Interpreting EEG alpha activity

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Abstract

Exploring EEG alpha oscillations has generated considerable interest, in particular with regards to the role they play in cognitive, psychomotor, psycho-emotional and physiological aspects of human life. However, there is no clearly agreed upon definition of what constitutes ‘alpha activity’ or which of the many indices should be used to characterize it.

To address these issues this review attempts to delineate EEG alpha-activity, its physical, molecular and morphological nature, and examine the following indices: (1) the individual alpha peak frequency; (2) activation magnitude, as measured by alpha amplitude suppression across the individual alpha bandwidth in response to eyes opening, and (3) alpha "auto-rhythmicity" indices: which include intra-spindle amplitude variability, spindle length and steepness.

Throughout, the article offers a number of suggestions regarding the mechanism(s) of alpha activity related to inter and intra-individual variability. In addition, it provides some insights into the various psychophysiological indices of alpha activity and highlights their role in optimal functioning and behavior.

Key words: *individual alpha peak frequency, individual alpha bandwidth, alpha amplitude suppression, spindle-form segments length, intra-spindle amplitude variability*

Highlights

1. Alpha indices are amplitude, peak frequency, band width and spindle structure
2. Alpha peak frequency could be an endophenotypic marker
3. Alpha band width and amplitude suppression reflect activation
4. The spindle-form, segment length and amplitude reflect a neuronal ensemble property
5. Alpha activity is manifested depending on the individual alpha peak frequency

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1. Introduction

Exploring EEG alpha oscillations has generated considerable interest with regard to their role in cognitive (Klimesch et al., 1993, 1996; Hanslmayr et al., 2005;), sensorimotor (Bernshtein, 1966; Baumeister et al., 2008; Bazanova et al., 2009; Sauseng et al., 2009), psycho-emotional (Aftanas & Golosheĭkin, 2003; Cacioppo, 2004) and physiological (Cooray et al., 2011; Kiyatkin, 2010; Kiyatkin & Lenoir, 2011) aspects of human life. However, at present there is no clear agreement regarding the functional meaning of 'alpha wave activity' and which measure, or measures, should be used to characterize it. In addition, the ambiguity of phrases such as 'the alpha rhythm is activated' (Babenko et al., 2003, p.1305) becomes apparent when considering the meaning of the term 'activated'. It is not clear if this refers to an increase or a decrease in amplitude. Further ambiguity is evident when attempting to identify the quantitative equivalents of terms such as the 'prominent rhythm', or an 'organized EEG', 'flat EEG' and 'regular oscillations' etc. (see e.g., Babenko et al., 2003). The fact that a variety of EEG rhythmical components are described by the same dominant frequency as the alpha rhythm, with distinct frequency and topographical boundaries, adds to the confusion. As such, speaking of alpha wave activity often implies some change in amplitude across a standard frequency range (e.g., 8-12Hz), invariably without reference to the oscillatory feature referred to as the 'Berger effect' (Kirschfeld, 2005) which in turn has led to inconsistencies regarding the psychophysiological role of alpha activity (Nunez et al., 2001) and this has led to divergent interpretations of the role of alpha activity (see e.g., Cooper et al., 2003; Klimesch et al., 2007; Palva & Palva, 2007).

LaVaque (1999) has suggested that it is easier to understand the role of alpha wave activity when viewed from an historical perspective. Hence, in an attempt to understand these issues and set them into context we begin with a brief historical reflection on the nature of alpha wave activity. This includes identification of the original 'Berger Effect'

along with the development of quantitative EEG (QEEG) measurements which represented an important step in realizing the necessity of evaluating the frequency when studying the nature of alpha waves (Fuentealba et al., 2005; Hughes et al., 2011; Steriade & Timofeev, 2003). We also highlight the notion that the EEG represents a dynamic signal and indicate how the development of non-stationary computer analysis has helped in defining phase modulation, including measures of auto-rhythmicity (Lehmann et al., 1994; Livanov, 1984; Kaplan et al., 2002).

In section 3 we outline a number of reasons why amplitude across a fixed frequency range of 8-12Hz should not be the sole measure of alpha activity. These include: (i) anatomical-physiological influences, (ii) the influence of topography, (iii) the effect of engaging in different tasks, and (iv) the divergent frequency ranges used to measure alpha amplitude. Discussing each of these issues also helps to highlight the benefits of studying alpha activity relative to other frequency ranges.

Following this, in section 4, we promote the idea that alpha activity can be measured using individual alpha peak frequency. Here we examine how frequency can be assessed, the effect of inter-individual variability and the influence of genetics on the production of alpha waves. This is important because it can help shed light on various brain activation models as well as provide insights for studying cognitive behaviour and devising EEG based neurofeedback training (NFT) protocols. In section 5 we examine alpha amplitude suppression as this is one of the key unique features of alpha waves. Finally, in section 6, we examine the micro-structural characteristics of the spindle-shaped bursting segments that play a key role in the processes of cognition, mood and sensorimotor performance. Such characteristics can provide useful additional information alongside the more traditional FFT analysis (see Figure 1).

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Figure 1 about here

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2. Historical reflections

2.1. Berger's waves

The history of investigating alpha waves is closely related to the progress in technological developments used to measures these waves. Given the profound difficulties associated with EEG signal acquisition and analysis, EEG researchers have invariably been early adopters of new technology. Due to the low sensitivity of the first Siemens galvanometer Berger (1932 as cited in LaVaque, 1999) could only record the high amplitude intra skin electrical potentials which coincided with 100-200 millisecond (ms) time intervals. In other words the frequency of these dominant amplitude waves may have been distinct in different subjects across the range of 8 to 12 Hz. It should be noted that Berger did not specify the width of the frequency range of these alpha waves he merely identified that such waves usually had a frequency of between 8 to 12 Hz. At that time the raw EEG signal was invariably recorded on paper and without the help of computers to process the signal it was impossible to determine the individual alpha band width. Hence, there was some initial agreement to simply name the alpha-band as the 8-12 Hz range. Subsequently Berger, and his students, noted that an important characteristic of these alpha waves was the suppression in amplitude seen in response to opening the eyes (Berger, 1932 as cited in LaVaque, 1999). Over time this has simply become known as the 'Berger effect' or reaction of activation (Barry et al., 2007).

This important alpha activity index is discussed below (see section 5).

2.2. Quantitative EEG

The next advancement in the psychophysiological study of alpha waves was the appearance of Quantitative EEG (QEEG) measures. QEEG began approximately 80 years ago when Dietsch (1932) applied a Fourier analysis to records of the EEG. The Fourier analysis remains one of the most popular analytical techniques in the field and though not the only measure it has become more widespread due to the advent of powerful personal computers which in turn have facilitated research in understanding the EEG (Niedermeyer, 2004). The Fourier transform has enabled researchers and clinicians to define a number of components of alpha wave activity, including spectral alpha peak frequency, the reaction activation or Berger-effect, the frequency of individual alpha sub-bands, and the power in these bands (Lopes de Silva, 1991; Fong and Fong, 2001; Barry et al., 2007; Bazanova and Aftanas, 2008; Hooper, 2005). Thus, such developments have made it possible to study and discuss a number of alpha activity indices, including the dominant amplitude and frequency as well as how they change across various conditions.

2.3. EEG is not a stationary recorded signal

However, it has been suggested that the dominance of the amplitude of the EEG in the parietal-occipital region along with the 'Berger effect' and frequency range where it occurs are not enough to provide a comprehensive understanding of alpha wave activity (Lansky et al., 1979). Lansky et al. (1979) proposed that power in the alpha band can only be a criterion for assessing alpha wave activity if alpha-spindle length is also simultaneously estimated. Indeed, conventional spectral analysis based on averaging procedures may be limited because the EEG is not a stationary recorded signal. To overcome these limitations of spectral analysis and to reveal the dynamic and temporal characteristics of alpha waves a number of researchers have proposed that a number of individual short-term stationary segments of the EEG need to be obtained (Kaplan et al.,

2002; Lorincz et al., 2008; Mazaheri & Jensen 2010; Towers & Allen, 2009). For instance, many researchers have focused on the analysis of multiple stationary segments of the raw EEG signal, from which the amplitude variation can be estimated (Livanov & Dumnov, 1984; Schomer 2007). However, theoretical assumptions (Hooper, 2005; Kirschfeld, 2005) and analysis of the empirical data on alpha oscillation generation (Hughes et al., 2011; Steriade & Timofeev, 2003) has provided a basis for considering a third and informative phenomenological characteristic of alpha activity, its spindle like bursting and the segmental organization of alpha waves or their auto-rhythmicity (Timofeev & Bazhenov, 2005; Timofeev et al., 2002).

