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Review

EEG delta oscillations as a correlate of basic homeostatic and motivational processes

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ABSTRACT

Functional significance of delta oscillations is not fully understood. One way to approach this question would be from an evolutionary perspective. Delta oscillations dominate the EEG of waking reptiles. In humans, they are prominent only in early developmental stages and during slow-wave sleep. Increase of delta power has been documented in a wide array of developmental disorders and pathological conditions. Considerable evidence on the association between delta waves and autonomic and metabolic processes hints that they may be involved in integration of cerebral activity with homeostatic processes. Much evidence suggests the involvement of delta oscillations in motivation. They increase during hunger, sexual arousal, and in substance users. They also increase during panic attacks and sustained pain. In cognitive domain, they are implicated in attention, salience detection, and subliminal perception. This evidence shows that delta oscillations are associated with evolutionary old basic processes, which in waking adults are overshadowed by more advanced processes associated with higher frequency oscillations. The former processes rise in activity, however, when the latter are dysfunctional.

Contents

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1. Introduction

Some time ago electroencephalogram (EEG) was considered to be useful only for making inferences about global states of sleep and wakefulness (e.g. Duffy, 1962; Thayer, 1989). The recent resurgence of interest in neuronal oscillations is a result of several developments, which showed that mammalian cortical neurons form behavior-dependent oscillating networks which bias input selection, temporally link neurons into assemblies, and facilitate synaptic plasticity (Buzsaki and Draguhn, 2004). The synchronous activity of oscillating networks is now viewed as the critical "middle ground" linking single-neuron activity to behavior. Growing body of evidence suggests that different levels of cerebral integration mediated by spatial and temporal synchrony over multiple frequency bands could play a key role in the emergence of percepts, memories, emotions, thoughts, and actions (Cantero and Atienza, 2005; Nunez, 2000; Varela et al., 2001). This understanding implies that different frequency oscillations must be associated with different processes.

Ongoing and event-related oscillations are usually categorized into five frequency bands: delta (0.5–3.5 Hz), theta (4–7 Hz), alpha (8–12 Hz), beta (13–30 Hz), and gamma (>30 Hz). These five bands could be meaningfully divided into two categories. Delta, theta, and alpha oscillations are examples of the so-called global processing modes which span relatively large cortical regions and have been hypothesized to serve the purpose of integration across diverse cortical sites by synchronizing coherent activity and phase coupling across widely spatially distributed neural assemblies. Oscillations of beta and gamma ranges, or local EEG modes, are higher in frequency, lower in amplitude, and distributed over a more limited topographic area (Nunez, 1995). It is suggested that dynamic coordination at a timescale of hundreds of milliseconds (such as delta oscillation) may be essential for optimization of distributed representations in the brain, whereas patterns of synchronous local interactions may be coordinated dynamically on a faster timescale. Processes occurring at these two timescales can mutually constrain each other through mechanisms of time-dependent plasticity and experience-dependent consolidation of architectures selected by synchronization (Engel et al., 2010). Fast oscillations (beta and gamma) are essential for coordination of computations in tens of millisecond range in specific cognitive processes. Slow oscillations modulate fast oscillations on a higher level of hierarchy and thus determine the general mode of processing. Highly localized computations may be able to oscillate at higher frequencies while more complex, integrative, or inherently slower computations may result in slower oscillations (Engel et al., 2010). Therefore, oscillations are hierarchically organized with slower oscillations being at higher levels of the hierarchy (meaning they are linked to more basic and general classes of processes).

A large body of evidence suggests that oscillatory activity in all frequency bands is linked to a broad variety of perceptual, sensorimotor, and cognitive operations (Singer, 1999; Basar et al., 2000; Klimesch, 1996; Palva and Palva, 2007). This evidence, however, yields a rather complex picture. While it provides clear cases of task- or context-related modulation of different frequency bands, it does not yet allow making conclusions regarding functional significance of these bands. One way to approach this difficult question would be from an evolutionary perspective (Knyazev and Slobodskaya, 2003; Knyazev et al., 2004). Such approach is inevitably speculative, but having considerable explanatory value it allows unifying data coming from different research domains. The evolutionary interpretation of brain oscillations mostly considers the global mode oscillations of delta, theta, and alpha ranges. It suggests that these three oscillatory modes are associated with brain systems that have different significance at different evolutionary stages. Specifically, delta oscillations are associated with the most ancient system, which was dominant in the brain of lower vertebrates (reptilian, amphibian, and fish). Theta oscillations dominate in lower mammals. Alpha oscillations are associated with the most advanced system, which dominates in adult humans (Knyazev and Slobodskaya, 2003). This idea is clearly reminiscent of the well known triune brain concept by Paul MacLean (MacLean, 1990), but Knyazev et al. (2004) emphasized that the triune brain concept was used more as a metaphor and the three oscillatory systems do not have to exactly correspond to the anatomical borders of the three brains of the MacLean's model. This review will concentrate on delta oscillations. First, predictions derived from the evolutionary point of view will be formulated. Next, available empirical evidence will be matched against these predictions. But I begin by discussing the viability of the triune brain concept.

1.1. MacLean's triune brain concept

MacLean originally formulated his model in the 1960s and propounded it at length in his 1990 book (MacLean, 1990). It posits that evolutionary processes have endowed humans with essentially three brains: the reptilian complex, the paleomammalian complex, and the neomammalian complex, viewed as structures sequentially added to the forebrain in the course of evolution. The reptilian complex was the name MacLean gave to the set of brain structures comprising the brain stem including much of the reticular system and basal ganglia. MacLean contended that the reptilian complex was responsible for species typical instinctual behaviors involved in aggression, dominance, territoriality, and ritual displays. The paleomammalian complex consists of the septum, amygdala, hypothalamus, hippocampal complex, and the cingulate cortex. Most of these structures are parts of the so-called limbic system (the term coined by MacLean), and MacLean maintained that these structures were responsible for the motivation and emotion involved in feeding, reproductive behavior, and parental behavior. The neomammalian complex consists of the cerebral neocortex. MacLean regarded its addition as the most recent step in the evolution of the human brain, conferring the ability for language, abstraction, planning, and perception. Subsequent findings have invalidated the traditional neuroanatomical ideas upon which MacLean based his hypothesis. The model of evolution characterized by accretion of parts that MacLean proposed has been entirely replaced by another model that emphasizes conservation of fundamental segmental structure (Rubenstein et al., 1994, 1998). For instance, most areas of neocortex (the neomammalian complex in the MacLean's model) have homologues in amphibian and other vertebrate brains, suggesting that the mammalian neocortex was not added like icing onto an already baked cake, but was radically transformed from a series of precursors (Striedter, 2005). Moreover, Striedter (2005) observes that "brains are like companies – they must reorganize as they increase in size in order to stay functional" (p. 127). Firstly, the brain's average connection density must decrease with increasing brain size; otherwise the number of axons would increase explosively with neuron number, racking up enormous costs in terms of space and metabolic energy (Striedter, 2005). This decrease in average connection density implies that brains become more modular, both structurally and functionally, as they increase in size (Jacobs and Jordan, 1992). Local connections alone place major constraints on global synchrony in growing brains. The densely connected local neuron networks are supplemented by a small fraction of long-range connections (Braitenberg and Schutz, 1998). Despite the progressively decreasing fraction of long-range connections in larger brains, synchronization of local and distant networks can be readily accomplished by oscillators because of the low energy costs involved in coupling rhythms (Buzsaki and Draguhn, 2004). That means that significance of oscillations must increase in large brains. Available evidence shows that

this is indeed the case. Invertebrates have much more obvious unit spiking than vertebrates, but much less relative amplitude of slow (<50 Hz) waves (Bullock, 1993). EEG-like activity could be recorded in the octopus brain but not in other, less developed invertebrates (Bullock, 1984; Bullock and Basar, 1988). Among vertebrates the degree of synchronization also increases during evolution. There is evidence of less synchrony or more rapid coherence decline with distance in reptiles, amphibians, and fish than in mammals (Bullock, 1997, 2002).

Secondly, the principle 'late equals large' causes late-born regions, such as the neocortex, to become disproportionately large as absolute brain size goes up (Striedter, 2005). As brain regions increase in absolute and/or proportional size, they frequently change in internal organization. Another principle that helps explain some variation in neuronal connectivity is Deacon's (1990) displacement hypothesis, which Striedter (2005) calls the rule of 'large equals well-connected.' It holds that, whenever a brain region increases in proportional size, it tends to receive more inputs and project to more targets than it did ancestrally. The human neocortex projects directly to and throughout the brain stem and spinal cord (Barret et al., 2007). As a result, humans (and other great apes) have greater direct and indirect cortical control over the subcortex and spinal cord than do rats, allowing greater autonomic and behavioral diversity and flexibility.

All these recent developments largely invalidate evolutionary ideas that were put in the base of the MacLean's triune model. However, this model continues to hold interest for some psychologists and members of the general public because of its focus on the recognizable differences between most reptiles, early mammals, and late mammals. Reasons for the success are its simplicity; the theory in this form recognizes three major evolutionary periods in the development of the brain that are characterized by three recognizably distinct ways of solving adaptive challenges. The three functional domains described by the original triune model, wherever located, remain useful organizing themes and concepts. Indeed, it is difficult to deny that evolution tends to preserve devices that proved to be useful at a certain stage, and late-born more advanced devices do not usually replace the old ones but coexist with them. Thus, neurobiologically, mammals are distinguished mainly by their neocortex, which has non-mammalian precursors but is highly modified and genuinely new. Looking beyond the neocortex, we find that mammal brains are similar, though not identical, to reptile brains (Striedter, 2005). These 'old' devices that are preserved in the mammal brain continue to do their job in much the same way they did it in lower vertebrates, though closely supervised by advanced 'new' devices. Much evidence shows that late-born advanced brain mechanisms are last to appear and first to extinct in the course of individual development and are more vulnerable to detrimental environmental influences. As Jackson (1958) once famously noted, "The higher nervous arrangement inhibit (or control) the lower, and thus, when the higher are suddenly rendered functionless, the lower rise in activity". Hence, although modern advances in the field of evolutionary neuroscience show that the general picture is much more complicated than perhaps John Hughlings Jackson and even Paul MacLean once described, their core idea about coexistence in the human brain of functional domains that in the evolution were associated with distinct ways of solving adaptive challenges is still viable and productive.

