced adjustment of neural response to higher task demands. It should be noted that these abnormalities were not accompanied by impairments in task performance. It was therefore suggested that these findings may reflect a compensatory mechanism of enhanced inhibitory control of internal distractors in order to maintain high task performance. Park et al. (2012) focused specifically on the information encoding phase of WM. Frontal and occipital alpha ERD (8–12 Hz) were examined with EEG in 15 medicated males and females patients and 15 healthy matched controls, that completed a modified version of the Sternberg working memory task (see Section 2.1 for details). In both groups, alpha ERD was more pronounced as memory load increased. However, OCD patients had reduced alpha ERD compared to controls. It has been suggested that such relative increased alpha power in OCD patients reflects a compensatory process in which enhanced effort is invested in order to inhibit task-irrelevant processing and to maintain behavioral performance.

Min et al. (2011) found abnormal modulation of pre-stimulus alpha power in OCD patients, which was suggested to reflect impaired top-down inhibitory control. They recorded EEG from a mixed gender group of 16 medicated OCD patients and a group of 16 healthy controls during the performance of a color (lower difficulty) and a shape (higher difficulty) discrimination task. Modulations of pre-stimulus occipital alpha (8–13 Hz) in the two tasks were examined. Higher pre-stimulus alpha power were found in the shape task condition, however, individuals with OCD had lower pre-stimulus alpha power compared with controls which was suppressed to the same extent regardless of task type. It was therefore concluded that OCD patients generally show abnormal alpha desynchronization before stimulus onset. Based on the suggested association between pre-stimulus alpha and cognitive preparedness to an upcoming task and the suggested role of alpha activity in the active suppression of task-irrelevant processing (see Section 1.2.3), findings were suggested to reflect the impaired top-down inhibitory control characteristic of OCD patients, who tend to show preoccupation with task-irrelevant processing.

Others demonstrated left lateralization of alpha activity using an emotional paradigm (Ischebeck et al., 2014), and decreased frontal alpha power during symptom provocation (Simpson et al., 2000). Ischebeck et al. (2014) investigated frontal EEG asymmetry in 20 males and females with OCD (some were medicated and some had comorbidity with depression) and in 20 healthy matched controls during rest and while viewing emotional (aversive and OCD-related) and neutral pictures. Alpha asymmetry was analyzed in the lower (8–10 Hz) and upper (10.5–12.5 Hz) bands. Compared to controls, OCD patients showed increased left-sided lower alpha in all experimental conditions. Results were not affected by medication and comorbidity, and were suggested to indicate increased activity of a motivational system associated with avoidance and withdrawal, indicated by enhanced right cortical activity (See Section 2.1 for theory). Finally, decreased frontal alpha (7.8–11.7 Hz) power was found in 6 patients with OCD (3 were medicated) during symptom provocation, in line with other works showing hyperactivation of frontal regions in OCD (Simpson et al., 2000).

2.5.3. Summary OCD

Results demonstrate alterations in alpha power associated with rest and with tasks related to cognitive functioning known to be disturbed in OCD. Resting state alpha power was found to be decreased in OCD patients (Karadag et al., 2003; Locatelli et al., 1996) and showed sensitivity to OCD subtypes and symptom severity (Karadag et al., 2003). It was also shown by some to be associated with decreased performance in cognitive tasks (Bucci et al., 2004; Shin et al., 2004). Decreased frontal alpha was also found during symptom provocation (Simpson et al., 2000). Results concerning alpha asymmetry were not consistent, showing enhanced left-sided alpha in rest and during emotional processing (Ischebeck et al., 2014), no alterations in asymmetry patterns in OCD patients compared to controls, but subtype-specific decrease in left vs. right alpha (Karadag et al., 2003). Alteration in working memory-related alpha modulations were demonstrated, showing decreased alpha ERD during the encoding (Park et al., 2012), retention and retrieval (Ciesielski et al., 2007) memory phases as well as decreased pre-stimulus alpha power (Ciesielski et al., 2007) which was evident also during examination of top-down inhibitory control (Min et al., 2011).