3. Why amplitude may not be the sole criterion of alpha waves activity

Possibly the most well known suggestion is that amplitude of the alpha frequency band is related to the synchrony of the underlying neuro-electrical source(s) (Nunez & Srinivasan, 2006). Consistent with this proposal a reduction in amplitude is often labeled as desynchronization (Pfurtscheller & Lopes da Silva, 1999). Of course, a reduction in amplitude may, in theory, occur as a result of either a reduction in the magnitude of the source or a reduction in the amplitude recorded on the scalp surface (Haueisen et al., 2000; Nunez & Srinivasan, 2006; Srinivasan 2006). For instance, it is well known that the value of any electrical potential measured on the surface of the scalp depends on a number of anatomical and functional factors (Akhtari et al., 2002; Dulla et al., 2005; Wen & Li 2006; Jochmann et al., 2011). Some of these are discussed below.

3.1. Anatomical and physiological factors

3.1.1. Conductivity

Firstly, volume conduction effects, such as poorly conducting bones or the more moderately conducting skin are known to influence the measurement, precision and

accuracy of the surface EEG amplitude (Wen, 2003, Wen & Li, 2006). The conductivity of living skull tissue is expected to be primarily due to the most abundant and most mobile (i.e., smallest) electrolytes such as Na+ and Cl-. In contrast to a saline soaked cadaver skull (Akhtari et al., 2002; Law, 1993), the living skull lattice consists of numerous charged molecules such as proteins occupying live cells and blood components. The interaction of these relatively immobile protein molecules with the more mobile ions is expected to affect the level of conductivity with respect to the frequency of the input current (see Akhtari et al., 2002). Therefore, the magnitude of conductivity of a living skull, although higher, is similar in order of magnitude to that of a saline soaked cadaver skull, however the frequency and current characteristics of conductivity in a living skull can be distinct from that of a cadaver skull. The research of Akhtari et al. (2002) indicates that the conductivity of the skull layers is frequency dependent across a range of 10 – 90 Hz. Hence, the lowest level of conductivity occurs within the low alpha frequency range (Akhtari et al., 2002). In addition, tissue disorders due to brain pathologies, like tumors, ischemia, or vasogenic edema, are known to impact the propagation of electrical fields (Jochmann et al., 2011). Remarkably, due to the 'shunting effect' and the diminishing anisotropy of tissue conductivity, the amplitude of a signal from a radial dipole located in a sulcus was found to be higher than a dipolar source on a gyrus, particularly if the ischemic area was located underneath the sulcus (Haueisen et al., 2000; Jochmann et al., 2011).

Such findings suggest that despite the fact that alpha EEG power and coherence are often used to assess functional connectivity in the human cortex, moderate to large EEG coherence can also arise simply as a function of the volume conduction of current through the tissue of the head (Srinivasan et al., 2007) or by increasing brain temperature (Kiyatkin, 2005). Thus, the age related decline seen in alpha amplitude across all areas of scalp (Chiang et al., 2011; Sebastián et al., 2011; Yordanova & Kolev, 1997) and the phenomenon of so called 'low amplitude alpha rhythm' could simply be associated with

reduced conductivity as a function of increasing age (Wendel et al., 2010) or with genetically determined low volume tissue conductivity.

Such results demonstrate that tissue conductivity changes need to be taken into account when evaluating the changing EEG amplitude signals, especially when performing source localization.

3.1.2. Cerebral blood flow

Important anatomical-physiological influences on amplitude measurements are also connected with changes in cerebral blood flow, which highlights the importance of the cardio-vascular and breathing systems. For example, it has been shown that baseline cerebral blood flow interacts with neural activity and influences evoked hemodynamic responses (Cook et al. 1998; Goldman et al., 2002). Goldman, Stern, Engel and Cohen (2002) also reported that increased alpha power was correlated with a decreased blood oxygenated level dependent (BOLD) signal in multiple regions of the occipital, superior temporal, inferior frontal, and cingulate cortex, but with an increased BOLD signal in the thalamus and insula. These results are consistent with animal experiments and point to the amplitude of the alpha rhythm as an index of cortical inactivity that may in part be generated by the thalamus. In addition, the work of Franceschini et al. (2010) has shown that the hemodynamic response is best correlated with secondary, late cortico-cortical transmissions, and not with the initial thalamic input activity. These findings, along with more recent data suggest that the hemodynamic response is predominantly driven by cortico-cortical interactions and not by the initial thalamocortical activity in layer IV (Franceschini et al., 2010; Radhakrishnan et al., 2011).

Cerebral blood flow is typically reduced during stable non-rapid eye movement (non-REM) sleep compared with waking activity. Kotajima, Meadows, Morrell and Corfield (2005) have shown that spontaneous fluctuations in power at a frequency of 3-9

Hz during sleep onset are associated with marked changes in cardio-respiratory control. They speculate that the changes in cerebral vascular tone during sleep onset are mediated neurally, by regulatory mechanisms linked to changes in cortical state. Recent data has shown that factors (such as caffeine or cocaine) that can produce changes in cerebral blood flow velocity can also simultaneously change the level of power in the lower frequency alpha range across all electrode sites influenced by cortical blood flow changes (Copersino et al., 2009; Sigmon et al., 2009).

Another example of neurovascular coupling is the recent finding that higher CO_2 partial pressure can have a profound effect on neural tissue including the reduction of pH levels, elevating adenosine concentration, and suppressing synaptic potentials (Dulla et al., 2005; Zappe et al., 2008). Scalp EEG studies comparing hypercapnia with normocapnia conditions have shown a relative increase in low frequency (5-9Hz) power in the EEG spectra, suggesting that the brain may be entering a low arousal state during CO_2 inhalation and that the slowing of the EEG signal appears in all electrode sites across the entire brain (Xu et al., 2011).

Interestingly, obesity has also been shown to influence the resting state of regional cerebral blood flow and consequently the amplitude of alpha (Babiloni et al., 2011). These results showed that alpha 1 sources fitted a pattern whereby underweight>normal-weight>overweight/obese and where alpha 2 power was stronger in the normal-weight subjects compared to either the underweight or overweight/obese subjects (Babiloni et al., 2011).

Thus, it is clear that a relationship exists between changes in alpha amplitude and blood flow. However, the nature of this relationship remains complex and it is not clear whether the cortical synaptic activity generated by thalamic input or the subsequent synaptic activity related to secondary cortical processing is driving the hemodynamic response. Initial investigations of prefrontal [oxy-Hb]/[deoxy-Hb] oscillations and central

EEG power changes in the upper alpha band have suggested that the positive [oxy-Hb] peaks preceded the central EEG upper alpha power peak and relates to the conscious intention to perform a motor act (Pfurtsheller et al., 2012). Nevertheless, it remains the domain of future research to elucidate more fully the relationship between changes in blood flow and changes in neuronal activity.

3.1.3. Hormonal and neurohumoral factors

There are suggestions that a direct hormonal modulation of brain electrophysiology or underlying factors (e.g. the corticotrophin-releasing hormone), pacing both stress hormones and EEG, may account for individual EEG differences (Sannita et al., 1999). However, there are limited and conflicting findings regarding the effects of hormones or neurohumoral status on the amplitude of alpha oscillations (Field et al., 1996; Sannita et al., 1999; Keogh et al., 2012;). It has been shown that concentrations of cortisol, glucose and adrenocorticotrophic hormone (ACTH) within the blood or saliva can vary spontaneously with EEG power across a range of 6.5-14.0 Hz, which includes the alpha rhythm (Sannita et al., 1999). Such a pattern of changes suggests an inverted U shaped relationship with ACTH concentration but remains independent of the extent of ACTH change or from cortisol/glucose concentrations (Sannita et al., 1999). Other hormonal and neurohumoral influences on resting alpha amplitude across a standard 8-12 Hz frequency range have yet to be examined in full, which is why only limited effects have been reported to date (Güntekin & Başar, 2007; Solís-Ortíz et al., 2004; 2009;).

3.1.4. Electromyogenic influences

Muscle or electromyogenic (EMG) artifact poses a serious risk to the inferential validity of any EEG investigation in the frequency-domain owing to its high amplitude, broad spectrum, and sensitivity to psychological processes of interest (Bautista, 2011;

Goncharova et al., 2003; Halliday et al., 1998; McClelland et al., 2012; Shackman et al., 2009). While EMG contamination is greatest at the periphery of the scalp, near the active muscles, even weak contractions can produce EMG interference that obscures or mimics the alpha, mu, or beta rhythms over the entire scalp (Goncharova et al., 2003). Moreover, cognitive task performance often activates EMG in scalp electrical recordings making it difficult to differentiate EEG from EMG signals in the theta, beta and gamma ranges (Whitham, et al., 2008). Generally, EMG has a broad frequency distribution from 0 to >200 Hz and spectra that often have peaks in the beta frequency range that resemble EEG beta peaks.