1.2. Delta oscillations from an evolutionary perspective

The most important difference between the MacLean's model and the evolutionary interpretation of brain oscillations is that the former deals with anatomically defined parts of the human brain whereas the latter deals with brain oscillations whose relation to specific brain structures is a complicated and not fully resolved question. Contemporary neuroanatomy emphasizes conservation of fundamental segmental structure as the main principle of the evolution of vertebrates (Rubenstein et al., 1994, 1998). Even neocortex, the most advanced part of the mammalian brain, is hypothesized to evolve from tri-laminar reptilian dorsal cortex (Puelles, 2001; Reiner, 1993). Applying this principle to oscillations, it is reasonable to suggest that the three oscillatory systems should exist at all stages of vertebrate evolution. Indeed, delta, theta and alpha frequencies could be found in each vertebrate (Basar, 1998), but their relative amplitudes are remarkably different in different species. Accordingly, functions, which are presumably linked with the three global oscillatory systems, should be present in all vertebrates, but emphasis and dominance of some functional and behavioral patterns should differ across different species.

In this review I will try to defend the thesis that delta oscillations manifest the most ancient oscillatory mode, comparative to higher frequency oscillations. To comply with this thesis, delta oscillations must (1) dominate in EEGs of lower vertebrates (particularly reptiles, the direct mammal's ancestors). 'Dominate' in this context means that they not only must have the highest power in the spectrum (comparative to theta and alpha frequencies), but be the most functionally 'active' oscillations, which are more pronounced during typical to these species activity and in response to environmental challenges. (2) In humans, in compliance with the principle first formulated by Jackson (1958), delta oscillations must be more pronounced in conditions that are associated with diminished activity of the 'higher', or more advanced 'nervous arrangements'. These conditions include: (a) earlier developmental stages, when 'higher nervous arrangements' are yet in the process of maturation; (b) deep sleep, when the brain is in a state of functional decortications (Rial et al., 2007b); (c) pathological states caused by detrimental environmental factors, developmental pathology, or damage to brain tissue.

Besides these general predictions, evolutionary interpretation allows a number of more specific predictions about association of delta oscillations with behavior and physiological processes. These predictions could be derived basing on the main premise of the evolutionary interpretation, namely, that the oscillatory system, which dominates the EEG of particular species, should be linked to functions and behavioral patterns that dominate the behavior of these species. The behavior of lower vertebrates is dominated by patterns directly oriented to the acquisition of biologically important goals such as physical maintenance, survival, dominance, and mating. To be adaptive, such behavior requires constant feedback from sensors signaling deviation from optimal homeostasis. Hence, delta activity is expected to be sensitive to internal stimuli signaling such deviations (e.g., during hypoxia, hypoglikemia, fatigue, pain), as well as to the stimuli signaling a need for sexual activity (e.g. the level of sexual hormones). It should be implicated in monitoring of autonomic functions, such as breathing and heartbeat, because these functions are critical for survival. Since behavior that is oriented to satisfaction of basic biological needs is mostly guided by activity of the brain reward systems, delta oscillations are expected to be sensitive to signals coming from these systems. In particular, that should be evident with respect to drugs of abuse which directly influence the brain reward systems. Because the detection of motivational salience of environmental stimuli is supposed to be the main function of the brain reward systems (Gray, 1999), delta activity is expected to play a leading part in this process. Besides, delta activity is expected to be associated with primitive defense mechanisms which in humans are mostly rudimentary.

In the following text, available empirical evidence will be matched against these predictions. This text will be divided into two sections. The first section will review the evidence on the presence of delta oscillations in lower vertebrates and at different developmental and circadian cycle stages, as well as the evidence

on association of delta oscillations with autonomic regulation and pathological processes. The second section will review the evidence on the place of generation of delta activity and its association with motivation and cognitive processes.

2. Delta oscillations and basic biological processes

2.1. Delta oscillations in lower vertebrates

If delta oscillations indeed represent an evolutionary old oscillatory mode, they must dominate in EEGs of lower vertebrates, particularly during typical for these species behavior. During his fruitful career, Theodor Bullock has undertaken the most considerable effort to collect comparative EEG data. Analysis of these data allowed him to formulate two basic generalizations. The first one reflects a drastic difference in the EEGs between all vertebrates and all invertebrates (with the exception of cephalopods, Bullock, 1984). The typical invertebrate record of ongoing electrical activity is spiky with single unit and compound spikes of one or a few milliseconds width, riding on slow waves that are usually inconspicuous, whereas in all vertebrates, slow waves are omnipresent and relatively stronger. This observation is in line with the above discussed idea that oscillations appear in vertebrates as a mechanism for integration of cerebral activity which is only needed in large brains and is not necessary in small brains of most invertebrates, with the exception of cephalopods, which have relatively larger brains. The second generalization affirms that the raw recordings and the power spectra look alike in all the vertebrates falling quite steeply on each side of a maximum around 5–15 Hz (Bullock, 1993), meaning that oscillations of all frequency ranges could be found in EEGs of all vertebrates. However, different species differ on which frequency dominates the EEG spectrum. In all poikilotherm vertebrates, the highest EEG power is found in the delta range (Bullock and Basar, 1988; Bullock, 2003; Nicolaua et al., 2000), whereas mammalian EEG is dominated by theta or alpha oscillations.

Several studies have been performed recently to directly compare reptilian EEG to that of mammals. To compare the ongoing electrical activity in possibly homologous structures of reptiles and mammals, Gaztelu et al. (1991) recorded the EEG from major parts of the cortex of unanesthetized turtles (Pseudemys) and geckos (Gekko) with and without acute and chronic stimuli. In both species, the EEG spectrum was dominated by a single maximum at about 2 Hz. The immobility-related theta activity, so characteristic of the hippocampus in a number of mammals, was not found in the medial cortex (the possible homolog of the mammalian hippocampus) of either species of reptile under a variety of conditions. De Vera et al. (1994) computed the power spectrum of *Gallotia galloti* lizard's EEG at different body temperatures. EEG power spectra were mainly characterized by a low frequency peak between 0.5 and 4 Hz which was present at the different body temperatures. Flash evoked potentials were characterized by a slow triphasic component upon which a spindle was superimposed, adopting morphology similar to the K complexes of mammalian sleep. The authors conclude that characteristics of this EEG and evoked potentials support the hypothesis of homology between the waking state of the reptiles and the slow-wave sleep of mammals. This hypothesis was further confirmed by comparing the presence of fractal or nonlinear structure in lizard *G. galloti* during open and closed eyes with those reported for human slow-wave sleep (Gonzalez et al., 1999).

On the other hand, many earlier studies repeatedly recognized the absence of delta waves in EEGs of turtles (Strejkowa and Servit, 1973; Walker and Berger, 1973; Flanigan, 1974; Flanigan et al., 1974), lizards (Flanigan, 1973), snakes (Peyreton and Dusan-Peyreton, 1969) and crocodiles (Flanigan et al., 1973; Warner and Huggins, 1978). Basing on these observations, Rattenborg (2006) put forward a hypothesis that the evolution of slow waves is linked to the independent evolution of extensive palliopallial connectivity in mammals and birds. The absence of slow waves in sleeping reptiles suggests that the neuroanatomical and neurophysiological traits necessary for the genesis of slow waves evolved independently in the mammalian and avian ancestors and are absent in reptiles (Rattenborg, 2006). This idea has been criticized by Rial et al. (2007a), who noted that the review by Rattenborg (2006) missed important aspects in relation to the characteristics of sleep in poikilotherm vertebrates and in the evolution of sleep. Poikilotherms continuously show an EEG dominated by slow waves, but its highest amplitude appears not during sleep, but during active waking. In addition, they show an arousal reaction which consists in an increase in EEG delta amplitude and synchrony, opposite to mammals and birds (Rial et al., 2007a).

Indeed, as Rial et al. (2010) show, the above noted contradiction may be explained by taking into account the relationship between EEG amplitude and animal's activity: cool and resting reptiles show minimal EEG amplitude. However, warm and active animals show slow waves with amplitudes which often surpass the normative value of 75 mV to be included within the delta EEG range (De Vera et al., 1994). Further, Rial et al. (2010) dismiss the assertion that the reptilian delta waves might be a reflection of respiratory activity (Rattenborg, 2007). They show that although EEG delta and breathing indeed significantly correlate with each other in Gallotia lizards, the power in the low frequency range turns out to be higher during apneic intervals which in these animals occur in states of maximal arousal. This fact confirms that the highest EEG delta amplitude occurs in these animals in parallel with behavioral activation. The authors conclude that reptilian wakefulness could be named Slow-Wave Wakefulness (Rial et al., 2010). In their comprehensive review of this issue, Rial et al. (2010) propose that (1) the active state of reptiles is a form of subcortical waking, without homology with the cortical waking of mammals; (2) reptilian waking gave origin to mammalian sleep; (3) reptilian basking behavior evolved into non-rapid eye movement sleep (NREM); (4) post-basking risk assessment behavior, with motor suspension, head dipping movements, eye scanning and stretch attending postures, evolved into phasic rapid eye movement sleep (REM); (5) post-basking, goal directed behavior evolved into tonic REM and (6) nocturnal rest evolved to shallow torpor.

From this brief review of comparative data we may conclude that there is a clear distinction between mammals and lower vertebrates in the spectral composition of EEG and its correlation with behavior. During wake, mammals' EEG is dominated by theta and alpha oscillations, and typical reaction to environmental events consists of alpha desynchronization and theta synchronization (Klimesch, 1999), although event-related delta enhancement is also frequently observed. During slow-wave sleep (SWS) theta and alpha disappear and delta oscillations dominate the EEG of mammals. In reptiles (and probably other lower vertebrates), theta and alpha oscillations are not conspicuous in the EEG, which in active states is dominated by delta oscillations. The typical reaction to environmental challenges consists of prominent increase of the amplitude of delta waves. During sleep, oscillations (including delta) are extinguished and EEG shows arrhythmic sporadic activity. Table 1 presents a summary of main EEG features in reptiles, rodents, and humans during sleep, quiet wake, and arousal.

2.2. Delta oscillations and development

In accord with the principle that ontogeny recapitulates phylogeny, low frequency oscillations dominate the human EEG during early developmental stages. The so-called delta brushes are the

Table 1
Summary of main EEG features in reptiles, rodents, and humans during sleep, quiet wake, and arousal (relevant literature is cited in the text).