3. Summary, methodological considerations, and future directions

Overall, studies examining alpha power in affective, obsessive-compulsive, and post-traumatic stress disorders underscore the relationship between abnormalities in oscillatory alpha activity and psychopathology (see Table 1 for summary). In some cases, a general association between abnormality patterns and a specific disorder can be detected. For example, while alpha asymmetry appears to be sensitive to depression and, to some extent, to anxiety, it does not seem to be the case in PTSD (for a recent review on alpha asymmetry in PTSD see Meyer et al., 2015). In addition, increased alpha is more often associated with depression and general anxiety, while in OCD alpha is mostly decreased. It appears that to date, no clear distinction can be made between the different disorders on the basis of the various alpha measures.

In addition to differences among disorders, much inconsistency exists among studies within each disorder. This inconsistency can be attributed to several factors. First, there is the great heterogeneity of the investigated populations in relation to gender, clinical profile, etiology, comorbidity and medication status. Studies on PTSD also vary in the nature of the traumatic event causing the disorder (e.g., combat, physical assault) and in the characteristics of the control group (trauma-exposed individuals with no PTSD diagnosis or trauma-unexposed participants), which may result in distinct brain activity patterns, as previously suggested (Patel et al., 2012).

Comorbidity with other psychopathologies is prevalent in individuals suffering from affective disorders, OCD, and PTSD. For example, high comorbidity levels were reported between depression and anxiety (Gorman, 1996; Shankman and Klein, 2003) and between PTSD and depression (Brady et al., 2000; Shalev et al., 1998). In some of the reviewed studies, additional analyses and experimental groups were included to test the effect of comorbidity on oscillatory abnormalities (Bruder et al., 1997; Ischebeck et al., 2014; Karadag et al., 2003; Mathersul et al., 2008; Rabe et al., 2006; Wise et al., 2011), however in others, such an examination was not

Table 1
Summary of reviewed literature.

Task/ rest	Disorder	Task type	Reference	Sample n (M/F)	Age[a] (range, mean ± SD/SE)	# of channels[b]	Medicated[c]	Primary findings[d]
Task	Depression	Working memory	Segrave et al. (2010)	30 (0/30)	21–59, 40.56 ± 12.12	64	Some	Increased ERS during retention phase
		Working memory, emotional	Segrave et al. (2012)	31 (0/31)	21–59,[e]	64	Some	Increased ERD during retention phase in negative distracter condition
		Working memory	Bailey et al. (2014)	48 (22/26)	17–65,[e]	64	Some	Decreased ERS during retention phase
		Emotional	Stewart et al. (2014)	306 (95/211)	17–34, 19.1 ± 0.1	64	No	Left asymmetry
	Bipolar disorder	Cognitive, potential reward/punishment	Harmon-Jones et al. (2008)	94 (43/51)	18–24,[e]	14	Some	Right asymmetry in preparation to a hard task with a potential reward
		Emotional	Lee et al. (2010)*	60 (24/36)	20–58,[e]	306	Yes	Distinct alterations in BD and MDD
	General anxiety	Induced anxiety	Knyazev et al. (2004)	30 (30/0)	18–25, 21.2 ± 3.5	32	No	Association between higher power and higher anxiety[f]
		Neutral words (auditory)	Knyazev et al. (2006)	30 (30/0)	18–25, 21.2 ± 3.5	32	No	Increased power during expectation for event in H anxious; Increased ERD in H anxious
		Personality feedback	Crost et al. (2008)	106 (106/0)	18–30, 24.2[g]	32	No	Enhanced left asymmetry in H anxious in negative feedback condition
	Social anxiety	Anticipation for public speaking	Davidson et al. (2000)	28 (n.a[h])	19–68,[e]	14	No	Left asymmetry
		Public speech	Beaton et al. (2008)	49 (12/37)	n.a,[i]	5	No	No group differences in asymmetry
	Panic disorder	Caffeine challenge	Newman et al. (1992)	14 (5/9)	n.a,[e]	28	No	No group differences in power
		Odor stimulation	Locatelli et al. (1993)	67 (20/47)	n.a,[e]	6	No	Distinct power alterations among PD subtypes
		Relaxation	Knott et al. (1997)	57 (24/33)	n.a,[e]	16	No	No effect on power
		Emotional	Wiedemann et al. (1999)	48 (9/39)	n.a,[e]	19	Some	Left asymmetry when viewing panic or anxiety-related stimuli; decreased power in response to panic-related stimuli
	PTSD	Emotional task	Rabe et al. (2006)	87 (27/60)	n.a,[e]	28	No	Left asymmetry while viewing trauma-related pictures
		Tactile stimulation	Badura-Brack et al. (2015)*	30 (30/0)	23–45,[e]	306	No	Decreased power
		Pain perception	Eidelman-Rothman et al. (2016)*	40 (40/0)	23–31, 27 ± 1.8	248	No	No differentiation in ERD response to painful vs. neutral stimuli
	OCD	Symptom provocation	Simpson et al. (2000)	6 (3/3)	31–37, 34.5 ± 2.2	17	Some	Decreased power
		Working memory	Ciesielski et al. (2007)*	16 (10/6)	15–39,[e]	122/306[j]	No	Decreased ERD during memory retention and decreased power during memory retrieval, when task difficulty increased; Decreased pre-stimulus power
		Cognitive	Min et al. (2011)	32 (19/13)	n.a,[e]	21	Yes	Lower pre-stimulus power;Similar ERD response during high/low task demands