There are a number of studies that have investigated the effects of peripheral afferent stimuli on the synchrony between brain and muscle activity as estimated by cortico-muscular coherence (Chakarov et al., 2009; Goncharova et al., 2003; Halliday et al., 1998; McClelland et al., 2012). The results from this research favors the view that the function of the beta range (>12Hz) is not specific for neural activity only. The sensorimotor system may also resort to stronger and broader beta-range cortico-muscular coherence to generate stable cortico-spinal interactions during increased force, as well as when compensating for dynamic modulated forces (Chakarov et al., 2009). This finding reinforces the importance of the upper alpha and beta range EEG-EMG coherence levels during sensorimotor integration (Chakarov et al., 2009; McClelland et al., 2012). It is suggested that both cutaneous and proprioceptive afferents have access to circuits generating cortico-muscular coherence, and that a functionally relevant stimulus can produce a significant modulation of 14-20 Hz-range coherence. Such findings have led to the conclusion that scalp EMG could be a contaminating factor while recording EEG particularly within the beta, gamma and theta ranges (Chakarov et al., 2009; Halliday et al., 1998; Hashimoto et al., 2010; McClelland et al., 2012). Hence, it is possible that lower

levels of EMG contamination may occur within the 8-12 Hz EEG spectrum providing a clear advantage to exploring the nature of alpha wave activity.

Some studies recommend elimination of EMG contamination by recording the EEG from an appropriate set of peripheral scalp locations (Goncharova et al., 2003). Shakman et al. (2009) reviewed recent work in their laboratory which investigated the validity of two popular EMG correction techniques, one using a general linear model (GLM) and the other using temporal independent component analysis (ICA). However, both of these methods exhibited difficulties when the amplitude of the EEG and EMG were comparable in magnitude. Interestingly, surface scalp Laplacian transformations have been shown to provide robust estimates for detecting high frequency EMG amplitude and also for providing a measure of electrical brain activity and as such could be used as a standard in the development of brain/muscle signal separation methods (see e.g., Fitzgibbon et al., 2012).

Taken together the data outlined above showing the dependence of alpha amplitude on a number of non-neuronal factors has led us to conclude that the terms ‘synchronization’ and ‘desynchronization’ do not always provide an unambiguous index of an increase and/or decrease in amplitude *alone.*

3.1.5 Low voltage subtype

It should also be noted that an EEG phenotype exists whereby little or no alpha is evident. This low voltage EEG (LVEEG) was originally described by Adrian and Mathews (1934) though there is no clear agreed upon definition (see Niedermeyer, 1986) as some use the term to refer to low voltage across the full spectrum of the EEG whilst others refer specifically to a lack of alpha in the resting EEG as a characteristic feature of such low voltage (see e.g., Anokhin et al., 1992). Prevalence rates also vary depending on the criteria and method used to assess it (ranging from 3% to 13% of the healthy population;

(Bodrov et al., 1984; Anokhin, 1988). Such a pattern has also been linked to alcoholism (Bierut et al., 2002), brain trauma (Nuwer, et al., 2005, 2012), and may be confused with depression caused by tension and anxiety (Schmidt et al., 2012). For those with LVEEG, alpha waves within the 8-12 Hz range may be diffuse and lacking in any rhythmical component. According to Anokhin et al (1992) this abnormal EEG pattern is the result of an autosomal dominant inherited gene.

The LVEEG also appears to be 'non-responsive', which Anokhin et al (1992) suggest indicates some difference between those with LVEEG and those with average alpha EEG. For instance, LVEEG participants may exhibit decreased performance in tests of concentration along with reduced spontaneous activity (Vogel, et al., 1979). Vogel et al. (1979) suggested that this pattern indicates poor modification and selective amplification of incoming stimuli as a result of weakened thalamocortical links. This was supported by research showing reduced amplitude short latency ERPs to visual and auditory stimuli (Vogel, 1986). Indicating a possible reduction in strength and speed of information processing. This is also consistent with more recent findings showing decreased alertness and sensory processing concomitant with decreased alpha (Braboszcz & Delorme, 2011).

Nevertheless, this phenotype has invariably been defined in terms of reduced amplitude. As outlined above amplitude alone can be influenced by a range of anatomical and functional factors. As such, future research may be able to elucidate further the underlying mechanisms associated with LVEEG by incorporating individual peak frequency range rather than relying solely on amplitude within a fixed frequency range.

3.2. Topographical factors

Alongside the anatomical and physiological factors that can often hinder the identification of alpha wave signals in the EEG are differences in topography (Kaiser, 2005). The description of topographic variability of alpha amplitude is inherent in the

literature exploring the phenomenon of alpha activity. Nevertheless, the choice of which recording location to use may influence and/or limit the interpretability of the quantitative EEG measures taken (Nuwer, 1988; 2003; Trambaiolli et al., 2011).

3.2.1. The effect of montage choice on amplitude

It has been proposed that spectral power calculations using different montage of electrodes can also have an influence on the classification and differentiation of results from both normal healthy subjects and patients with mental impairment (Trambaiolli et al., 2011). For instance, topographical focus on the amplitude of low frequency ranges (<8 Hz) is dependent on the referent electrode position such that the range of amplitude may be unreliable by itself (Nuwer, et al., 1987; Nuwer, 1988). Hence, the selection of a particular montage may also influence how alpha wave activity is discerned.

3.2.2. The importance of spatial resolution

Jensen and Mazaheri (2010) have proposed that additional insights into the functional role of alpha activity could be brought about by simultaneous high-density EEG and MEG recordings. However, the improved spatial resolution of these techniques has facilitated only the spatial, not the functional interpretation (Jensen & Mazaheri, 2010; Moore et al., 2008). Furthermore, circulatory arrest has been shown to have an impact on decreased alpha power appearing across all sites on the scalp. A factor analysis, conducted by Visser et al. (2001), revealed four factors that could account for the spectral EEG changes occurring during circulatory arrest and recovery. The frequency intervals of these factors were 0 to 0.5 Hz, 1.5 to 3 Hz, 7.5 to 9.5 Hz, and 15 to 20 Hz for all channels. In addition, only minor topographical differences were found in the power of the spectral changes. This provides compelling evidence that spatial resolution is not essential for studying changes in alpha power (Behrens et al., 1995; Visser et al., 2001).

Similarly, it's not clear from a topographical point of view why alpha neurofeedback training, or repetitive transcranial magnetic stimulation, at frontal (F3) or occipital (O1) sites aimed at increasing alpha power elicited simultaneous increases in alpha amplitude across other non-trained sites (Bazanova et al., 2009; Jin et al., 2011; Johnson et al., 2010). Furthermore, the existence of 'flat alpha EEG' suggests evidence of an absence of topographical specificity for alpha amplitude. However, given that low amplitude is a characteristic evident across all EEG sensor locations, a possible cause of the flat alpha EEG is the epigenetic influence of psychological and/or biological factors such as increased anxiety (Ehlers, 2007), age (Basar & Schurmann, 1996, Bazanova, 2008), increased levels of the corticotrophin releasing factor (Enoch et al., 2008), steroid hormones (Asbury et al., 1998, Kamei, 2000), and neuropeptides (Kaur et al., 2007).

3.2.3. The influence of localization

Such data provide some indication that alpha wave functions have no topographical difference. However, we believe that such a conclusion could only be made regarding the alpha waves of a particular frequency. For example, Mizuhara (2012) reported on simultaneous fMRI and EEG measurements taken during a visually guided motor execution task in order to investigate whether the amplitude of the upper alpha rhythm at 11.8 Hz was an indication of sensorimotor activity across the cortex. It was found that the amplitude of this rhythm appeared suppressed not only at the lateral central electrode sites, but also at occipital sites, and this correlated with changes in the fMRI signal in the occipital and the supplementary motor cortices, respectively (Mizuhara, 2012). In addition, Litvak et al. (2011) concluded that frequencies involved in the alpha and beta networks, which are involved in the same attentional and executive functions, and in particular motor planning, have distinct temporoparietal-brainstem networks (Litvak, et al., 2011). The

subthalamic activity was predominantly led by activity in the cortex in both alpha (7–13 Hz) and beta (15–35 Hz) frequency bands (Litvak, et al., 2011). This would suggest that localization may a stronger feature of the upper alpha bandwidth.

In contrast, event related decreases in the power of the upper alpha frequency have been reported over respective areas of the homunculus, indicative of physical movement, whereas event related increases have been observed over surrounding areas and more distant areas that are non-task relevant suggesting a lack of involvement in the movement (Neuper & Pfurtscheller, 2001). It is important to note that this pattern of decreasing/increasing amplitude is specific to the upper alpha frequency range (Neuper & Pfurtscheller, 2001). In a recent study Pfurtscheller et al. (2012) noted that a decrease in the central alpha amplitude, relating to the conscious intention to perform a motor act, was only apparent in subjects with a resting individual alpha peak frequency of >10 Hz (Pfurtsheller et al., 2012). Similar alpha frequency dependent changes have been reported by others (Segrave et al., 2011; Moretti et al., 2011). This suggests that some spatial resolution may take place, but only in the individual upper frequency range of alpha.