	Sleep	Wake	Arousal
Reptiles	No waves, or low amplitude delta	EEG is dominated by delta	Increase of delta amplitude + spindles 7–14 Hz
Rodents	SWS-EEG is dominated by delta	EEG is dominated by theta	Increase of theta amplitude + alpha desynchronization
Humans	SWS-EEG is dominated by delta	EEG is dominated by alpha	Mostly alpha desynchronization

dominant pattern of oscillatory activity in the human cortex during the third trimester of gestation (Scher, 2008). Random or briefly rhythmic 0.3–1.5 Hz delta activity of 50–250 μV is associated with a superimposed rhythm of low to moderately faster frequencies of 10–20 Hz. Historically, different authors have described these complexes as spindle delta bursts, brushes, spindle-like fast waves or ripples of prematurity (Scher, 2008). Delta patterns in the central or midline locations are predominant for the infant at less than 28 weeks' gestation. Other delta rhythms occur in the temporal and occipital locations particularly after 28 weeks' gestation (Selton et al., 2000). Between 30 and 34 weeks' postmenstrual age, temporal and occipital delta rhythms become quite prominent and rhythmic, with durations that may exceed 30 s to 1 min (Scher, 2008). In infants who were < or =30 weeks of gestation, the median relative power of the delta band increased significantly from 68% on day 1 to 81% on day 4 (Bell et al., 1991; Victor et al., 2005). In healthy infants in the eyes open awake condition, the delta activity does not substantially change at the end of the first year of life, compared with the first 3 months, whereas theta and alpha activity notably increases (Vladimirova, 1991). During further development of resting state activity, there is a reduction in the amplitude of slow-wave (delta and theta) rhythms, while faster rhythms (alpha, beta, and gamma) increase during childhood and adolescence (John et al., 1980; Matousek and Petersen, 1973). This dynamic is generally considered as a sign of maturation (Clarke et al., 2001).

2.3. Delta oscillations and sleep

From the standpoint of this review, sleep is an interesting natural model of functional decortication (Rial et al., 2007b), when advanced operational brain systems are inactive and, in accordance with the principle formulated by Jackson (1958), 'lower nervous arrangements' must rise in activity. Indeed, in adult humans, delta oscillations are most evident during the slow-wave sleep (SWS) and till now overwhelming majority of delta oscillation-related publications concern sleep. Abundance of these publications precludes their adequate coverage here and reader is referred to many recently published reviews discussing different aspects of sleep including its evolutionary roots (Lee Kavanau, 2002; Rattenborg, 2006; Rattenborg et al., 2009; Rial et al., 2010) and possible functional significance (Diekelmann and Born, 2010; Frank, 2006; Rial et al., 2007b; Vassalli and Dijk, 2009).

Most animals have been found to sleep or exhibit a sleep-like state (Siegel, 2005). The fact that animals die when deprived of sleep (Montagna et al., 2003; Rechtschaffen and Bergmann, 2002), indicates that sleep serves a vital function. This function, however, is still a matter of debate (Rechtschaffen and Bergmann, 2002; Siegel, 2005; Stickgold and Walker, 2005; Vertes and Siegel, 2005). Although most animals have sleep-like states, only mammals and birds exhibit the electroencephalographic patterns that characterize the sub-states of mammalian sleep, slow-wave sleep (SWS) and rapid eye movement (REM) sleep (Campbell and Tobler, 1984). The SWS consists in a general slowing of most bodily functions and in a diminished responsiveness to sensory stimuli. The EEG is characterized by high voltage low frequency activity, spindles in the 14 Hz range, and high voltage isolated waves, which often occur either spontaneously or after sensory stimulation (K-complexes). REM sleep shows a deep muscular relaxation, but sudden eye movements and muscular twitches occur. EEG is similar to that of waking, with low voltage, mixed frequencies (Nicolaua et al., 2000). The taxonomy of SWS waves is not always clear in humans. Unit recordings have shown that neuronal activity during SWS is characterized by a fundamental oscillation of membrane potential. This so-called 'slow oscillation' (<1 Hz) is recorded in all major types of neocortical neurons during SWS (Steriade and McCarley, 2005; Steriade et al., 1993b). Delta rhythm (1–4 Hz) is another characteristic oscillation of SWS. The neural underpinnings of SWS delta rhythm remain uncertain. In the dorsal thalamus, a clock-like delta rhythm is generated by the interplay of two intrinsic membrane currents, although another delta rhythm survives complete thalamectomy (Steriade and McCarley, 2005). Whether the difference between slow waves and delta waves is qualitative or quantitative remains unclear (Dang-Vu et al., 2008). It is suggested that the slow waves are generated directly within cortical circuits, whereas delta rhythm is derived from intrinsic properties of thalamocortical cells and from intracortical network interactions (Steriade and McCarley, 2005). Therefore, the slow and the delta waves should be considered distinct processes. In line with this hypothesis, EEG power densities within slow and delta frequency bands differ in their dynamics through the night (Achermann and Borbely, 1997). The other hypothesis views slow waves and delta waves as realizations of a unique process, which varies in a continuous parameter space. The amplitude and slope of the waves reflect the level of synchronization achieved in cortical neural ensembles, which depends on the local homeostatically regulated average synaptic strength (Esser et al., 2007). In keeping with this hypothesis, Dang-Vu et al. (2008) have not found significant difference in brain activation when comparing slow waves and delta waves at the macroscopic systems level. Because the distinction between delta oscillations and the 'slow waves' (i.e., <1 Hz) is not definitely determined, for the purpose of this review, I will consider all oscillations below 4 Hz as a single phenomenon.

Another important question is whether SWS delta and waking delta represent the same oscillation. Studies correlating positron emission tomography (PET) and EEG showed a positive correlation between waking delta and PET metabolism in the medial frontal cortex (Alper et al., 1998, 2006). On the other hand, earlier studies of SWS sleep by using PET (Maquet, 2000) or block-design fMRI (Kaufmann et al., 2006) found decreased brain activity during SWS sleep, suggesting that SWS delta differs from waking delta in its relation to brain metabolism (e.g., Alper, 1999). However, a later more stringent fMRI study have found only positive correlations between slow waves and delta on the one hand and fMRI BOLD signal on the other (Dang-Vu et al., 2008).

Three theories have been proposed to explain the evolution of the two mammalian sleep stages. The 'REM first' hypothesis suggests that REM should be considered the primitive sleep (Tauber et al., 1966). The opposite hypothesis ('SWS first') was proposed by Allison and collaborators (Allyson and Van Twiver, 1970). Finally, Rial et al. (1993, 2010) proposed that the SWS is the result of converting what could be called the Slow-Wave Waking of poikilotherms into the SWS (Nicolaua et al., 2000). This latter theory has been criticized by Rattenborg (2007) who argued that although waking and active reptiles indeed show delta activity similar to the one observed in mammals during SWS, this activity is still less synchronized or lower in amplitude. Besides, the proposed conversion

of reptilian wakefulness into SWS is untenable from behavioral, mechanistic, and functional perspectives. A more parsimonious explanation is that the precursor state to SWS in homeotherms was a state comparable to reptilian sleep, rather than wakefulness, with the primary difference being that the reptilian dorsal cortex lacks the interconnectivity necessary to generate sleep-related slow waves in the EEG. Actually, the hypotheses proposed by Rial et al. (1993, 2010) and by Rattenborg (2006) do not seem mutually exclusive. The fact that oscillation amplitudes and the degree of their synchronization are larger in mammals than in reptiles (Bullock, 1997) is in line with current understanding that the need for oscillation as a mechanism for cerebral integration dramatically increases with increasing brain size. In keeping with that, mammals have larger synchrony of all oscillations, including the delta waves. This, however, may not deny the fact that in mammals delta waves dominate only during SWS, whereas in reptiles they are most pronounced during active waking. Interestingly, both in cats and humans, EEG responses to external stimuli during SWS are predominantly evident in the delta range of frequencies (Basar, 1999), which again is similar to what is observed in waking reptiles. This similarity makes it possible to consider mammalian SWS and reptilian waking as possible evolutionary homologues in relation to delta oscillations.

Such understanding has profound implications for deducing possible significance of delta oscillations in mammals. Indeed, we have to accept that in reptiles, delta oscillations represent the dominant oscillatory mode which is most prominent during active behavioral states and demonstrates the most salient oscillatory response to environmental challenges, while EEG of behaviorally active mammals is dominated by theta and alpha oscillations, which in these animals show the most salient responses to environmental challenges. During SWS, when active behavior is not on agenda, the dominant operational oscillatory modes pass away and, in line with the John Hughlings Jackson's principle, more ancient and fundamental oscillatory mode rises in activity. It could be speculated that this periodic return to an evolutionary prototypal state is necessary for reconciling the experience acquired during the active state with the fundamental biological self. Indeed, the most currently popular hypothesis about functional significance of SWS posits that it is necessary for consolidation of memory traces acquired during the wake (Ji and Wilson, 2007; Marshall et al., 2006; Peigneux et al., 2004; Rasch et al., 2007). Using simultaneous EEG and fMRI recording, Dang-Vu et al. (2008) have recently shown that during SWS, significant increases in activity were associated with slow waves (<1 Hz, >140 μV) and delta waves (1–4 Hz, 75–140 μV) in several cortical areas, including the inferior frontal, medial prefrontal, precuneus, and posterior cingulate areas. Compared with baseline activity, slow waves were associated with significant activity in the parahippocampal gyrus, cerebellum, and brainstem, whereas delta waves were related to frontal responses. However, no significant difference in brain activation was found when comparing slow waves and delta waves at the macroscopic systems level. The pattern of activation that is related to delta and slow-wave oscillations during SWS allows put forward an interesting hypothesis. The brain regions that are obviously integrated by slow oscillations during SWS include 'old' pontine tegmentum and midbrain nuclei, the parahippocampal gyrus, a major relay area between the hippocampus and neocortex (Mohedano-Moriano et al., 2007), and a set of cortical regions associated with the so-called default mode network (DMN, Raichle, 2006). Ample evidence suggests that this latter network, which is mostly active during resting wakefulness, is associated with self-referential processes that constitute the core of the self (Gobbini et al., 2007; Mitchell, 2006). Noteworthy, the association of sleep slow waves with DMN structures has been confirmed in a study using high-density EEG source modeling (Murphy et al., 2009). Therefore, the triangle of co-activated areas consists of the 'old' brainstem structures involved in major homeostatic regulation, the parahippocampal areas associated with acquisition and interpretation of new experience, and the DMN structures representing the core self-referential processes. It appears that this every-night reconciliation of daytime experience with basic biological and self-referential processes is vital for preserving integrity of the self.