Table 1 (*Continued*)

Task/ rest	Disorder	Task type	Reference	Sample n (M/F)	Age[a] (range, mean ± SD/SE)	# of channels[b]	Medicated[c]	Primary findings[d]
		Working memory	Park et al. (2012)	30 (22/8)	17–39,[e]	21	Yes	Reduced ERD during memory encoding
		Emotional	Ischebeck et al. (2014)	40 (17/23)	n.a,[e]	64	Some	Left asymmetry
Rest	Depression	–	Henriques and Davidson (1990)	14 (3/11)	n.a,[e]	14	No	Left anterior and right posterior asymmetry
		–	Henriques and Davidson (1991)	28 (11/17)	31–57,[e]	14	Some	Left asymmetry
		–	Bruder et al. (1997)	70 (35/35)	20–60,[e]	30	No	Left asymmetry in depressed patients with anxiety; Right asymmetry in depressed patients without anxiety
		–	Fingelkurts et al. (2006)	22 (12/10)	n.a,[e]	60	No	Increased power
		–	Mathersul et al. (2008)	156 (72/84)	n.a[k,e,k]	26	No	Decreased power
		–	Putnam and McSweeney (2008)	13 (4/9)	n.a,[e]	128	No	Left asymmetry
		–	Grin-Yatsenko et al. (2010)	637 (267/370)	17–60,[e]	21	Some	Increased power
		–	Kemp et al. (2010)	30 (12/18)	18–65,[e]	26	No	Left asymmetry; Increased power
		–	Segrave et al. (2011)	34 (0/34)	20–59, 41.47 ± 12.12	64	Some	No group differences in asymmetry
		–	Chang et al. (2012)	60 (0/60)	n.a,[e]	3	Yes	Left asymmetry
		–	Jaworska et al. (2012)	96 (44/52)	n.a,[e]	32	No	Left asymmetry; Increased power
		–	Gold et al. (2013)	757 (n.a[l])	n.a [l],[l]	32	Yes[m]	No group difference in asymmetry
		–	Stewart et al. (2014)	306 (95/211)	17–34, 19.1 ± 0.1	64	No	No group difference in asymmetry[n]
		–	Cantisani et al. (2015)	39 (18/21)	21–66, 42.21 ± 13.79	70	Yes[m]	Left asymmetry
	Bipolar disorder	–	Allen et al. (1993)	8 (0/8)	n.a, n.a	5	No	Left asymmetry
		–	Clementz et al. (1994)	144 (82/62)	n.a,[e]	3	Yes	Left asymmetry; Decreased power
		–	El-Badri et al. (2001)	55 (24/31)	19–39,[e]	10	Yes	Increased power
		–	Başar et al. (2012)	36 (n.a[o])	n.a[o,e]	30	No	Decreased power
	General anxiety	–	Saletu-Zyhlarz et al. (1997)	88 (39/49)	24–65,[e]	21	No	Enhanced power
		–	Knyazev et al. (2004)	30 (30/0)	18–25, 21.2 ± 3.5	32	No	Association between higher power and higher state/trait anxiety[f]
		–	Crost et al. (2008)	106 (106/0)	18–30, 24.2[p]	32	No	No group differences in asymmetry
		–	Mathersul et al. (2008)	104 (48/56)	n.a[k,e,k]	26	No	Left asymmetry; Distinct asymmetry patterns in anxiety subtypes
	Social anxiety	–	Davidson et al. (2000)	28 (n.a[h])	19–68,[e]	14	No	No group differences in asymmetry
		–	Sachs et al. (2004)	50 (n.a)	n.a	21	No	No group differences in power
		–	Beaton et al. (2008)	49 (12/37)	n.a,[i]	5	No	No group differences in asymmetry
	Panic disorder	–	Newman et al. (1992)	14 (5/9)	n.a,[e]	28	No	Decreased power
		–	Locatelli et al. (1993)	67 (20/47)	n.a,[e]	6	No	No group differences in power
		–	Wiedemann et al. (1999)	48 (9/39)	n.a,[e]	19	Yes	Left asymmetry