3.2.4. Focal amplitude changes reflect selective activation/ inhibition hypotheses

An accepted hypothesis by many is the idea that a decrease in focal amplitude reflects activation of a distinct cortical area whilst an increase in surrounding amplitude denotes inhibition of neighboring cortical areas and that such amplitude changes may well be frequency dependent (Suffczynski et al., 2001; Basar & Schurmann, 1996; Basar, 2006; Klimesch et al., 2007; Tuladhar et al., 2007; Baumeister et al, 2008; Ben-Simon et al., 2008; Del Percio et al., 2011; Avanzini et al., 2012;). This view is encapsulated within the neural efficiency hypothesis discussed below (see section 3.3.1).

3.2.5. Alpha amplitude reflects generalized cortical processes

An alternative view involves the cholinergic system activating oscillations in brain areas that are intimately linked to cognitive function and memory processing (Elmer et al., 2006; Mann et al., 2005; Traub et al., 2005). According to proponents of this approach activating muscarinic receptors induces robust and dynamically complex oscillations in sensory thalamic nuclei which have been taken to suggest that alpha EEG rhythms represent more than a simple measure of idling (Hughes et al., 2011). The principle of individual emerging brain systems, formulated by Bechtereva et al. (2007), suggests that implementation of the same mental activity can be achieved topographically by different brain systems. A similar view is put forward by Cook et al. (1998) based on the correlations between PET and EEG amplitude signals. They have proposed that the functional role of the amplitude (i.e., power) of the alpha waves does not depend on the topographic localization, but mainly reflects generalized cortical processes. Hence, it is possible to conclude that changes in alpha wave amplitude, recorded on the scalp surface and regardless of topography, reflect some generalized cerebral processes, but event related changes in upper frequency alpha power could reflect local distinct cortical processes.

3.3. Alpha amplitude changes are dependent on task engagement

It has been acknowledged that engaging in a task such as perceptual judgment or increased attentiveness leads to a decrease in alpha power (Adrian & Matthews, 1934; Niedermeyer & Lopes da Silva, 2004). This is consistent with the classical view of alpha rhythms which suggests that the amplitude of these oscillations, in terms of cognition, reflects an idling state of primary cortical areas. The idea of increased alpha amplitude as reflecting an idling state is supported by findings of increased alpha power in 8-12 Hz in posterior electrodes when eyes are closed (Treder et al., 2011) and in 9-11 Hz

(Niedermeyer, 2004) as well as 11-13 Hz (Sterman & Egner, 2006) across the motor cortex when limbs are at rest. Nevertheless, there is a long history of research indicating that alpha waves may play an important role in a variety of cognitive processes, including sensory perception and memory (see e.g., VanRullen & Koch, 2003). These are discussed below.

3.3.1 The neural efficiency hypothesis

Over time it has been suggested that upper frequency alpha amplitude is associated with the inhibition of non-essential processing and as such a greater level of alpha amplitude reflects the inhibition of non-essential activity which in turn may facilitate performance on the task (Klimesch et al., 2007). It may also be seen as an index of top-down processing representing a mechanism for increasing the signal to noise ratio within the cortex by actively inhibiting non-essential or conflicting processes (see Cooper et al., 2003; von Stein et al., 2000) as encapsulated within the *neural efficiency hypothesis* (e.g., Doppelmayr et al., 1998, 2005). The idea is that effective cognition is not a function of how *hard* the brain works but rather how *efficiently* it works (Del Percio et al., 2011; Klimesch et al., 2007). In line with this interpretation Tuladhar et al. (2007) have reported findings that suggest that alpha amplitude reflects the disengagement or inhibition of non-essential visual processes to support working memory processes. Further support for this idea comes from literature showing that people classified as more intelligent exhibit greater levels of alpha power in 10-12 Hz compared to those with average levels of intelligence (Basar, 2006; Doppelmayr et al., 2005). Thus, it would seem that those with higher frequency of resting alpha power may be able to utilise this to actively inhibit irrelevant processes, depending on the needs of the task.

Nevertheless, the debate over the precise neural function of the alpha rhythm continues with theories suggesting that an increase in alpha power from 8-14 Hz may

either reflect active processing related to memory maintenance (Palva & Palva, 2007) or the inhibition of posterior regions not required for the task (Basar, 2006; Klimesch et al., 2007).

3.3.2. Eyes open vs. closed

When attempting to interpret alpha wave activity from the amplitude measurement it is important to consider not only anatomical, functional, topographical and psychological factors but also the experimental conditions under which the amplitude is measured. In particular, whether the eyes remain open or closed. According to Barry et al. (2007) the decrease in amplitude seen with eyes open indicates an increasing in activation, whereas closing the eyes leads to an increase in the amplitude indicating less activation. Furthermore, neurofeedback training (NFT) aimed at enhancing alpha power has been shown to elicit benefits in cognition (Ros et al., 2009; Alekseeva et al., 2012) and psychomotor performance (Gruzelier et al., 2009; Bazanova et al., 2009) when the training was conducted with eyes closed. However, eyes open alpha amplitude increasing NFT proved ineffective at helping participants decrease levels of arousal during a stressful situation (Holmes et al., 1980). This may be because when the eyes are open the brain is in a pre-activated condition and as such any subsequent increase in alpha amplitude/power via NFT could in fact lead to a decrease in activation of the brain which in turn may impair cognitive processing/performance (Bazanova & Aftanas, 2010). In contrast, eyes closed NFT aimed at increasing alpha amplitude/power may lead to selective inhibition of non-relevant cognitive activity, that is, improved neural efficiency (Bazanova & Aftanas, 2010).

3.4. Divergent frequency ranges of alpha amplitude

The question of which frequency range belongs to alpha is one of the most important. This lack of standardization when defining the alpha bandwidth has also led to difficulties in the field of neurofeedback (see Vernon et al., 2009). A striking example of divergent interpretations regarding alpha activity due to differing terminology of the various frequency ranges belonging to the alpha rhythm is the so called rolandic rhythm, recorded in the central region which exhibits a decrease in amplitude concurrent with increased cognitive and psychomotor load (Gastaut et al., 1954). This component, sometimes also referred to as the Wicket, or mu rhythm has been reported with varying frequency ranges by different authors from 7-11 Hz (Willemse et al., 2010), around 10 Hz (Lachat et al., 2012), 8-13 Hz (Pineda, 2005), and 6-9 Hz (Marshall et al., 2011). The amplitude of the mu rhythm in human adults is suppressed during both action execution and action observation (Muthukumaraswamy et al., 2004; Perry & Bentin, 2009; Pineda, 2005). This variability in frequency range illustrates a key problem when bandwidths are not identified on the basis of individually determined functions. Moreover it's difficult to understand why a frequency range of 8–35 Hz, that marks Parkinson's disease by excessive amplitude throughout the cortico-basal ganglia network, has been referred to as 'beta' in one investigation (Whitmer et al., 2012) whereas Litvak et al. (2011) propose that the same frequency range, with the same functions should be divided into two sub-ranges: 7–13 Hz for alpha and 15–35 Hz – for beta. Furthermore, Chapman and Lacaille (1999) referred to a frequency range of 4-12 Hz recorded in the central regions scalp as a theta, whilst Moretti et al. (2007) identified the same frequency range as an alpha rhythm. We believe that the arbitrary use of the term alpha frequency range when utilized in this way creates additional difficulties when attempting to understand the phenomenon of alpha activity.

Early proposals by Walter (1963) suggested that the term 'family of alpha rhythms' be used to describe those EEG components that exhibit the effect of suppression in

amplitude in response to motor or cognitive load. However, it is growing increasingly evident that there are at least two independent alpha rhythmical components, often referred to as the lower and upper frequencies, or alpha 1 and alpha 2 sub-bands (Petshe et al., 1997Klimesch et al., 1996; Angelakis & Lubar, 2002; Tenke & Kayser, 2005;Michels et al., 2008).

Thus, the measurement of amplitude within a certain frequency range and without knowledge of the anatomical and physiological characteristics of the organism, regardless of the electrical characteristics of scalp muscles, provides no basis for concluding that it is alpha-wave activity. Such a proposal naturally leads on to a discussion of the individual alpha frequency range.

4. Individual alpha frequency.

4.1. Alpha frequency assessment approaches

Early reports on changes in alpha amplitude, from the pre quantitative EEG era, invariably failed to report alpha peak frequency. Meanwhile the individual alpha frequency could be measured not only by peak frequency, but as the mean frequency in a fixed range or center of gravity in some individual range (Hooper, 2005) and has been one of the most common tools used to study the variability of EEG rhythms among subjects (Creutzfeldt et al., 1976; Kaiser, 2001; Klimesch et al., 1993). In the last 30 years a number of different alpha frequency measurements have appeared, these include: (1) individual alpha peak frequency (IAPF) (Angelakis et al., 2004); (2) mean peak frequency within a fixed bandwidth (Hooper, 2005) and (3) individual alpha peak at the center of gravity within IAF (Klimesch et al., 1993). Comparing these three measurements Hooper (2005) concluded that only the amplitude of IAPF during an eyes closed resting condition reflects the aggregate generation of alpha (Hooper, 2005). Klimesch et al. (1993) made a special study of alpha frequency assessment and compared individual alpha peak frequency (IAPF) with

peak frequency center of gravity (IAF). Their conclusion was that the measurement of the IAF was more valuable when examining event related state alpha frequency changes when eyes are open, whereas the IAPF may be preferred for studying endophenotipic qualities during resting eyes closed sessions (Klimesch et al., 1993).