2.4. Delta activity and autonomic regulation

If delta oscillations are implicated in coordination of behavior with basic biological and homeostatic needs, they must participate in synchronization of brain activity with autonomic functions. That may be particularly so during sleep, when active behavior is not on agenda and mostly restorative physiological processes take place. It has been shown that the prevalence and amplitude of delta oscillations during sleep are critically dependent on the time we are awake before we fall asleep, as well as the time passed since sleep onset. This observation led Alexander Borbely (1982) to propose that delta power is an EEG correlate of a homeostatically regulated sleep-dependent process, which he coined 'Process S'. Although later observations indicate that it is not all that simple and that differences in waking 'quality' do affect EEG delta power during subsequent SWS sleep (Meerlo et al., 1997), slow-wave activity is still thought to mediate the restorative function of SWS (Borbely and Achermann, 2000; Tononi and Cirelli, 2006). It is suggested that slow oscillations might help synaptic consolidation or produce synaptic downscaling to increase signal-to-noise ratios (Tononi and Cirelli, 2006). Another possible mechanism of the restorative function of slow-wave activity is replenishment of the glycogen that is depleted during wakefulness (Benington and Heller, 1995). SWS may have restorative properties not only for the brain, but also for peripheral organs. A selective reduction in SWS and slow-wave activity for only a few nights was shown to result in a clear adverse effect on glucose homeostasis and increased risk of type 2 diabetes in young healthy adults (Tasali et al., 2008). Taken together, these studies suggest that delta wave generation is important for normal functioning of both the brain and peripheral organs. This implies that measures of delta activity must correlate with measures of autonomic activity and metabolism. In this section, evidence showing that this is indeed the case will be reviewed.

Within the so-called common brainstem system (CBS), which implements basic regulation and exerts influences on cardiovascular system, respiration, motor systems, processing of the afferences, and on vigilance (Moruzzi, 1972; Langhorst et al., 1992), mainly respiratory related rhythms, cardiac rhythms, and rhythms related to the delta–theta rhythms of the EEG occur (Langhorst et al., 1981; Lambertz and Langhorst, 1998). Lambertz et al. (2000) showed that the functional organization of this system seems to be dependent on the coupling between the cortical and the reticular formation (RF) delta oscillations. The authors suppose that "either higher brain structures signalize the beginning of the phase transition to the RF or – even more probable – the CBS in the RF induces a general activation in the CNS via its ascending pathways" (Lambertz et al., 2000). These results confirm and complement previous investigations on the rhythmic coordination of different peripheral and central subsystems which occurs within the range of low EEG frequencies (Lambertz and Langhorst, 1998). The bulk of evidence suggests that the cortical delta rhythm may not be implemented in the direct on-line autonomic regulation but rather reflects a central representation of peripherally occurring autonomic changes (Lambertz and Langhorst, 1998).

Heart rate variability is one of the most frequently used indicators of autonomic regulation. High-frequency (HF) heart rate variability is associated with breathing and depends on vagal regulation (for a review and theory see Porges, 1995). Low frequency

(LF) heart rate variability is believed to more reflect sympathetic than parasympathetic influences (e.g. Snidman et al., 1995). Existing evidence shows an association between heart rate variability and delta activity during NREM and REM sleep. Modifications in HF heart rate variability show parallel changes with changes in delta EEG band both in young and in middle-aged men (Jurysta et al., 2005). Contrariwise, LF heart rate variability moderately negatively relates to the sleep delta power (Ako et al., 2003). Correspondingly, the LF/HF ratio is significantly negatively correlated with delta power in humans (Yang et al., 2002) and in rats (Yang et al., 2003) which is taken as an evidence of negative association between delta and sympathetic activity. The link between cardiac autonomic activity and sleep delta power is altered in patients with major depressive disorder (Jurysta et al., 2010) and sleep apnea-hypopnea syndrome (Jurysta et al., 2006), as well as in patients with chronic primary insomnia (Jurysta et al., 2009). The association between delta power and heart rate variability was also observed during REM sleep (Pedemonte et al., 2005). Overnight delta activity also negatively relates to the mean arterial pressure (Charloux et al., 2002) and the cortisol secretory rate (Gronfier et al., 1999), which again suggests that during sleep, power of delta oscillations is negatively related to sympathetic activity. It is important to emphasize that observed in these studies modifications in autonomic measures precede changes in delta EEG band activity by several minutes (Brandenberger et al., 2001) implying a reflective rather than a regulative role for the EEG delta activity. In patients with periodic limb movement disorder, the periodic limb movement onset is heralded by changes of HR and EEG delta activity (Ferrillo et al., 2004; Allena et al., 2009). The authors suggest that this and similar phenomena can be linked to the activity of a common brainstem system, which receives peripheral inputs, regulating the vascular, cardiac, and respiratory activities and synchronizing them to cortical oscillations of EEG. It is shown also that SWS delta power is sensitive to interleukin-1β (Yasuda et al., 2005) and lipopolysaccharide (Lancel et al., 1995), suggesting a role in information exchange between immune and nervous systems. Preoptic/anterior hypothalamic warming has been shown to increase EEG delta activity within NREM sleep with no effect on theta or sigma frequencies (McGinty et al., 1994). SWS delta power also correlates with the restorative biosynthetic processes occurring during sleep in the brain (Dworak et al., 2010).

In one of a few studies where an association of delta activity with autonomic measures was observed in waking, the total severity of cybersickness during exposure of subjects to virtual reality had a significant positive correlation with gastric tachyarrhythmia, eyeblink rate, heart period, and EEG delta wave power (Kim et al., 2005). EEG delta power recorded in 1831 healthy subjects aged 6–86 years in eyes open condition correlated positively with basal metabolic rate over the human lifespan (Boord et al., 2007). It has been also shown that in 28 hypertensives, audiovisual entrainment in the subdelta frequency (0.5–1 Hz) had marked effects on blood pressure, reducing the systolic 20 points and diastolic 15 points (Siever and Berg, 2002). The authors suggest that this treatment directly impacts physiological functions rather than psychological ones. In sum, reviewed in this section data support the idea that delta oscillations (particularly during sleep) may participate in synchronization of brain activity with autonomic functions.

2.5. Pathological states and delta oscillations

Because the new-born more advanced brain devices specialize on performing more complex computations, they should be generally more sensitive to detrimental influences, and, when these devices are rendered functionless, 'lower nervous arrangements' must rise in activity (Jackson, 1958). Indeed in the pre-quantitative EEG era, appearance of prominent delta waves during wake was treated as a sign of pathology. Existing evidence shows that delta activity increases in almost any pathological state associated with brain tissue damage, developmental disorder, or even more subtle disorders without known organic cause. Localized delta activity appears in cortex overlying a circumscribed white matter lesion, or after a localized thalamic or midbrain tegmentum lesion (Gloor et al., 1977). The volume of lesions was correlated with delta power and the origin of the equivalent dipoles for delta oscillations was shown to be within the volume of the lesion (Harmony et al., 1995). The so-called frontal intermittent rhythmic delta activity (FIRDA) is associated with a wide range of lesions and encephalopathic conditions (Ettore et al., 2011). Delta amplitude is overall higher in chronic aphasic patients with structural lesion in the left cortical–subcortical perisylvian areas. Delta band, in addition to its ability to reflect structural damage, was effective in the assessment of functional impairment in these patients as well (Spironelli and Angrilli, 2009).

Hypoxia is yet another condition associated with brain tissue damage or suppression, because the brain is particularly sensitive with respect to its oxygen requirement (Sudarsky, 1990). In EEG, hypoxia is associated with a decrease of alpha and prevalence of slow waves. Thus, the reduction of cerebral blood flow caused by cerebral ischemia has been suggested to be related to the slowing of the background EEG activity (Ingvar and Sulg, 1969). Increased slow-wave components have also been observed in patients with cerebral ischemia caused by reduction of the cortical blood flow (Nagata et al., 1989; Tolonen and Sulg, 1981). The EEG change in hypoxic hypoxia has been reported to be similar to that in ischemic hypoxia (Meyer and Waltz, 1960; Saletu and Grunberger, 1985; Kraaier et al., 1988b). In the experimental study of cerebral hypoxia, Kraaier et al. (1988a) compared the spectral power under the normobaric normoxic condition with that recorded under the hypobaric hypoxic condition corresponding to the altitude of 6096 m. They found a significant increase in slow activity as well as a significant decrease in alpha activity under the simulated hypobaric condition. Ozaki et al. (1995) reported similar results. EEG study during a Himalayan expedition showed that subjects who later developed symptoms of acute mountain sickness exhibited an increase of slow cerebral activity in the right temporal region already at 3440 m. These findings indicate that regional brain dysfunction, which can be documented by slow-wave EEG, heralds the appearance of clinical symptoms of acute mountain sickness (Feddersen et al., 2007).

Wide range of developmental disorders is associated with the increase of relative power in low EEG frequencies including delta and theta waves. The most consistent finding in EEG studies of attention-deficit/hyperactivity disorder (ADHD) has been increased low frequency activity, predominantly theta, but also delta in children with ADHD compared with control children (for a review, see Barry et al., 2003). Excess of spectral power in low frequency bands is also associated with disorders of learning and attention in children (Barry et al., 2003; Chabot et al., 2001). Children with dyslexia showed greater overall delta amplitude (Penolazzi et al., 2008). Studies which have examined the influence of adverse early rearing conditions or sociocultural risk factors on the development of the EEG in children have generally reported higher EEG power at low frequencies to be associated with detrimental aspects of the child's living environment (Harmony et al., 1990; Kreppner et al., 2001; Marshall and Fox, 2004; Otero et al., 1997; Raine et al., 2001; Vorria et al., 1998). Moreover, increases in delta and theta activity were found in the EEGs of fetal alcohol syndrome and Down syndrome children (Babiloni et al., 2009a; Kaneko, 1995). In adult populations, increase of baseline delta power was observed in patients with schizophrenia (Alfimova and Uvarova, 2003; Bates et al., 2009; Boutros et al., 2008; Fehr et al., 2001; Karson et al., 1987), Parkinson's disease (Soikkeli et al., 1991;

Zijlmans et al., 1998), Alzheimer's disease (Babiloni et al., 2009b; Valladares-Neto et al., 1995), depression (Bjørk et al., 2008; Gatt et al., 2008; Korb et al., 2008; Saletu et al., 2010), anxiety disorder (Gauthier et al., 2009), phylogenetic fears (Bornas et al., 2009), obsessive compulsive disorder (Olbrich et al., 2009; Velikova et al., 2010), and in young adults born at extremely low birth weight (Miskovic et al., 2009). It has been shown also that dissociative experiences which appear in torture victims are associated with slow waves generated in the left ventrolateral frontal cortex (Ray et al., 2006).