Table 1 (*Continued*)

Task/ rest	Disorder	Task type	Reference	Sample n (M/F)	Age[a] (range, mean ± SD/SE)	# of channels[b]	Medicated[c]	Primary findings[d]
		–	Wise et al. (2011)	156 (45/111)	n.a,[e]	16	Some	Decreased power; left asymmetry
	PTSD	–	Begić et al. (2001)	38 (38/0)	n.a,[e]	21	No	No group differences in power
		–	Jokic-begic and Begic (2003)	116 (116/0)	22–45,[e]	21	No	Decreased power
		–	Metzger et al. (2004)	50 (0/50)	48–61, 53.7 ± 2.8	6	No	Association between PTSD arousal symptoms and depressive symptoms and left asymmetry[f]
		–	Rabe et al. (2006)	87 (27/60)	n.a,[e]	28	No	No group differences in asymmetry
		–	Veltmeyer et al. (2006)	170 (85/85)	n.a,[e]	26	Some	Decreased power
		–	Shankman et al. (2008)	74 (43/31)	18–64,[e]	26	Yes	No group differences in asymmetry
		–	Gordon et al. (2010)	1956 (996/960)	n.a[q], n.a	32	Not specified	No group differences in asymmetry
		–	Kemp et al. (2010)	29 (11/18)	18–65,[e]	26	Some	No group differences in power or asymmetry
		–	Loganovsky and Zdanevich (2013)	241 (152/89)	36–75, 52.9 ± 7.8	16	Some	No group differences in power
		–	Wahbeh and Oken (2013)	86 (86/0)	n.a,[e]	32	Some	No group differences in power or asymmetry
		–	Huang et al. (2014)*	55 (53/2)	n.a,[e]	306	No	Decreased power
		–	Imperatori et al. (2014)	34 (14/20(	20–58,[e]	19	No	No group differences in power
		–	Eidelman-Rothman et al. (2015)*	40 (40/0)	23–31, 27 ± 1.8	248	No	Increased power
	OCD	–	Locatelli et al. (1996)	67 (34/33)	n.a,[e]	6	No	Decreased power
		–	Karadag et al. (2003)	63 (17/46)	n.a,[e]	19	No	Decreased power; Distinct power alterations in OCD subtypes
		–	Bucci et al. (2004)	64 (34/30)	16–44,[e]	16	No	Decreased power
		–	Shin et al. (2004)	23 (14/9)	n.a,[e]	21	No	Correlation between decreased *left* frontal power and decreased task performance and between decreased *right* frontal power and increased task performance[f]
		–	Ischebeck et al. (2014)	40 (17/23)	n.a,[e]	64	Some	Left asymmetry