In an attempt to identify the experimental conditions which would most usefully be used to identify the frequency range of the IAPF and its topography Bazanova (2011) conducted a number of test-retest EEG recordings of 96 male subjects, aged 26-40, over a period of 14-15 days, with participants resting with both eyes closed and eyes open. The EEG was examined using a standard fixed 8-12 Hz band as well as the individually determined alpha band. It appeared that the intra-individual correlation coefficient (ICC) was the strongest in the posterior brain area in the eyes closed condition and determined with the individual alpha band, while it was weakest in the anterior areas in the eyes open condition and defined within the fixed standard 8-12 Hz range (Bazanova, 2011). There was no evidence of a lateralization influence on the mean IAPF in these healthy subjects, something which others have also reported (Bodenmann et al., 2009; Klimesch et al., 1993).

4.2 Inter-individual variability of alpha peak frequency

When Bazanova and Aftanas (2008) compared two groups of healthy male subjects with either low (LAF - IAPF<10 Hz) or high (HAF, - IAPF≥10 Hz. [see Figure 2]) alpha frequency, according to median posterior IAPF when resting with eyes closed (fig.2) they found that the LAF and HAF subjects differed in psychometric strategies for achieving success in nonverbal creative tasks as well as their ability to respond to neurofeedback training. LAF subjects emphasized originality whilst HAF subjects emphasized fluency in reaching the same score of the Torrance test performance (Bazanova & Aftanas, 2008).

……

Figure 2 about here

……

The HAF subjects with highest and LAF subjects with lowest IAPF levels showed the highest originality score in the nonverbal creativity task performance (fig.3). Such findings are consistent with others reporting inter-individual differences in alpha peak frequency correlating with performance on memory (Doppelmayr et al., 2005), IQ (Jausovec & Jausovec, 2000), speed of information processing (Bornkessel et al., 2004), and efficiency of biofeedback training (Alekseeva et al., 2012; Bazanova et al., 2009).

put fig 3 here

4.3. Intra-individual variability of alpha peak frequency

In contrast, many empirical investigations have presented evidence that IAPF varies intra-individually as a function of age, for example increasing through childhood till pubertal age (Bazanova, 2008; Niedermeyer & Lopes da Silva, 2004; Stroganova et al., 1999) and decreasing after 40 years (Clarke et al., 2004; Osaka et al., 1999). A few investigations have also shown that hormonal changes can influence alpha frequency. For instance, there is an increase in IAPF concurrent with enhanced progesterone activity during the menstrual cycle (Creutzfeldt et al., 1976; Solis-Ortiz et al., 2004; Bazanova & Mernaya 2008; Baker & Colrain, 2010) and in conditions when cortisol blood level increases (Tops et al., 2007). In addition, IAPF has been shown to vary with personal cognitive involvement in task performance (Klimesch et al., 2007; Ng & Raveendran, 2007). Good performance is associated with increased IAPF, but a drop in performance and fatigue are related to a decrease in IAPF (Klimesch et al., 1993; Ng & Raveendran, 2007). Alekseeva et al. (2012) also noted that IAPF increased after upper alpha power

neurofeedback training (NFT), though not in all cases. The increase in IAPF was only evident in students with baseline resting alpha frequency lower than 10Hz (LAF subjects), whereas the HAF students did not exhibit any change in IAPF (Alekseeva et al., 2012; Bazanova et al., 2009).

Lebedev (1994; 2006) has proposed that cyclical oscillations in the alpha rhythm determine the capacity and speed of working memory. The higher the frequency the greater the capacity and speed of memory. His hypotheses are supported by the results of a number of empirical studies (Klimesch et al., 1993; Angelakis et al., 2007; Bazanova & Aftanas, 2006, 2008; Bodenmann et al., 2009; Zoefel et al., 2011). Furthermore, Klimesch Doppelmayr, Schimke, and Pachinger (1996) have argued that thalamo-cortical feedback loops oscillating within the alpha frequency range are involved in the search for identification of encoded information. They speculate that faster oscillating feedback loops would correspond to faster access to encoded information (Klimesch et al., 1996).

Later it appeared that whilst some EEG traits for an individual are stable, others are variable between individuals, and moderately to highly heritable (Hodgkinson et al., 2010). Although twin studies (Enoch et al., 2008; Gavrish & Malykh 1994; Smit et al., 2006) have long shown that heritability of EEG amplitude in the waking state is substantial, very little is known about the genes underlying distinct EEG frequency traits.

Thus, it is possible to conclude that distinct behavioral strategies observed in dominant low vs. high alpha peak frequency subjects and different functional appearances of alpha activity in lower and upper frequency sub-bands could be due to genetic and epigenetic factors influencing the individual waking EEG patterns. Such differences could reflect distinct neurophysiological mechanisms of brain activation in both low and high alpha frequency ranges.

4.4. Genetic influence on alpha frequency generation

It is likely that a family of genes, rather than a single gene, correlates with the activity of the EEG alpha rhythm (Lopes da Silva, 1991; Timofeev, 2003). Details on the main ones are examined below.

4.4.1 The frequency of alpha rhythm is determined by the activity of Ca^{2+} T-channels

A distinctive feature of the alpha waves pacemaker, which includes the thalamic relay cells, is the high density of calcium channels in the membranes of T cell surfaces and the endoplasmic reticulum (Destexhe & Sejnowski, 2003; Sherman & Guillery, 1996). The alpha rhythm is a result of tuning the local cortical network, which depends on genetically determined Ca2+ T-channel activity (Lopes da Silva, 1991; Steriade et al., 1990; Steriade, & Timofeev, 2003) and underlies the dominant brain frequency (Jones et al., 2000; Luthi et al., 1998). It was found that the calcium channels of T-type cells, by adjusting their concentration of calcium, can inhibit the activation of signal transmission through the thalamus and thus stabilize the resting state (Page et al., 2006). As shown by recent in-vitro experiments the intracellular calcium current in a thalamic nuclei relay cell of a cat produces a temporary depolarization of the cell membrane at a frequency of approximately 10 Hz, as the refractoriness of activation of calcium channels is approximately 100 ms (Bollimunta et al., 2011; Bright et al., 2007; Hughes & Crunelli, 2005; Hughes et al., 2011). Thus, deletion of the gene in transgenic mice causes a reduction in the refractory period and therefore produces more frequent oscillations (Anderson et al., 2005). Recent studies have demonstrated that knocking out the subunits of metabotropic GABA-B receptors in mice violated the processes of their inhibitory effect on the activity of calcium channels. Hence, there was no refractory period which in turn increased the frequency and disrupted alpha spindle oscillations (Emson, 2007; Winterer et al., 2003). Such experimental data has confirmed Livanov's (1984) conjecture that the organization of rhythmic activity in the brain is caused by the excitation properties of the refractory

calcium current which determines the frequency of the rhythmic discharge of neurons (Eccles, 1994). Recently, Lőrincz, Crunelli, and Hughes (2008) highlighted a subset of thalamocortical neurons in the lateral geniculate nucleus (LGN) of a cat which can exhibit a novel type of intrinsic burst firing at alpha frequencies, termed high-threshold bursting. This activity was unmasked by activation of the metabotropic glutamate receptors (mGluRs) that are postsynaptic to cortico-thalamic fibres (i.e. mGluR1a) (McCormick & von Krosigk, 1992) and could be synchronized by gap junctions to form a local alpha rhythm generator (Hughes et al., 2004).

4.4.2. Synapsin dissociation rate

During an action potential the dissociation rate and dispersion of synapsin from synaptic vesicles controls the rate of vesicle availability for exocytosis at the plasma membrane. Chi Ping et al. (2003) have shown that synapsin dispersion rate tracks the synaptic vesicle pool turnover rate linearly across the frequency range of 5–20 Hz and that the molecular basis for this is in the regulation which occurs at two types of kinases site. Their results show that calcium-calmodulin-dependent kinase sites control vesicle mobilization at low stimulus frequency, while mitogen-activated protein kinase/calcineurin sites are critical at both lower and higher stimulus frequencies. Thus, genetically determined multiple signaling pathways serve to allow synapsin's control of vesicle mobilization over distinct stimulus frequencies.