Because anhedonia, the reduced propensity to experience pleasure, is a vulnerability factor for several psychiatric disorders, including depression and schizophrenia, it is important to note that in a non-clinical sample, anhedonia, but not other symptoms of depression or anxiety, was correlated with increased resting delta current density in the rostral anterior cingulate cortex (Wacker et al., 2009). Increased theta and delta power have been associated with poor antidepressant treatment response in patients with depression (Knott et al., 2000). However, conflicting evidence has also been reported. For example, Coutin-Churchman et al. (2003) compared 67 normal human beings and 340 psychiatric patients on EEG spectral power measures and found increase in slow bands in only 24 recordings that were scattered between various conditions, including polysubstance dependence, epilepsy, bipolar disorder, and also two normal subjects, whereas decrease in slow bands was found in 75 recordings from psychiatric patients. These discrepancies could be attributed to the different methodologies used for the calculation of EEG measures (e.g., absolute vs. relative power), different conditions (e.g., eyes open vs. eyes-closed condition), different periods of time (e.g., evening vs. morning: see e.g., Gauthier et al., 2009) and so on.

Summing up, a variety of mental conditions associated with brain tissue damage or pathology, like Alzheimer's disease, as well as developmental disorders and prominent psychopathologies, like schizophrenia or Down syndrome are undoubtedly associated with an increase of resting state delta oscillations. Some more subtle disorders, like depression, obsessive compulsive, and anxiety disorder have also been reported to be associated with an increase of delta power, although further research is needed to clarify whether this is indeed so.

2.6. Interim discussion

Reviewed so far evidence shows that delta oscillations dominate the EEGs of waking and active reptiles. In humans, delta oscillations are prominent during early developmental stages and decrease in the course of normal development. In adult healthy humans, EEG dominated by delta waves could usually be seen only during SWS. That prompted Rial et al. (1993) to consider reptilian waking as evolutionary antecedent of mammalian sleep. Increase of delta power in resting EEG has been documented in a wide array of developmental disorders and pathological conditions. The lack of specificity of this effect prompts one to suggest that increase of slow waves and/or decrease of alpha are almost universal responses to any brain damage or pathology, or even deviation from homeostatic optimum. The appearance of delta oscillations in EEG is traditionally interpreted as a correlate of inhibition. This interpretation, however, has little support in contemporary neuroscience. Many studies have described event-related increase of delta power as a correlate of 'active' cognitive processes (see later in this review). Moreover, even during SWS, which is considered a state of generalized inhibition, delta oscillations continue to respond to environmental signals with an increase of power (Basar, 1999) in much the same way as they do in waking reptiles or humans. Besides, simultaneous EEG and fMRI recording during SWS shows that delta waves correlate positively with the BOLD signal (Dang-Vu et al., 2008). Compared with baseline (a state with no delta waves), delta waves are associated with increase of activity in many brain regions. This prompted authors to suggest that SWS is not a state of brain quiescence, but rather is an active state during which brain activity is consistently synchronized to the slow oscillation in specific cerebral regions (Dang-Vu et al., 2008). Recent studies suggesting a role for slow oscillations in memory consolidation are also in line with the emerging understanding of these waves as correlates of active functional processes, rather than inhibition. Considerable evidence on the association between delta waves and autonomic and metabolic processes during SWS shows that integration of cerebral activity with homeostatic processes might be one of these waves' functions. As for the increase of delta waves in pathological conditions, a bulk of evidence shows that these conditions are rather associated with behavioral disinhibition (see Knyazev, 2007 for a review) than inhibition. Overall, all this evidence is reconcilable with the idea that slow EEG oscillations are associated with evolutionary old basic processes, which in waking adult humans are overshadowed by more advanced and operationally flexible processes associated with higher frequency oscillations. The former processes rise in activity, however, when the latter are dysfunctional. In the following sections I will review evidence showing that in waking adult humans, delta oscillations are associated with basic biological motivations and the detection of motivationally salient stimuli in the environment, but first I will discuss evidence about the place of generation of delta activity.

3. Delta oscillations, motivation, and cognition

3.1. The place of generation of delta activity

If we suppose that the function of delta oscillations is somehow linked with motivation, we should expect that the generation of delta activity should be associated with motivational brain circuits. Unfortunately, most of these circuits are located deep in the brain and their electrical activity is not directly accessible on the scalp. Although we may learn much from findings that come from fMRI, PET, and animal research, caution should be exercised when these findings are applied to the explanation of human EEG data. All of these tools provide very different points of view of the underlying neuronal activity and it is not obvious, for example, if findings from cell recordings in rodents will have strong bearing for scalp EEG in humans.

There is no definitive evidence regarding the place of origin of delta activity in the brain. Low frequency (<1 Hz) oscillations are presumably generated in sleep directly in the cortex (Steriade et al., 1993b). This ultra-slow activity is supposed to reflect cortical reorganization of waking circuits (Steriade et al., 1993b). According to Steriade et al. (1990, 1993a) the ascending cholinergic subcortical–cortical projections from the thalamus are critical to the generation of slow-wave EEG rhythms. However, studies using dipole modeling place the site of waking delta generation in anterior medial frontal cortex, rather than in thalamus (Michel et al., 1992, 1993). Neuroimaging of sleep by means of low-resolution brain electromagnetic tomography (LORETA) has also found that in the delta frequency range, activity was maximal in the medial prefrontal cortex and spread into the anterior cingulate and orbitofrontal cortex (Anderer et al., 2002). Murphy et al. (2009) using high-density EEG source modeling showed that SWS individual spontaneous slow waves (0.5–6 Hz) have distinct cortical origins, propagate uniquely across the cortex, and involve unique subsets of cortical structures. However, when the waves are examined en masse, diffuse hot spots of slow wave origins could be noted. These hot spots are centered at the left insula and the medial cingulate gyrus. Slow-wave propagation along the anterior

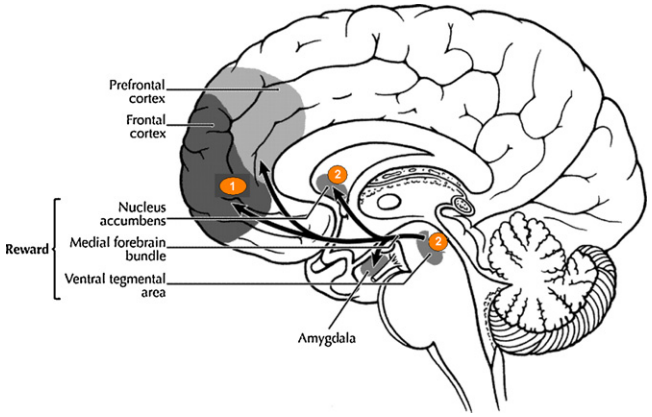

Fig. 1. Localization of potential sources of EEG delta activity superimposed on localization of brain reward circuitry. Red circles show the localization of potential sources of EEG delta activity as revealed by source modeling and correlation of EEG with PET and fMRI signal in humans (1) and by direct registration of electric activity in subcortical regions of waking animals (2). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

posterior axis of the brain is largely mediated by a cingulate highway. The highest density of streamlines was in the anterior portions of the cingulate. As a group, slow waves are associated with large currents in the medial frontal gyrus, the middle frontal gyrus, the inferior frontal gyrus, the anterior cingulate, the precuneus, and the posterior cingulate.

Studies correlating positron emission tomography (PET) and EEG indicate a positive correlation between waking delta and PET metabolism in the medial frontal cortex (Alper et al., 1998, 2006). This positive correlation contrasts with the negative relationship of the delta of SWS to PET metabolism (Maquet et al., 1997) or regional blood flow in the medial frontal cortex (Hofle et al., 1997). These apparently opposite relationships of delta to metabolism and blood flow in medial frontal cortex in sleep vs. waking states have been taken as an argument of the functional distinction between the waking and SWS delta (e.g. Alper, 1999). However, as Dang-Vu et al. (2008) note, the statistical contrasts used in many of these earlier studies compared the distribution of brain activity during sleep and wakefulness. Besides, because of its low temporal resolution, PET is poorly adapted to detect transient brain activities compared with event-related fMRI. fMRI analysis describing the transient changes in regional brain activity associated with discrete SWS delta waves relative to a baseline consisting of low-power theta, alpha, and beta activity during NREM sleep has found only positive correlations between delta waves and BOLD signal in the medial frontal gyrus (Dang-Vu et al., 2008). Thus, source modeling and correlation of EEG with PET and fMRI signal consistently show location of both waking and SWS delta sources in the medial prefrontal, orbitofrontal, and anterior cingulate cortices. Direct registration of electric activity in subcortical regions of waking animals show existence of delta oscillations in the common brainstem system (Lambertz and Langhorst, 1998), the nucleus accumbens (Leung and Yim, 1993), the ventral pallidum (Lavin and Grace, 1996), and dopaminergic neurons in the ventral tegmental area (VTA, Grace, 1995). Noteworthy, most of these structures (i.e. the VTA, the nucleus accumbens, the medial prefrontal cortex, and the nucleus reticularis thalami) are implicated in the brain reward system (Gray, 1999). Fig. 1 shows localization of potential sources of EEG delta activity superimposed on localization of brain reward circuitry.

Summing up, much evidence converges on showing that both waking and sleep delta waves mostly originate from the medial frontal cortical regions. Other hot spots include the insula, which is a higher association area for bodily signals (Mesulam and Mufson, 1982), the nucleus accumbens, and tegmental brainstem area. Because all these brain regions are highly involved in motivational brain circuits, in the following sections I will discuss evidence linking delta oscillations with motivational states. Much of this evidence has been reviewed elsewhere (Knyazev, 2007), so I will mostly concentrate on topics that were not covered in this previous review.