M = males; F = females; SD = standard deviation; SE = standard error; ERS = event-related synchronization; ERD = event-related desynchronization; BD = bipolar disorder; MDD = major depression disorder; H = high; PD = panic disorder; PTSD = post-traumatic stress disorder; OCD = obsessive compulsive disorder

[a] Years.
[b] Number of EEG electrodes/MEG sensors.
[c] At the time of the experiment.
[d] Findings refer to the clinical/symptomatic groups only.
[e] M age and SD/SE are available only for subgroups.
[f] Result showing an association between alpha activity and behavior are listed only for studies in which this was the only/primary result.
[g] Mean age and age range refer to the final sample of analyzed participants.
[h] M/F ratio is available only for a broader, initial sample.
[i] Mean age is available only for a broader, initial pool of potential participants.
[j] Two different MEG systems were used.
[k] Age range, mean age and SD are available for a broader initial sample.
[l] M/F ratio, mean age SD and age range are available only for the depressed group.
[m] 97% medicated.
[n] Resting state asymmetry were found when an EEG reference-free measure was applied, see text.
[o] M/F ratio and age range are available only for patients.
[p] Age range refers to the final sample of analyzed participants.
[q] Age range is available only for controls.
* MEG study (otherwise the study was conducted using EEG).

included (Badura-Brack et al., 2015; Bailey et al., 2014; Eidelman-Rothman et al., 2015; Harmon-Jones et al., 2008; Jaworska et al., 2012; Knott et al., 1997; Metzger et al., 2004). The observation by some of the studies (Bruder et al., 1997; Karadag et al., 2003) that alpha patterns were affected by comorbidity highlights the need to consider comorbidity status when studying individual difference in alpha activity.

Similarly, many of the clinical participants were treated with various medications (yet, clinically symptomatic), whose effects were controlled in some (e.g., Clementz et al., 1994; Henriques and Davidson, 1991; Ischebeck et al., 2014; Veltmeyer et al., 2006 Ischebeck et al., 2014; Veltmeyer et al., 2006), but not in all (e.g., Chang et al., 2012; Kemp et al., 2010; Shankman et al., 2008) studies. Since neural oscillations are known to be affected by a range of psychotropic medications (Buzsáki et al., 2013), including in the alpha frequency band, as was demonstrate in one of the reviewed studies on PTSD (Veltmeyer et al., 2006) it is important to consider medication status in such studies.

Other methodological variabilities may also contribute to inconsistency of findings. For example, different alpha frequency ranges were examined across studies. While some studies examined a wide, standard alpha frequency range, others included investigation of alpha sub-bands, based on the view that different alpha frequencies are associated with different functionality (Klimesch, 1999 and see Section 1.2.3). In some cases, such investigation revealed band-specific results, including in MDD (Jaworska et al., 2012), anxiety (Crost et al., 2008; Davidson et al., 2000; Wise et al., 2011), OCD (Bucci et al., 2004; Ischebeck et al., 2014; Locatelli et al., 1996) and PTSD (Jokic-begic and Begic, 2003; Veltmeyer et al., 2006). Therefore, analyzing different frequencies of alpha may be important in specifying alpha abnormalities and their possible relation to behavioral disturbances in the different disorders and should be considered in future research. In addition, while most of the studies used a fixed alpha range, in others, individually adjusted bands (see Section 1.2.3) were calculated to account for individual variations in peak alpha (Knyazev et al., 2006, 2004; Segrave et al., 2012, 2011, 2010).

Studies also differed in the EEG reference montage used, a parameter that has been shown to affect EEG results (Hagemann, 2004; Salinsky et al., 1991). Indeed, in some of the studies, different EEG references were tested, which produced variability in results (Henriques and Davidson, 1991; Jaworska et al., 2012 Jaworska et al., 2012).