4.4.3. Catechol-O-methyltransferase (COMT) gene

Early linkage analyses identified a genetic locus on the distal part of chromosome 20q (Anokhin et al., 1992), where the COMT gene is located. Data reported by Bodenmann et al. (2009) demonstrated mechanisms involving a COMT enzyme playing an important role in cortical dopamine metabolism contributing to inter-individual differences

in alpha oscillation frequency, which were functionally related to executive performance. They showed that the functional polymorphism in the COMT gene causes a common substitution of methionine (Met) for valine (Val) at codon 158 of the COMT protein. It was shown that individual alpha peak frequency during the rest condition in Val/Val subjects (i.e., with less dopaminiergic activity) was lower by 1.4 Hz than those in the Met/Met genotype (Bodenmann et al., 2009). Interestingly it has also been shown that the relationship between dopamine projection neurons firing at a low-frequency and GABA projection neurons firing at a high-frequency (Ding et al., 2011) directly influences the number and/or strength of thalamo-cortical connections (Thatcher et al., 2009).

Thus, it is now evident that alpha peak frequency reflects individual genetic influences on the underlying neural mechanisms of the generation of alpha activity (Lopes da Silva, 1991; Steriade et al., 1990, Steriade & Timofeev, 2003). Meanwhile, with regards to the question of whether smarter brains run 'faster' Posthuma et al. (2001) have concluded that both peak frequency and the dimensions of IQ are highly heritable, ranging from 66% to 83%. Nevertheless, a large part of the genetic variance in alpha peak frequency, as well as in working memory and processing speed, may be due to non-additive factors such as activation (Li et al., 2011) or EEG voltage (Arns et al., 2008). As such, there may be additional EEG indices predicting cognitive ability (Posthuma et al., 2001), that could be connected with the pattern of EEG activation (Cho et al., 2008; Tenke & Kaiser, 2004).

4.5. Non-additive factors influencing the alpha frequency

The corticotrophin releasing hormone-binding protein (CRF-BP) gene has attracted the attention of researchers interested in finding a suitable candidate gene for inheritance of alpha EEG activity. This is because CRF-BP is the major hypothalamic releasing factor for pituitary adrenocorticotrophin secretion and acts as a neurotransmitter, or neuromodulator,

at other sites in the central nervous system as well as being a primary mediator of the neuroendocrine stress response. It has been shown that CRF-BP modulates both the power and frequency of alpha oscillations (Enoch et al., 2008; Winterer et al., 2003). For instance, Enoch et al. (2008) identified *CRH-BP* as a strong candidate gene associated with the production of alpha peak frequency.

More recently Hodgkinson et al. (2010) performed a whole-genome association study on alpha, beta, and theta EEG power in a Native American cohort of 322 individuals to take advantage of the genetic and environmental homogeneity of this isolated population. They identified three genes: (1) SGIP1 (SH3-domain of Growth factor receptor-bound protein 2 interacting protein 1) which functions as an endocytic protein that affects signaling by receptors in neuronal systems involved in energy homeostasis via its interaction with endophilins (Uezu et al., 2007). The increase in endophilin levels in neurons is linked to an increase in the activation of the stress kinase (Ren, et al., 2008) and SGIP1 was estimated to account for 8.8% of variance in 4-8Hz power; (2) gene ST6GALNAC3 which belongs to a family of sialyltransferases that transfer sialic acids from CMP-sialic acid to terminal positions of carbohydrate groups in glycoproteins and glycolipids to provide energy (Lee et al., 2000). The ST6GALNAC3 gene has been associated with alpha power (Hodgkinson et al., 2010); (3) power in the alpha range has also been associated with the UDP-glucose dehydrogenase gene (Hodgkinson et al., 2010). UDP-glucose dehydrogenase belongs to the family of oxidoreductases and is an integral Golgi membrane protein whose expression is up-regulated in response to hypoxia, a risk factor for schizophrenia *(*Bauer et al., 1975). Interestingly Hodgkinson et al. (2010) have demonstrated that the ST6GALNAC3 gene overlaps with findings for theta (4-8Hz) and alpha (8-13 Hz) power-associated markers, both of which lie within the third intron of ST6GALNAC3. Such findings provide evidence that many, if not all, of the genes identified here are associated with activation processes. Hence, it can be assumed that if

Hodgkinson et al. (2010) had not considered the correlation with individually defined alpha frequency ranges, this overlap may not have been detected.

Recently Ben-Simon et al. (2008) combined fMRI and EEG to examine two parallel patterns of alpha modulations and explore their anatomical basis in the human brain. Their findings suggest that the human alpha rhythm represents at least two simultaneously occurring processes which characterize the resting brain. The first is related to expected change in sensory information, while the second is endogenous and independent of any stimulus change. (Ding et al., 2011) Nonetheless, the exact mechanisms for generating an oscillation may differ widely between the different alpha frequency waves depending on individual network properties, cell types, cell physiology, hormone level and blood feeding.

Thus, it could be proposed that differences in alpha peak frequency in the resting condition reflect an endophenotypic trait indicative of distinct mechanisms of brain activation and alpha wave generation.

5. Alpha activation or the Berger effect

5.1. Magnitude of alpha amplitude suppression

The alpha rhythm is one of the main EEG rhythms which has a well-defined physiological property, that is, the suppression of amplitude in response to opening the eyes or increasing cognitive load. Obviously, some have used the amount of alpha suppression as an index of cortical activation (Barry 2007; Cho et al., 2008; Laufs et al., 2006; Schimke et al., 1990). This suppression, or ‘Berger effect’, might explain the large inter-individual variability in the power and frequency of the alpha rhythm (Kirschfeld, 2005). Using alpha amplitude suppression as a measure of activation the ‘magnitude’ of such a decrease has recently been explored during visual and cognitive processing proving a promising avenue of study in the search for putative endophenotypes (Loo et al., 2010) as

well as helping to identify individual cognitive strategies (Ivanitsky et al., 2009; Loo & Smalley, 2008). For instance, it has been shown that the magnitude of activation in patients with cognitive impairment (Alexander et al., 2006), impaired response times (Vaez Mousavi et al., 2007), spinal cord injury (Thuraisingham et al., 2007) and attention processing deficits (Barry et al., 2003) was decreased in comparison with healthy able-bodied participants. Moreover, examination of predictors such as lack of reactivity to opening eyes was found to be highly sensitive to predicting poor outcome (Zhang et al., 2011).

The magnitude of alpha amplitude suppression in response to action perception and production appears to be smaller for infants than for adults and older children, suggesting developmental changes (Marshall et al., 2011; Stroganova, Orekhova & Posikera, 1999). However, in contrast Doppelmayr et al. (2005) have shown decreased task related alpha suppression in intelligent participants in response to easy mental tasks.

Hence, alpha suppression has been associated with both age and cognitive performance. Del Percio et al. (2011) also found that the reactivity of alpha rhythms to eyes opening is lower in athletes than non-athletes. In contrast, Pfurtscheller and Lopes da Silva (1999) proposed that the level of amplitude suppression would correlate with different EEG components across distinct alpha frequency peaks and that with an increasing number of interconnecting neurons amplitude increases whilst frequency decreases (Pfurtscheller & Lopes da Silva,1999). This may indicate that higher amplitude in eyes closed resting condition predicts a greater level of brain activation.

According to Cook et al. (1998) a reduction in EEG alpha amplitude occurs simultaneously with an increase in the amplitude of a PET signal. This suggests a relationship between changes in alpha activation and changes in metabolic intensity. A possible addition to this view could be made based on the findings of the hormonal influence on the EEG (Bazanova & Mernaya, 2008; Mantanus et al., 1988). For instance,

Bazanova and Mernaya (2008) found a negative relationship between the magnitude of activation and the cyclic change in saline progesterone concentration in women. That is, greater activation in the low alpha frequency range during the follicular phase (i.e., low progesterone level and low alpha peak frequency), and less activation in the luteal phase (i.e., increased progesterone level along with increased individual alpha peak frequency and cognitive efficiency) (Bazanova&Mernaya, 2008). Hence, in the same women lower alpha frequency is related to high activation, but higher alpha frequency is associated with lower activation. In addition, Jann et al. (2010) have put forward the idea that subjects with higher alpha frequency are able to pre-activate task-relevant networks and are thus more efficient in executing the task and show a reduced fMRI-BOLD response to the stimulus. However, this reduction in blood flow is not because the absolute amount of activation is smaller but rather due to the idea that the additional activation resulting from processing of external input is limited due to a higher resting baseline (Jann et al., 2010). These finding may be explained by the data reported by two independent research groups who have found that 8 Hz and 10 Hz oscillations respond differently to visual stimulation (Hanslmayr et al., 2007; Mazaheri & Jensen, 2010). Hence, it is possible to conclude that the magnitude of alpha amplitude suppression could reflect the activation of the brain in response to a visual or cognitive load and as such may also depend on the frequency range.