3.2. Delta oscillations and motivation

Corr (2009) in his chapter devoted to the Reinforcement Sensitivity Theory of Personality cites Jeremy Bentham, an English 18th century jurist and philosopher: "Nature has placed mankind under the governance of two sovereign masters, pain and pleasure". In this view, organisms are seen as maximizing exposure to rewarding ('appetitive') events and minimizing exposure to punishing ('aversive') events. Rewarding or appetitive events consist of the presentation of a reward, termination of a punishment, or omission of an expected punishment, while punishing or aversive events consist of the punishment, termination of reward, and omission of an expected reward (Fowles, 2006). An important point to note here is the fact that reward itself and the termination of a punishment or omission of an expected punishment, share much in common in terms of their functions and pharmacology; and in a complementary way, punishment itself and the termination of reward, and omission of an expected reward, are similarly common (Corr, 2009). In the following, I will summarize evidence linking delta oscillations with brain reward and punishment systems.

3.2.1. Delta oscillations and the brain reward systems

For a better understanding of different effects of the rewarding drugs, a basic sketch of the brain reward systems has to be presented (for a fuller description of the reward systems and their implication in the addictive behavior see e.g. Gianoulakis, 2004). A midbrain–forebrain–extrapyramidal circuit centered on the nucleus accumbens composes the anatomical pathway of the brain reward system. Significant experimental evidence suggests that the reinforcing effects of many drugs of abuse are mediated by the mesolimbic dopamine pathway (Koob, 1992; Spanagel and Weiss, 1999; Zadina et al., 1997). The cell bodies of these neurons are located in the ventral tegmental area and project to nuclei of the forebrain, mainly the nucleus accumbens, caudate, olfactory tubercles, medial frontal cortex, amygdala and septum (see Fig. 1). Both behavioral and pharmacological evidence suggests an important role of the dopaminergic pathways of the nucleus accumbens in drug reward. Furthermore, there is significant evidence indicating an interaction between the opioid and dopaminergic systems in mediating reward of opiates as well as of some other drugs of abuse, such as ethanol (Gianoulakis, 2004). Systemic administration of many drugs of abuse increases dopamine release in the nucleus accumbens. This increase is proposed to be a sufficient condition to produce reward (Chick and Erickson, 1996; Koob, 1992; Spanagel and Weiss, 1999). Activation of μ and δ opioid receptors by morphine-like drugs increases dopamine release (Chesselet et al., 1983; Devine et al., 1993; Di Chiara and Imperato, 1988). Cocaine increases extracellular dopamine by acting at the level of the dopamine transporter and blocking its reuptake (Heikkila et al., 1975). It is argued however that the assertions that dopamine mediates 'reward' or 'reinforcement' is inaccurate and grossly oversimplified. A hypothesis has been put forward that dopamine systems are rather involved in 'wanting', but not 'liking' (Berridge and Robinson, 1998). It is suggested that 'wanting' has both directional aspects (e.g. appetite to consume food) and activational aspects (e.g. activation for initiating and sustaining instrumental actions; tendency to work for food, Salamone and Correa, 2002). A distinction between functions of phasic and tonic dopamine release has also been recognized. It has been shown

that dopamine neurons demonstrate phasic firing in response to unpredicted reward (Schultz, 1998). It has been proposed that this phasic dopamine response drives learning by signaling a prediction error that effectively labels events as 'better than expected'. Although not rewarding in itself, the phasic dopamine release must evoke feelings pleasant enough to prompt self-stimulation of areas conducive to this release. It is suggested that 'intense interest', 'engaged curiosity', and 'eager anticipation' are the types of feelings that reflect arousal of this system in humans (Panksepp, 1998). Most of the evidence linking delta oscillations with the brain reward circuits has been reviewed elsewhere (Knyazev, 2007) and will be only briefly mentioned here with more emphasis on recently published data. I will start with natural rewards and continue with drugs of abuse.

3.2.1.1. Food and sex reward and delta oscillations. Both food-related and sexual arousal is associated with the increase of delta waves and their coherence (see Knyazev, 2007 for a review). In a recent study, magnetoencephalographic recordings were performed in obese and lean female adolescents during an eyes-closed resting state condition. The obese adolescents had increased synchronization in delta and beta frequency bands compared to lean controls (Dubbelink et al., 2008). The authors suggest that these differences might be related to disturbed motivational pathways. In restricted-fed chickens, relative powers in delta and theta frequency bands were highest during feeder directed behavior (Savory and Kostal, 2006). Another study investigated EEG activity in the orbitofrontal cortex in rats during the development of food reward and craving. In the food-related environment the EEG activity peaking in the delta band (2–4 Hz) was significantly correlated with the stimulus, increasing during food reward and decreasing during food craving when compared with that in the control environment (Fu et al., 2008). It is important to note that in this study, the animals were not actually hungry. In the craving condition they were presented with a tasty food, but had no access to it, whereas the access was offered in the reward condition.

3.2.1.2. Fatigue and delta oscillations. Need for rest and sleep is one of the basic biological needs. Empirical evidence shows that fatigue is mostly associated with increase of delta and theta oscillations (Kiroy et al., 1996; Lal and Craig, 2002, 2005; Makeig and Jung, 1996; Yamamoto and Matsuoka, 1990) although some increase of alpha activity could also be observed (Ninomija et al., 1993; Torsvall and Åkerstedt, 1988). It should be noted that different spectral constituents of the EEG may correlate with different components of such a complex phenomenon as e.g. driver fatigue. For example, enhanced theta oscillations may be associated with appearance of drowsiness, whereas increase of alpha may reflect attempts to overcome this drowsiness. However, in normal condition, a distinct increase of delta oscillations is usually evident only with the sleep onset (Tanaka et al., 1997) which is not the case of fatigued subjects. In this case delta increase probably signals an urgent need for rest.

3.2.1.3. Drugs of abuse and delta oscillations. This topic has been extensively reviewed in a review by Knyazev (2007), as well as in some other reviews (e.g., Alper, 1999), so it will be only briefly summarized here. The evidence that has been reviewed previously appears to indicate that the amplitude of delta oscillations increases in states of craving and decreases upon actual reward. From this point of view, administration of drugs of abuse must decrease the amplitude of delta oscillations. Indeed, acute administration of cocaine in rats results in diminished slow EEG power with this effect being mediated by dopamine (Chang et al., 1995; Ferger et al., 1994; Kiyatkin and Smirnov, 2009; Kropf and Kuschinsky, 1993; Luoh et al., 1994). However, in humans, the picture is more complex. While most studies note changes in delta power at different stages following the intake of these drugs, the direction of these changes is not straightforwardly reconcilable with the above-presented simple idea. It should be borne in mind, however, that in human beings, drugs of abuse produce complex effects which are not reducible to just simply turning on the brain's natural 'reward system' (Salamone and Correa, 2002).

In opioid-abusing individuals (Fink et al., 1971; Phillips et al., 1994; Volavka et al., 1970) and in surgical patients (Bovill et al., 1983; Scott et al., 1991; Sebel et al., 1981; Wauquier et al., 1984), morphine-like drugs increase wake EEG power in the delta and theta frequency range. Cocaine produces complex effects consisting of an increase in beta, alpha, and delta power (Herning et al., 1985, 1994; Lukas, 1991) and delta coherence over the prefrontal cortex (Reid et al., 2006), with delta and theta EEG activity being related to the rewarding properties of cocaine (Reid et al., 2003, 2006). Thus, it appears that in humans, contrary to what has been observed in rats, morphine-like drugs and cocaine produce an increase of delta power. This discrepancy may reflect the dynamics of craving and reward in humans that takes place after administration of these drugs. It appears that in humans, the initial stages after administration of drugs of abuse are associated with an increase of craving and, respectively, with an increase of delta activity (Reid et al., 2006). Indeed, the effects of cocaine on ratings of high and 'good drug effect' are rapid and somewhat short lasting and are accompanied by increased ratings of cocaine craving, which persist at or near their peak levels for a longer duration (Foltin and Haney, 2000; Hart et al., 2004; Reid et al., 2006; Ward et al., 1997). Moreover, evidence for a more prolonged effect on cocaine craving is consistent with previous neuroimaging study that reported a short high followed by a longer period of cocaine craving following intravenous dosing (Breiter et al., 1997). Most importantly, the increase of delta power during the first 5 min following cocaine was correlated with increased ratings of cocaine craving (Reid et al., 2006). Reward-related decrease of delta activity has been observed after administration of legal psycho-active drugs, such as alcohol (Sanz-Martin et al., 2011), tobacco (Knott et al., 2008) and caffeine (Hammond, 2003).

If we assume that reward-related dopamine release is associated with a decrease of delta activity, then abstinence in addicted persons should be associated with an increase of delta activity. Indeed, opiate addicts show increase of delta and decrease of alpha waves during abstinence (Shufman et al., 1996; Synytsky et al., 2002). Methamphetamine dependent patients with 4 days of abstinence also had increased EEG power in the delta and theta bands with no differences in the alpha and beta bands (Newton et al., 2003). MDMA ('ecstasy') abusers show significantly higher absolute delta power than control subjects (Herning et al., 2005). In a sample of Native Americans, increases in spectral power in the delta frequency range were significantly correlated with marijuana dependence (Ehlers et al., 2010). There is also evidence that high-binge alcohol drinkers exhibit more spectral power in the delta band (Courtney and Polich, 2010). Increases in delta and theta power were also observed during withdrawal from caffeine (Hammond, 2003).