Another parameter which varied across studies is the experimental condition in which alpha abnormalities were assessed, i.e., resting state vs. task activation. While overall alpha alterations were demonstrated in both conditions, some of the reviewed studies compared between experimental conditions and demonstrated context-dependent alterations in alpha activity patterns, which were evident only when specific stimuli/tasks were included, but not during rest (Crost et al., 2008; Davidson et al., 2000; Rabe et al., 2006; Stewart et al., 2014; Wiedemann et al., 1999; but see Beaton et al., 2008; Ischebeck et al., 2014 and also Stewart et al., 2014 for methodological considerations). Therefore, including measurements in specific contexts may reveal more pronounced disorder-related alteration, as suggested previously (Coan et al., 2006; Wahbeh and Oken, 2013).

Notably, with the exception of two studies (Beaton et al., 2008; Knott et al., 1997) all studies in which tasks were included were successful in demonstrating individual differences in alpha patterns. These include memory-related tasks in depression (Bailey et al., 2014; Segrave et al., 2011, 2010; Stewart et al., 2014) and OCD (Ciesielski et al., 2007; Park et al., 2012) studies, a cognitive-motivational task in BD (Harmon-Jones et al., 2008), a top-down inhibitory control tasks in OCD (Min et al., 2011) and a sensory stimulation in PTSD (Badura-Brack et al., 2015). In addition, induction of psychiatric symptoms in anxiety disorders (Beaton et al., 2008; Crost et al., 2008; Davidson et al., 2000; Knyazev et al., 2004; Wiedemann et al., 1999) and OCD (Simpson et al., 2000), also revealed alterations in alpha patterns, as well as the presentation of emotional stimuli or material specifically associated with the disorder in BD (Lee et al., 2010), OCD (Ischebeck et al., 2014) and PTSD (Eidelman-Rothman et al., 2016; In Press). Although most of the resting state studies also revealed individual differences in alpha power measures (albeit not always in all measures tested) and thus provided important information regarding the association between aberrant alpha patterns and psychiatric conditions, by using specific tasks, an association between alpha patterns and specific perceptual, cognitive and emotional processes known to be disrupted in psychiatric disorders was demonstrated. While the majority of the studies reviewed here used resting state paradigm, more studies that employ specific tasks may shed further light on alpha-power abnormalities related to specific aspects of affective disorders, OCD and PTSD. This is particularly relevant in light of the fact that these disorders are characterized by deficits in attention and cognitive functioning, processes in which alpha activity has been shown to play an important role (Clayton et al., 2015; Jensen and Mazaheri, 2010; Klimesch, 1999).

Most studies reviewed above were conducted with EEG and did not include a source localization analysis. Additional MEG and EEG studies including source-level analysis are needed in order to identify the origins of alpha activity in health and in pathology, which may provide valuable information regarding specific networks that are disturbed in these disorders. Finally, while some disorders were more extensively studied (e.g., depression), others received less attention, including bipolar and panic disorders, and more research on these disorders is needed. In addition, some of the studies were conducted with a very small sample size and replications with larger samples are warranted.

Future research, which will consider such methodological issues, may enhance our understating of the abnormalities associated with different types of disorders, towards the goal of improving diagnosis and therapeutic approaches.

4. Interpretation of alpha power in psychiatric disorders

The majority of resting state studies relied on the inverse relations between alpha power and neural activity when interpreting the results and thus, used alpha power as an indication for increased or decreased cortical activity in a certain region. However, in one study, decreased alpha seemed to be interpreted as decreased activity in the corresponding region. This study recorded resting state MEG from individuals with PTSD and found decreased alpha power in the dlPFC and the precuneus. These results were described as consistent with fMRI studies showing decreased activity in similar regions, and were suggested to reflect a decrease in cognitive-emotional functioning attributed to these regions (Huang et al., 2014). Similarly, in Badura-Brack et al. (2015), decreased alpha in the prefrontal cortex was suggested to indicate decrease in attentional processing, associated with activity in this region.

In studies which employed tasks, increased alpha power in specific phases of the tasks (e.g., before stimulus presentation – pre-stimulus alpha, and during memory retention phase) were interpreted, based on previous research, as reflecting active inhibition of task-irrelevant information, as well as a state of cognitive readiness and preparedness to an upcoming tasks (See Section 1.2.3). However, in others, enhanced event-related alpha power seemed to be interpreted as increased activity in the underlying cortex. This was demonstrated in a MEG study (Lee et al., 2010) showing that BD was associated with increased alpha in regions of the inferior frontal gyrus and the insula, and based on the associ-

ation between these regions and affective evaluation and emotion detection as observed with fMRI, results were suggested to reflect increased sensitivity to emotionally salient information.