5.2. Individual alpha bandwidth

It has been argued that the correct evaluation of alpha activation should be based on an individually determined alpha band (Bazanova, 2011). The argument here is that when comparing the two methods of analyzing alpha suppression, those alpha desynchronization values that have been calculated using an individually determined alpha-band may be superior when compared to those using a fixed standard band, particularly when attempting to differentiate inter-individual differences (Bazanova, 2011; Schimke et al., 1990). It is

possible that using a fixed frequency band may blur the real alpha peak, masking the age- or functions related modifications. Thus, alpha measures are influenced by the boundaries chosen for the frequency band. Yet no definitive division of the human EEG frequency range has been found. More than 20 arbitrary frequency boundaries have been specified in the literature for studying the alpha rhythm (e.g., 7.81-14.06 Hz, 7.03-12.89 Hz, 8-15 Hz) (Etevenon et al., 1989; de Toffel & Autret, 1991; Moretti et al., 2004 respectively). Lack of standardization in specifying the alpha frequency band fosters confusion between laboratory findings, but may be required due to the range of variables addressed by quantitative EEG. Moreover, it has been suggested that defining alpha power using a fixed bandwidth is likely to reduce experimental sensitivity and increase the chance of error (Klimesch et al., 1993; Bazanova & Aftanas, 2008; Kaiser, 2001; Bazanova, 2011; Segrave et al., 2011). In a dual EEG-fMRI investigation Laufs et al. (2006) showed that spontaneous reductions in alpha amplitude were associated with increased cognitive activity associated with general activation of the brain across a wide (i.e., not only 8-12Hz) spectral frequency range. Several approaches have been suggested for distinguishing between individually based lower and upper frequency boundaries of the alpha band. These include; (1) those based on peak frequency (Angelakis et al., 2006, Segrave et al., 2011); (2) those utilizing an extended 5-14 Hz alpha band (Moretti et al., 2011) and (3) the use of transition frequency methods (Doppelmayr et al., 1998; Bazanova & Aftanas, 2008). Accordingly, Klimesch's method was to use the center of gravity or individual alpha frequency as an anchor point for distinguishing between a lower and an upper alpha band (Klimesch et al., 1997). Although this method proved superior to the use of fixed frequency bands, the question remains as to whether the bandwidth may be considered a constant value that does not vary. Obviously the plus or minus 2-2.5 Hz in association with the peak alpha frequency is the type of pragmatic decision that is often seen in psychophysiology, based on both empirical data and ease (Klimesch et al., 1997). But, it is

known that some subjects will have a narrow dominant frequency range others might hit the mark exactly and a third group have a wider frequency range (Sterman, 1996; Thatcher, 1998). It is possible that by refining the formula to include a mixture of percent attenuation and topography might produce a truly customized dominant frequency bandwidth. This could then be used as a more accurate anchor point and enable researchers to move outwards towards other bandwidths of interest. However, as Goljahani et al. (2011) point out, techniques for individual alpha frequency range determination can be over-reliant on the presence of peaks in the EEG spectrum and are based on qualitative criteria that require visual inspection of every individual EEG spectrum, a task that can be both time consuming and difficult to replicate. Such issues led Goljahani et al. (2011) to propose a method for identifying the individual alpha frequency center of gravity based on channel reactivity to activation. This method utilizes quantitative indices and relies on task-specific alpha reactivity patterns rather than on the presence of specific peaks in the EEG spectrum. For instance, Bazanova and Aftanas (2006) defined the individual alpha band width as the frequency range that encompasses the part of the EEG spectrum which shows suppression of amplitude by at least 20% in response to opening the eyes compared to eyes closed (fig. 4)

put fig.4 here

Furthermore, it has been shown that the individual alpha band width (IABW) can vary in accordance with brain activation (Kaiser, 2005) and efficiency of cognitive performance (Bazanova & Aftanas, 2008). Narrow in the less academically successful student and wider in the more successful student (Bazanova & Aftanas, 2008). For example, when completing a musical performance the IABW is wider for highly-skilled professionals than for those with low musical skill (Bazanova et al., 2003). It has also been shown to be wider in those with higher creativity as assessed by the Torrance creativity

coefficient (Bazanova & Aftanas, 2008) and the width also correlates positively with biofeedback training efficiency (Bazanova et al., 2009). Additionally, it has been shown that individual alpha bandwidth (IABW) is dependent on age – increasing from 3 to 20 years (Bazanova, 2008). It has also been reported that women in the follicular phase of the menstrual cycle have a narrower alpha band than men (Bazanova & Mernaya, 2008).

Hence, the benefits of utilizing an individual alpha band width measurement are evident. Furthermore, this would suggest that a key aspect of alpha wave activity is that it can be assessed not only by the level (i.e., amount) of amplitude suppression but also by the width of the frequency range that such suppression occurs in. As such, the magnitude of alpha suppression taken together with individual alpha band width could be used not only as a characteristic of brain activation but as an index of the neuronal generators used in cognitive processes.

Thus, in spite of a well described traditional approach to defining alpha activity as well as the more individually tailored recent attempts the relationship between alpha activity and activation remains a matter for debate (Toscani et al., 2010). There are some questions which it may not be possible to answer with a simple spectral analysis. For example, it is not clear: (1) whether a change in total power of particular alpha oscillations results in a change in the number of occurrences per minute rather than a change in the average amplitude of oscillation, and (2) whether change in the total power of alpha oscillations affects the whole analyzed signal or only a small portion (Kaplan et al., 2002). Thus, regardless of how powerful or statistically significant the different estimations of averaged EEG effects may be, it is difficult to make meaningful interpretations if the estimations are not linked to the specific structure of the EEG (Towers & Allen, 2009).

6 The segmental structure of the alpha waves

Since oscillatory phase at a given frequency reflects the cyclical fluctuations of a network's excitability that occurs on much shorter timescales than variations in oscillatory power at the same frequency (Klimesch et al., 2007; Lakatos et al., 2008; Rajkai et al., 2008) phase effects may provide deeper insights into the fine-grained coding of sensory information processing (Oprisan et al., 2004). So the phase modulation process could characterize another unique alpha wave functional trait which has been referred to as excitability cycles (Hughes et al., 2011), alpha bursting segments (Kaplan et al., 2003), operational architectonics of brain functioning (Fingelkurts & Fingelkurts, 2006), pulsed inhibition (Jensen & Mazahery, 2010; Mathewson et al., 2009; VanRullen & Koch, 2003), and spindle-form segments (Livanov,1984) that act within a temporal frame which reduces the processing capabilities of a given area (Jensen & Mazahery, 2010). Ultimately, such spindle-form segments are considered to be a potential basis for explaining discrete processing in the brain or its auto-rhythmicity (Livanov & Dumenko, 1987). Over time it has been proposed that oscillatory alpha activity operates in a spindle-form manner (Jensen & Mazaheri, 2010; Livanov & Dumenko, 1987; VanRullen & Koch, 2003).

To overcome the limitations of conventional spectral analysis based on averaging procedures and to reveal both the dynamic and temporal characteristics of alpha activity an entire set of individual short-term stationary EEG segments may need to be obtained (Kaplan, 1999; Mazaheri & Jensen, 2010; Towers & Allen, 2009). Non-stationary phenomena are present in the EEG, usually in the form of transient events, such as relatively alternative homogenous intervals (i.e., bursting segments) with different statistical features (e.g., amplitude or variance) (Lopes da Silva, 1991; Simon et al., 2011). The idea that alpha oscillations have a spindle-like form only during sleep (Niedermeyer, 1999) has been contradicted by the findings of Simon et al. (2011) and Kellaway (2003), who have described the so-called lambda waves (8-13 Hz). This wave is believed to represent alpha spindle-form oscillations. Furthermore, Kellaway (2003) has proposed that

the physiological basis of sleep spindles is probably very similar to lambda and alpha waves. Simon et al. (2011) demonstrated that alpha spindles are superior to EEG band power measures for assessing driver fatigue under real traffic conditions (Simon et al., 2011).

To determine whether the activation state of the brain would modulate the composition of alpha spatial microstates (i.e., spindles) Cantero et al. (2004) used spatial segmentation methods to show that the mean duration of alpha spindles is longer in relaxed wakefulness than in drowsy periods and REM sleep, and that the number of different amplitude values are more abundant in drowsiness than in other brain states.

6.1. Physiological mechanisms that serve to provide the spindle-form of alpha oscillations

Firstly, spindle like segments could be associated with short- and mid-term synaptic plasticity (Steriade & Timofeev, 2003). In addition, Luthi et al. (1998) have shown that the blocking of Ca2+ oscillations is associated with inhibition of the spindle wave refractory period such that continuous 6-10Hz oscillations were generated throughout the network. A probable molecular mechanism for this phenomenon was proposed by Destexhe and Sejnowski (2003). They suggested that spindling may activate the protein kinase A molecular gate, thus opening the door for gene expression and allowing long-term changes to take place following subsequent inputs (Destexhe & Sejnowski, 2003). Furthermore, it is possible that rhythmic GABA-ergic input from the inter-neuronal network is a key mechanism for producing the ‘pulsed inhibition’ or spindle-form segments. For instance, GABA-ergic feedback from interneurons has been strongly implicated in the physiological mechanism(s) generating the alpha rhythm (Jones et al., 2000; Lorincz et al., 2009). More recently Hughes et al. (2011) highlighted a subset of thalamocortical neurons that can

exhibit a type of intrinsic burst firing at frequencies termed high-threshold bursting (Hughes et al., 2004).