On the other hand, alcoholic patients show decreased power in slow (delta and theta) bands both in wake (Coutin-Churchman et al., 2006; Saletu-Zyhlarz et al., 2004) and during SWS (Colrain et al., 2009a). Moreover, in contrast to non-alcoholic population, alcoholic patients with clinical depression show lower current density than non-depressed alcoholic patients in delta band (Coutin-Churchman and Moreno, 2008). EEG studies in cocaine-dependent patients show deficits in slow-wave brain activity and reduced interhemispheric coherence in delta and theta bands during drug-abstinence (Alper et al., 1998b; Prichep et al., 1996; Roemer et al., 1995). This seemingly contradicts the idea that craving in drug-abstinent alcohol- or cocaine-dependant humans

should be associated with enhanced delta activity. It should be borne in mind, however, that the clinical syndrome of drug dependence may include severe changes in the brain neurochemistry and electrophysiology as well as in motivational and emotional spheres which may pervert the observed relationships between psychological and physiological variables. For example, the EEG changes observed in the drug-abstinent, cocaine-dependant humans may reflect a neuroadaptation due to chronic use, possibly sensitization (Alper, 1999). Binienda et al. (2002) showed that in nonanesthetized, adult male rats, repeated exposures to cocaine resulted in a significant decrease in electrocorticogram delta power paralleled by marked increases in DA concentrations in caudate nucleus and frontal cortex. The DA turnover decreased significantly. Further studies are necessary to establish whether regional alterations in blood flow and metabolic activity may underlie such observations. Frequently observed increase of alpha and high-frequency oscillations in cocaine-dependent patients during drug-abstinence (Alper, 1999) might be attributed to high anxiety and depressive comorbidity in these samples in view or reports of increased alpha power both in anxiety (Herrmann and Winterer, 1996; Knyazev et al., 2004, 2005, 2006) and depression (Costa and Bauer, 1997; Alper, 1995).

Much evidence shows that event-related delta oscillations are disrupted in drug- or alcohol-dependent individuals as well. Thus, significantly lower amplitude of the P3 event-related potential (ERP) component has been reported for human subjects at high risk for alcohol dependence (for review, see Porjesz et al., 2005). Numerous studies have shown also that chronic substance use reduces the P3 amplitude and delays its latency. This applies to abstinent heroin (Attou et al., 2001; Papageorgiou et al., 2003, 2004) and cocaine (Biggins et al., 1997; Moeller et al., 2004) users. Even smoking has a significant reducing effect on P3 amplitude but only in the presence of the A1 allele of the D2 dopamine receptor gene locus (Anokhin et al., 1999). Many studies have demonstrated that delta and theta event-related oscillations (ERO) are the primary contributors to the P3 ERP component (Demiralp et al., 2001; Basar-Erogly et al., 1992; Karakas et al., 2000; Schurmann et al., 2001; Stampfer and Basar, 1985). These oscillations have been linked to several relevant genes associated with alcohol dependence phenotypes (Edenberg et al., 2004; Jones et al., 2004; Porjesz et al., 2005; Begleiter and Porjesz, 2006; Rangaswamy et al., 2007). Accordingly, several studies have shown that ERO measures of EEG activity recorded in P3 tasks provide comparable or even more powerful biomarkers of alcoholism than ERP measures (Andrew and Fein, 2010; Rangaswamy et al., 2007). It has been shown that the decrease in P3 amplitudes in genetic mouse models of high as compared to low alcohol preference is related to reductions in evoked delta oscillations energy and delta and theta phase locking (Criado and Ehlers, 2009). Beyond the P3 paradigm, it was found that alcoholics (Kamarajan et al., 2004) and their offspring (Kamarajan et al., 2006) showed significant reduction in event-related delta and theta power in the Go/No-Go paradigm and had lower evoked delta frequency responses to auditory stimuli during SWS (Colrain et al., 2009b).

In sum, much evidence shows that drugs of abuse definitely influence delta oscillations. Although in humans actual effect depends on a host of circumstances, such as the kind of drug, time after administration, mode of administration, previous experience with drugs, and so on, the general pattern fits the idea that actual reward associated with the dopamine release is accompanied by a decrease of delta activity whereas withdrawal and craving are associated with an increase of delta activity. Animal data show that delta oscillations are diminished by electrical stimulation of the VTA and acute administration of cocaine with this effect being mediated by dopamine (Chang et al., 1995; Ferger et al., 1994; Kropf and Kuschinsky, 1993; Leung and Yim, 1993; Luoh et al., 1994). In the context of neuroimaging, a direct linkage between delta-band activity and fMRI signal in the nucleus accumbens of the rat brain after heroin challenge was reported by Li et al. (2006). Chronic alcohol or cocaine use causes neuroadaptation, possibly sensitization (Alper, 1999), which results in a decrease in delta power and disruption of event-related delta responses.

3.2.1.4. Reward deficiency and delta oscillations.

3.2.1.4.1. Anhedonia. There is considerable evidence suggesting that the nucleus accumbens reward system is dysfunctional in patients who suffer from depression (Berton et al., 2006; Tremblay et al., 2005). We have already discussed the evidence showing increase of delta power in patients with major depressive disorder. Pizzagalli et al. (2004) have shown that melancholia, a subtype of depression characterized by anhedonia, was associated with increased delta activity in the subgenual prefrontal cortex. Following antidepressant treatment, depressed subjects with the largest reductions in depression severity showed the lowest post-treatment subgenual prefrontal cortex delta activity. Wacker et al. (2009) have shown that delta activity in the rostral anterior cingulate cortex correlates negatively with the nucleus accumbens responses to reward and is positively associated with anhedonia scores. Knyazev (2011) using independent component analysis and source localization techniques showed that when subjects expected bad news delta power and connectivity increased in a network of cortical areas which includes the orbitofrontal and the anterior cingulate cortices as its main node. This increase was more pronounced in subjects with higher scores on state anxiety. These data imply that frontal medial delta activity increases when this area receives less dopaminergic firing from the nucleus accumbens. In the domain of evoked responses, the most common ERP finding in depression is a reduction of P3 amplitude during the oddball task (Bruder et al., 1991; Gangadharar et al., 1993; Urretavizcaya et al., 2003).

3.2.1.4.2. Reward deficiency syndrome and impulsive behavior. Comings and Blum (2000) have proposed that defects in various combinations of the genes for 'reward' neurotransmitters result in a Reward Deficiency Syndrome (RDS) and that such individuals are at risk for abuse of the unnatural rewards including alcohol, drug, tobacco, and food and other related behaviors, such as pathological gambling, Tourette's syndrome, and ADHD (Blum et al., 2008). All these behaviors frequently co-occur (Comings et al., 2005) and are associated with traits that fall into category of impulsivity or sensation seeking (Cloninger, 1987; Gray, 1987; Zuckerman, 1994). Behavioral impulsivity is characterized by intolerance of delayed reward (Corr, 2002). Cloninger (1987) suggested lower baseline dopaminergic activity in high sensation seeking individuals. Indeed, tonic dopamine release is supposedly linked with a prediction or anticipation of delayed future rewards (Grace, 1995) and it is shown that dopamine depletion shifts the preference in favor of the small 'proximal' rewards (Wade et al., 2000; Cardinal et al., 2001) thus making the animal behavior more 'impulsive'. From the perspective of this review, it is suggested that the RDS and impulsivity must have excess of slow waves in EEG as one of its markers. The association between delta waves and addictive behavior has been already discussed above. The prevalence of slow waves (both theta and delta) in EEGs of ADHD subjects is also well established (see e.g., Barry et al., 2003 for a review). It is also shown that stimulant medication with drugs targeted to dopamine circuits, such as methylphenidate, frequently improves symptomatology, concomitantly 'normalizing' the EEG (Clarke et al., 2002a). Moreover, good responders to methylphenidate had EEG profiles that suggested that they were more cortically 'hypoaroused' (i.e., had more slow waves) than poor responders (Clarke et al., 2002b).

Considerable literature links unconstrained urge towards biological rewards with impulsive, aggressive, and antisocial behavior. Prevalence of slow-wave activity in the EEG of aggressive,

antisocial, and criminal individuals was noted in earlier observations and was confirmed in later quantitative EEG studies (see Knyazev, 2007 for a review). In addition to the slowing of 'spontaneous' EEG, considerable literature shows impairment of the evoked P3 responses in impulsive and antisocial individuals (Bond and Surguy, 2000; Gao and Raine, 2009; Kiehl et al., 2006; Mathias and Stanford, 1999; Raine and Venables, 1987). The behavior and EEG abnormalities in violent and antisocial persons tend to be explained as a consequence of hypoarousal. Hare (1970) suggested that antisocial individuals were hypoactive compared with normal individuals and consequently existed in a chronic stage of 'stimulus hunger'. The hypoarousal hypothesis seems to stem from Eysenck's (1967) theory which suggested that individual differences in the activity of the corticoreticular loop are responsible for the individual's position on the extraversion–introversion dimension. Interestingly, for an EEG index of hypoarousal Eysenck suggested high amplitude alpha oscillations whereas the above theories imply that hypoarousal is manifested by increase of slow waves which is frequently accompanied by a decrease of alpha oscillations. The hypoarousal theory of delinquent and aggressive behavior is supported by autonomic regulation data consistently showing low skin conductance (for a review see Raine, 1993) and heart rate in antisocial population (see e.g. a meta-analysis by Ortiz and Raine, 2004). However, the low autonomic arousal registered in the antisocial population does not necessarily mean that it itself causes aggressive and antisocial behavior. Existing evidence rather suggests that anger-induced or emotionally driven aggression is facilitated by increased arousal, which essentially acts as an energizer of ongoing behavior (for a review see Zillman, 1983). Scarpa and Raine (1997) suggested that under-arousal may be more relevant to milder forms of aggression, whereas overarousal is more relevant to the facilitation of emotional aggression. One plausible explanation of low autonomic arousal registered in the antisocial population during mildly stressful psychophysiological test sessions would be low level of fear and anxiety (Ortiz and Raine, 2004) which also implies a low sympathetic tone. In framework of the evolutionary approach, prevalence of slow oscillations could be considered (in a metaphorical sense) as an evidence of 'sleepiness' of higher regulatory mechanisms. Thus, a 'lazy' frontal lobe has been suggested to underlie disinhibition of motor activity in ADHD patients (Niedermeyer and Naidu, 1998a,b). However, linking delta oscillations with inhibition is not supported by recent evidence showing positive correlations of both SWS (Dang-Vu et al., 2008) and waking (Lu et al., 2007) delta with brain activity.

Summing up, it appears that need for reinforcement increases delta oscillations whereas actual reinforcement causes their decrease. Pathological states associated with anhedonia or reward deficiency are associated with increase of slow waves in 'spontaneous' EEG and impairment of slow wave-related evoked responses.