In some of these studies, specific alpha sub-bands were analyzed based on their suggested association with distinct cognitive processes during task performance (Klimesch, 1999 and see Section 1.2.3). However, while in some cases, the activation of a certain cognitive process was suggested to be reflected by increased *desynchronization* in a specific alpha sub-band (e.g.,Wise et al., 2011) as previously suggested (Klimesch, 1999), in others, it was suggested to be indicated by increased alpha *synchronization* (Knyazev et al., 2004). In the latter case, which was demonstrated in anxious individuals, the authors pointed at the seemingly contradiction of their observed enhanced alpha ERS with the traditional view of alpha desynchronization as a measure of response to task activation. They raised some methodological considerations, emphasizing that the alpha increase was measured a number of minutes following the application of an anxiety provoking event and suggested that the increase in alpha power may reflect an adjustment process that occur after the expected ERD. Although the latter was not measured, such a phenomenon of increased alpha power following alpha ERD has been previously described in the literature as a rebound of alpha power (Caetano et al., 2007; Pratt et al., 2016). Alternatively, the authors suggested that these results may reflect a state of readiness in anxious individuals which can also be seen during rest and is further enhanced following a provoking event. Following a further analysis of their data (Knyazev et al., 2006) showing enhanced alpha in reference periods along with enhanced alpha ERD in response to a non-provoking task, they suggested that the alpha enhancement observed in anxious individuals should not be considered as a sign of inhibition of irrelevant processes as suggested by the inhibition theory (e.g., Klimesch, 1999; Klimesch et al., 2000) but rather, as a state of enhanced readiness of corresponding networks to information processing. Their theory posits that any state involving enhanced attention should be associated with alpha enhancement, and that anxious individuals in particular, will tend to show higher alpha power in reference intervals as well as higher magnitude of alpha desynchronization.

Some of the observed task-related alterations in alpha power relative to controls were suggested to reflect pathological characteristics. However, since such abnormalities were not necessarily accompanied by a decrease in cognitive performance, these were alternatively suggested to reflect an adaptive/compensatory mechanism, including enhanced inhibition of distracting, task-irrelevant information and enhanced recruitment of neural resources, developed in order to maintain normal cognitive functioning (e.g., Chang et al., 2012; Ciesielski et al., 2007; Segrave et al., 2012, 2010).

The accumulated evidence and theory on alpha functionality and its central role in different aspects of information processing provided a framework for establishing an association between abnormal brain activity and the different psychiatric disorders. Although the findings described in the reviewed studies are correlational and no causality can be inferred, they demonstrate aberrant alpha behavior in these disorders. Abnormal alpha patterns observed during rest may point at individual differences in intrinsic neural dynamic which in some cases was related to disorder symptoms or personality tendencies. Atypical alpha observed during task application, combined with information regarding task performance and the specific behavioral characteristics of the disorder can provide insights into possible mechanisms underlying phenomena such as distractibility, altered response flexibility and response bias, and into possible adaptive or compensatory alpha responses applied by individuals with psychiatric disorders. Conversely, such observations may also shed further light on alpha functionality and the modulations and possibly adaptation of alpha patterns in different contexts.

While correlations between neural oscillations and human healthy and pathological behavior is well established, evidence for causal association is still sparse (Thut et al., 2012). Neural oscillations and alpha oscillations in particular, have been found to be amenable to external intervention and can be experimentally manipulated and measured using noninvasive techniques such as transcranial magnetic stimulation (TMS) with concurrent EEG (Fingelkurts and Fingelkurts, 2015; Herrmann et al., 2015) and EEG-neurofeedback (Herrmann et al., 2015; Ros et al., 2014). Direct stimulation of the brain using repetitive (rhythmic) TMS (rTMS) in the alpha frequency range has been shown to induce alpha oscillations (Thut et al., 2011b) and to produce effects on task performance, which are in line with those associated with the alpha rhythm (Herring et al., 2015; Ruzzoli and Soto-Faraco, 2014). EEG-neurofeedback, which enables individuals to self-regulate their neural oscillations, has similarly been shown to affect alpha activity and subsequent behavior (Ros et al., 2013) and to alter alpha asymmetry patterns (Quaedflieg et al., 2015)