6.2 Measurement of the microstructure of the spindle-form segments

Recent work has challenged the dogma that ongoing activity can simply be averaged out across trials (Mazaheri & Jensen, 2010). The key aspect of this research was the revelation that the ongoing activity in the frequency of 10 Hz (i.e., alpha) contains a non-sinusoidal property referred to as amplitude asymmetry or baseline shift. Mazaheri and Jensen (2008) propose that the amplitude modulations of the oscillatory activity are asymmetric, such that the peaks are more strongly modulated than the troughs. In this study, a measure referred to as the Amplitude Fluctuation Asymmetry Index (AFA-index) was developed to quantify the asymmetry of amplitude fluctuations. The AFA-index compares the variance of the peaks with the variance of the troughs by considering the normalized difference between the two measures. Using this AFA-index Mazaheri and Jensen (2008) were able to show that the direction (i.e., stronger modulation of peaks than troughs or *vice versa*) and magnitude of the AFA-index during a resting condition correlated respectively with the amplitude and polarity of slow ERPs in response to simple visual stimuli.

The other trait of the spindle-form segment microstructure is average amplitude (i.e., μV) within a segment which indicates the volume of the neuronal population (Kaplan et al., 1999; Lopes da Silva, 1991). Indeed, the more neurons recruited into an assembly through local synchronization of their activity the higher will be the oscillation amplitude of the corresponding assembly (Kaplan et al., 1999; Livanov & Dumenk 1987; Lopes da Silva, 1991).

Average spindle lifetime represents the functional lifespan of the neuronal population or the duration of operations produced by such a population (Kaplan et al.,

2002). It has been shown that longer spindles indicate a more relaxed state (Huupponen et al., 2008). In addition, the lifetime of the spindle-form segment is correlated with fluency in cognitive task performance (Bazanova & Aftanas, 2008; Maltseva & Masloboiev, 1997) and efficiency in biofeedback training (Bazanova et al., 2007; 2009). Interestingly the shortest alpha segments belong to HAF subjects with the highest individual alpha peak frequencies and LAF subjects with the lowest individual alpha peak frequencies (Bazanova & Aftanas, 2008). Hence, the longest spindle-form segments belong to individuals with an average, or approximately average, 10Hz individual alpha peak frequency. It could be speculated that the different neural mechanisms producing the spindle formation in LAF and HAF subjects is due to the distinct patterns of the spindle-forming mechanism displayed by thalamocortical neurons (Brown et al., 1993). Indeed, Fuentealba et al. (2005) have shown that the reticular neurons display membrane bi-stability as indicated by two discrete electrical potential modes, with differential responsiveness to cortical inputs. Additionally, in vivo (Steriade & Llinas, 1988; Steriade & Timofeev, 2003) and in vitro (Bal et al., 1996) intracellular studies have revealed at least two distinct patterns during spontaneously occurring spindle-form waves, which may be related to the actions exerted by non-bi-stable and bi-stable neurons, respectively. Indeed, non-bi-stable neurons fired stronger bursts with higher intra-burst frequencies, which are assumed to generate inhibitory postsynaptic potentials of around 7–10 Hz. In contrast, these potentials with a lower amplitude and a higher frequency are likely to be generated by single action potentials, as they occur during the depolarizing plateau in bi-stable cells (Fuentealba et al., 2005). If we assume that longer spindles of stable brain activity imply less information to process (as reflected by higher stability of the brain generator) and shorter segments imply a higher number of brain microstates, caused by an increased number of steps of information processing, it is possible to suggest that the intra-segment alpha amplitude variability could be indexing phase resetting activity (Oprisan et al., 2004). Indeed, intra-

spindle segment amplitude variability decreases in coma or stupor (Brenner, 2005), but has been shown to increase during cognitive loading (Kaplan & Borisov, 2003) and generally increases as a function of age (Bazanova, 2008; Thatcher et al., 2008). To some extent this may reflect the ability for self-control which develops with age (Mischel, 2004; Orekhova et al., 2003). Hence, amplitude variability, which is associated with phase resetting intensity (Oprisan et al., 2004), may reflect the engagement of cognitive control mechanisms (Hanlsmayr et al., 2005; 2007; Lebedev 1994; Livanov & Dumenko, 1987).

Thus, the experimental results indicate that alpha spindle-form segments are the product of the dynamics of neuronal assemblies in the underlying cortex (Dorokhov, 2003; Lehmann et al., 1994, Singer et al., 1997). These bursting segments play an inhibitory role in delaying the rhythmic waves generated in the thalamus for the self-control of brain and mind (Eccles, 1994; Livanov, 1984; Livanov & Dumenko, 1987), and are essential for memory formation (Lebedev, 1994; 2006) and perceptual processing (Jensen & Mazaheri, 2010).

7. Conclusion

In this review we have assumed that alpha activity phenomena involves; (1) individual spectral alpha peak frequency, (2) power within an individually determined alpha range, (3) the level of alpha amplitude suppression in the individual alpha frequency range and (4) micro structural characteristics of spindle-shaped bursting segments. An historical reflection has shown that the measurement of alpha EEG oscillation activity involves an assessment not only of the amplitude, because of varying anatomical and physiological factors, but also the frequency and phase. In addition, interpretation of alpha activity exclusively in terms of changes in amplitude is also somewhat limited because it is necessary to take into account the variability of topographical factors. With this in mind the review provides information that topographical variability may occur, or not, depending on the frequency range within which

amplitude is measured. As such, it seems that alpha activity as measured by amplitude does not depend only on the topographic localization, but also reflects generalized cortical processes. The most probable reasons why alpha amplitude as a measurement may not be the sole criterion of alpha wave activity are dependent on the level of engagement of a task and the divergent frequency ranges with which amplitude is assessed. Divergent interpretations of the change in alpha amplitude could be related to different hypotheses regarding the neuronal mechanisms generating alpha rhythms. It was demonstrated that certain thalamic nuclei have a strong influence in determining the magnitude of alpha power at the cortex. Furthermore, early research led to two basic assumptions regarding alpha that are still valid today: (1) that cortical alpha is modulated by a thalamo-cortico-thalamic re-entrant network, and (2) that alpha is not a unitary phenomenon, rather it is comprised of different oscillations with different frequencies across a broad range.

Hence, we concluded that analysis of EEG alpha activity should include amplitude alongside two other important physical characteristics: frequency and phase resetting of alpha oscillations.

Emerging research has provided evidence that the alpha frequency range as measured using alpha peak frequency reflects the influence of individual genes on the underlying neural mechanisms generating alpha activity (Hughes et al., 2011; Lopes da Silva, 1991; Steriade et al., 1990; Steriade & Timofeev, 2003). We discussed the possibility that several factors were common in the generation of different types of oscillations. For instance, intra-individual variability in alpha peak frequency provides a mechanism for searching and identifying encoded information (Klimesch et al., 1993; Angelakis et al., 2007; Bazanova & Aftanas, 2006, 2008; Bodenmann et al., 2009; Zoefel et al., 2011). Hence, by examining peak alpha frequency it may be possible to understand not only why it appears but also what mechanisms mediate its variability.

Two alpha frequency patterns have been presented in this review indicating that the human alpha rhythm represents at least two simultaneously occurring processes characterized by the expectation of change in sensory information and an endogenous rhythm that is independent of stimulus change. Hence, we have argued that brain activation should depend not only on changes in amplitude but essentially on where such changes occur across the frequency spectrum. Together with the magnitude of suppression, individual alpha band width could be used as a characteristic of brain activation and consequently as an index of various neuronal generators included in activation.

According to the time inhibition theory (Klimesch et al., 2007) the active role of alpha waves is seen as a mechanism that may also underlie the functional role of other oscillations (Klimesch et al., 2007; Mazaheri & Jensen, 2010). Synchronization in the alpha frequency range helps neurons in distributed networks to effectively activate common target cells (Basar, 2006; Klimesch et al., 2007). This alpha-frequency dependent mechanism plays an important role in the top-down control of cortical activation and excitability. Hence, the brain is organized into dynamic functional networks and activity within one of these, such as the default network, can be dissociated from that in other task-specific networks. This would suggest that all brain networks may be structurally connected but only transiently connected functionally. One hypothesis as to how such transient functional coupling occurs is that network formation and dissolution is mediated by increases and decreases in different frequency oscillatory synchronization. So the phase of low frequency electrophysiological oscillations is coupled to high gamma (80-150 Hz) amplitude, which suggests that low-frequency oscillations modulate local cortical activity (Doesburg, Vinette, Cheung & Pang, 2012).

As such, it may be concluded that alpha oscillations play an active role in cognitive processing and self-regulation, though it may be that such oscillations are frequency dependent. Hence, the neuronal activation strategies for achieving enhanced alpha wave

activity during biofeedback training may be different according to the individual alpha frequency.

Conflict of Interest Statement

The authors declare that the research was conducted in the absence of any commercial or financial relationship that could be construed as a potential conflict of interest.

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