3.2.2. Delta oscillations and primitive defensive mechanisms

The behavior of lower vertebrates is not guided exclusively by reward motivation. The delta oscillatory system is also expected to participate in atavistic defensive responses and reactions to unavoidable aversive stimuli. The most primitive circuits that coordinated defensive responses in ancient vertebrates are located within the periacqueductal gray region (PAG). As a primitive defensive mechanism, there are several noteworthy characteristics of the PAG (Bandler and Keay, 1996). First, cells in this region are relatively cut off from direct exteroceptive sensory input and are primarily responsive to somatosensory and visceral information. Moreover, there appears to be a specialization for painful input, with different sets of cells responding predominantly to cutaneous pain (lateral PAG) and to deep pain from the joints and viscera (ventrolateral PAG). These sensory inputs suggest that, at its most primitive levels, defensive behavior is based upon stimulation that actually contacts the individual and requires an immediate response (Luu et al., 1998). The following is a brief discussion of two examples of the PAG-related behaviors and their association with the EEG delta activity. These are the states of panic attacks and the enduring pain.

A number of researchers have related activity within primitive structures such as the PAG to emotional states of 'panic' (Barlow et al., 1996; Gray and McNaughton, 1996; Heller et al., 1997). These states, which are often triggered by somatic sensations (e.g., breathlessness), involve an abrupt onset, rapidly intensifying autonomic reactions, feelings of immediate danger, and an overwhelming urge to escape. If, as is hypothesized here, delta oscillatory system is linked with evolutionary primitive behavioral patterns, panic should be associated with increase of its activity. And, vice versa, in individuals predisposed to panic attacks, increased background delta activity should favor appearance of these attacks. Existing evidence seems to confirm such a claim. Thus, patients vulnerable to lactate-induced panic exhibit higher than normal pre-panic autonomic activity, elevated autonomic-somatic activity during lactate-induced panic and an EEG response to provoked panic which appears to be comprised of a 'paradoxical' shift towards slow-wave delta activity and an altered brainstem evoked response (for a review see Knott and Lapierre, 1988). EEG recordings throughout a lactate challenge indicate that slow-wave activity associated with panic does not appear to be characterized by an abrupt, sudden onset but tends to increase gradually throughout the infusion (Knott, 1990). In patients with panic disorder who experience panic attacks during sleep, all the panic attacks occur suddenly, arising from stage 3 to 4 (delta sleep) or during a transition from stage 2 towards delta sleep (Lesser et al., 1985; Mellman and Uhde, 1989). In a study designed to examine the sensitivity of different sleep stages to the pharmacological provocation of nocturnal panic attacks, cholecystokinin tetrapeptide (CCK-4), which demonstrates an abrupt onset of action making it possible to provoke panic attacks precisely during a particular sleep stage, was used as a challenge agent (Kronenberg et al., 2001). In a balanced cross-over design, healthy participants were challenged with identical doses of CCK-4 both during REM sleep and during delta sleep. Stimulation with 50 microg CCK-4 during REM sleep failed to elicit a full-blown panic awakening, while the same dose, administered during delta sleep, did produce full-blown panic attacks. Similarly, stimulation of six subjects with 100 microg CCK-4 during REM sleep resulted in only one panic response, whereas four of nine subjects awoke experiencing a panic attack following stimulation with the identical dose during delta sleep. Severity of panic symptomatology, as measured by the self-rated Acute Panic Inventory, was also significantly increased when CCK-4 was administered during delta sleep. The authors conclude that sensitivity to the panicogenic effects of CCK-4 seems to be higher during delta sleep than REM sleep.

Short-lasting painful stimuli mostly induce appearance of high-frequency oscillations although parametric spectral analysis of late cerebral potential components (later than 80 ms) evoked by brief painful somatosensory stimuli does reveal a stimulus-induced increase of power in the low frequencies, delta and theta (Bromm et al., 1989). In this placebo-controlled double-blind cross-over study with 20 healthy male subjects, the effect kinetics of the opioid meperidine and the antidepressant imipramine on spontaneous EEG activity and EEG activity that was evoked by brief painful electrical stimuli were investigated. Both drugs increased the power in the low frequencies in the pre-stimulus and decreased it in the post-stimulus EEG, thus effectively abolishing the EEG effect of painful stimulation. The pre-/post-stimulus relationship of the delta power was found to be the most sensitive measure for monitoring the cerebral bioavailability of the tested drugs (Bromm et al., 1989). Increase of delta power and coherence was observed during tonic

pain evoked by the cold pressor test (Chang et al., 2002; Chen et al., 1998; Ferracuti et al., 1994; Stevens et al., 2000), by a series of heat pulses (Huber et al., 2006), during tonic experimental muscle pain (Le Pera et al., 2000), in patients with chronic pancreatitis (Olesen et al., 2011), and in migraine patients (Bjørk et al., 2009; Genco et al., 1994; Muellbacher and Mamoli, 1994; Passier et al., 1994; Ramelli et al., 1998; Sand, 1991; Seri et al., 1993; Siniatchkin et al., 1999; Thomaides et al., 1996).

All these data appear to confirm that primitive defensive behaviors associated with PAG activity are accompanied by an increase of delta oscillations.

3.3. Cognitive processing and delta oscillations

In sharp contrast to clinical EEG researchers who tend to treat delta oscillations as a correlate of pathological processes or inhibition, cognitive neuroscientists tend to consider delta as a 'cognitive' rhythm (e.g., Basar et al., 1999; Schurmann et al., 2001). Perhaps the most significant finding in cognitive studies concerning the delta activity is its commonality with the P3, an evoked response to stimuli that are unexpected, infrequent, or motivationally salient. This association has already been briefly discussed above and the relevant literature has been reviewed elsewhere (e.g., Basar-Erogly et al., 1992; Knyazev, 2007). In short, delta and theta ERO have been shown to be the primary contributors to the P3 ERP component (Demiralp et al., 2001; Basar-Erogly et al., 1992; Karakas et al., 2000; Schurmann et al., 2001; Stampfer and Basar, 1985). In view of the evidence linking the P3 with dopaminergic neurotransmission (Berman and Noble, 1997; Berman et al., 2006; Blum et al., 1994; Mulert et al., 2006; Noble et al., 1994), Knyazev (2007) suggested that the motivational relevance of the task and the salience of the target stimulus seem to be related to the enhanced delta activity in the P3 paradigm, because the brain motivational systems signal salience and make the brain paying attention to biological relevant stimuli (Gray, 1999). This interpretation implies an important role for delta oscillations in attention. Indeed, many investigators have linked delta oscillations with attention processes (Harmony et al., 1996; Schroeder and Lakatos, 2009; Lakatos et al., 2008; Will and Berg, 2007). The entrainment of cortical delta oscillations has been suggested as a key mechanism of selective attention to rhythmic auditory or visual stimulus streams (Lakatos et al., 2008). Interestingly, a recent combined fMRI and EEG study has shown that the resting state networks (RSN) associated with higher cognitive functions such as self reflection, working memory and language all displayed a positive association with higher EEG frequency bands while negatively related to delta and theta. In contrast, the RSNs that delineate the sensory cortices, i.e. somatomotor, auditory and visual cortices showed positive associations with the lower EEG frequencies but negative association with the higher frequencies (Jann et al., 2010).

If, as the evidence discussed in the previous sections implies, delta oscillations are associated with basic motivational processes, they might be involved in constant screening of internal and external stimuli in search of motivationally salient cues that signal potential threat or reward. Existing evidence implies that this screening does not stop even in deep sleep (Basar, 1999) and includes stimuli that fall below the threshold of conscious perception. Study of evoked oscillations on the hearing threshold in waking humans show that the stimulus intensity is one of the factors determining which frequency component of the auditory ERP is dominant. At high stimulation intensities, the dominant frequency component is in the alpha to theta range. At 40–20 dB stimulation, the dominant frequency component is in the theta range. At threshold stimulation, the frequency characteristics are dominated by a clear delta activity (Parnefjord and Basar, 1999). A waveform observed in response to deviant stimuli not attended by the subject, the mismatch negativity (Naatanen, 1992) is shaped by a delayed delta response superimposed with a theta response. Interestingly, for near-threshold visual stimuli, it has been shown that the pre-stimulus state of alpha oscillations determines whether the stimulus will be consciously perceived or not (Ergenoglu et al., 2004). Thus, the conscious perception depends on alpha activity whereas subconscious perception could be attributed to the low frequency oscillations. Much data show that delta (along with theta) oscillations are also implicated in processing emotional stimuli (e.g., Bhattacharya and Petsche, 2002; Klados et al., 2009).

In sum, it appears that existing data linking delta oscillations with cognitive processes do not contradict the defended in this review idea that these oscillations are mostly associated with evolutionary old basic motivational processes. Indeed, much psychological data show that cognition in humans is inseparably linked with motivation and emotion and the above reviewed evidence emphasizes that the involvement of delta oscillations in cognitive processes is mostly limited to the function of salience detection which is undoubtedly related to motivational brain circuits.

3.4. Conclusion

The reviewed evidence appears to confirm the involvement of delta oscillations in motivational processes. Generation of delta oscillations seems to depend on activity of the VTA, the nucleus accumbens, the medial prefrontal cortex, and the nucleus reticularis thalami, all of which are implicated in the brain reward system. Delta oscillations appear to increase in states of motivational urges, which are triggered by biological rewards and dangers. They are implicated in attention and salience detection. All this fits to the hypothesis that delta oscillations represent the most basic evolutionary old oscillatory mode which dominated in reptiles but is still associated with very important primary processes in humans.

4. General conclusion

In this review, the evidence on functional correlates of delta oscillations has been reviewed from an evolutionary point of view. Predictions derived from the evolutionary interpretation were formulated and matched against existing empirical evidence. In spite of scarceness of this evidence, the general pattern appears to fit the idea that delta oscillations represent an evolutionary old oscillatory mode that dominated in lower vertebrates, but in waking adult humans it is overshadowed by more advanced and operationally flexible processes associated with higher frequency oscillations. This oscillatory mode rises in activity when for some or another reason the more advanced systems lose their priority. These conditions include earlier developmental stages, sleep, and an array of pathological states. Functionally delta oscillations appear to be implicated in synchronization of brain activity with autonomic functions, in motivational processes associated both with reward and atavistic defensive mechanisms, and in cognitive processes mostly related to attention and the detection of motivationally salient stimuli in the environment. Although mostly speculative, the evolutionary interpretation has considerable explanatory value and allows unify and explain seemingly isolated and sometimes even contradictory data coming from different research domains.

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