In light of the abnormalities in alpha activity evident in the psychiatric conditions discussed above, such external interventions may have potential therapeutic implications. Indeed, controlled manipulation of alpha activity has been shown to exert positive therapeutic effects in psychiatric conditions, including rTMS treatment for depression (see in Fingelkurts and Fingelkurts, 2015) and neurofeedback in PTSD (see in Ros et al., 2014). Thus, non-invasive interventions in alpha activity may be important not only for furthering our understanding on the role of oscillatory activity in brain function in health and psychopathology, but also for its applicability as a potential treatment target (Fingelkurts and Fingelkurts, 2015; Thut and Miniussi, 2009). This broad and interesting subject requires much further attention and discussing the use of such interventions is beyond the scope of this review. For relevant reviews see (Fingelkurts and Fingelkurts, 2015; Herrmann et al., 2015; Ros et al., 2014; Thut et al., 2012, 2011a; Thut and Miniussi, 2009).

5. Conclusions: can the study of alpha oscillations contribute to research on psychiatric disorders?

Neural oscillations are fundamental to brain functioning and understanding their functioning in healthy and high-risk conditions can contribute to the pinpointing abnormalities in brain functioning in specific psychopathologies, potentially leading to better diagnosis and more targeted therapeutic interventions (Başar, 2013; Buzsáki and Watson, 2012; Uhlhaas and Singer, 2012).

Assessing the effects of pharmacological interventions may also shed light on alpha activity in psychopathology and its mechanisms, particularly by examining the effect of pharmacological agents with known mechanisms of action on alpha oscillations. Several studies have demonstrated alterations of alpha activity by external intervention in populations with affective disorders and PTSD. For example, Özerdem et al. (2008) tested oscillatory activity in bipolar patients before and after a six week treatment with valproate, an anti-manic agent (Bowden, 2003) which acts on GABA-related inhibitory neurotransmission (O'Donnell et al., 2003), known to be attenuated in bipolar disorder. Reduced alpha activity was found post-treatment, which was suggested to reflect an inhibitory effect mediated by GABAergic activity. In a research conducted in our lab, although mechanism of action is not understood, a single administration of the neuropeptide oxytocin was found to decrease abnormally increased alpha activity in a group of veterans with a PTSD and partial PTSD diagnosis (Eidelman-Rothman et al., 2015).

This review demonstrates that alpha rhythm, a fundamental mechanism of brain activity, is disturbed in individuals with psy-

chiatric disorders, and that these disturbances are not only revealed by task activation but are rather present during a baseline, resting condition. We suspect that, as in the case of genetic susceptibility to affective and other psychiatric disorders, alpha abnormalities exist among individuals, and in some cases, develop into psychopathology, possibly through a combination with other physiological and behavioral factors. Alpha activity has been shown to play a pivotal role in basic social processes (Section 1.2.2.1), including the processing of another person's pain and inter-individual, brain to brain synchrony. Therefore, these disorders involve impairment to a basic mechanism supporting perceptual, attentive, and cognitive processing as well as important aspects of social functioning, which are critical to the human ability to connect to others. This highlights the unique perspective of the alpha rhythm and the importance of further exploring it in the research on brain mechanisms underlying these disorders, which are characterized by aberrant cognitive processing, severe social impairments, sense of lowliness, and dysregulation of emotions and response to internal and external stimuli.

All of the above, combined with knowledge accumulated from animal research, may further our understanding of psychiatric conditions on a more mechanistic level and help improve diagnostic and therapeutic approaches. As the role, significance, and mechanisms of neural oscillations in human behavior are still under investigation, insight gained from the investigation of alpha activity in various psychopathologies, particularly those involving social dysfunction, may provide a valuable contribution to the general understanding of neural oscillations.

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