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EEG-Biofeedback as a Tool to Modulate Arousal: Trends and Perspectives for Treatment of ADHD and Insomnia

B. Alexander Diaz, Lizeth H. Sloot,
Huibert D. Mansvelder and Klaus Linkenkaer-Hansen
Department of Integrative Neurophysiology, Center for Neurogenomics and Cognitive Research (CNCR), Neuroscience Campus Amsterdam, VU University Amsterdam, Amsterdam
The Netherlands

1. Introduction

EEG-biofeedback (EBF) is a method to provide information about a person's brain state using real-time processing of electroencephalographic data (Budzynski, 1973; Morin, 2006). The idea behind EBF training is that by giving the participant access to a physiological state she will be able to modulate this state in a desired direction. As such EBF makes use of a brain-computer interface (BCI), in itself a field of study that has seen rapidly growing interest over recent years (Felton et al., 2007; Kübler, Kotchoubey et al., 2001; Leuthardt et al., 2006; Schalk et al., 2007). There is a distinction between using BCI to gain control over an external device or to use it to modify the internal state of the user. The former has seen fascinating applications in facilitating control of prosthetics (Nicolelis, 2003) or in offering new channels of communication to the paralysed (Birbaumer et al., 1999; Krusienski et al., 2006; Krusienski et al., 2008). EEG biofeedback belongs to the latter category as it aims to provide a means for the user to modify her own cognition or behaviour through feedback on specific EEG characteristics (Fig. 1). EBF therapy should, after repeated training, result in improved brain states or an effective internalized strategy to invoke such a brain state.

EEG-biofeedback (EBF) was first used in operant conditioning studies on cats in the 1960s. By rewarding the generation of the sensori-motor rhythm (SMR, Table 1), cats learned to increase SMR by suppression of voluntary movement (Roth et al., 1967; Sterman et al., 1969; Sterman & Wyrwicka, 1967; Wyrwicka & Sterman, 1968). Interestingly, a lasting effect of the biofeedback training became apparent when the same cats were later used in a dose-response study of an epileptogenic compound in which they showed significantly elevated seizure thresholds (Sterman, 1977; Sterman et al., 1969). These serendipitous findings motivated the use of biofeedback in research on humans with epilepsy (Sterman, 2006). Because the EEG is altered in several other disorders, biofeedback research has expanded to a range of clinical disorders including addiction (Passini et al., 1977; Peniston & Kulkosky, 1989; Saxby & Peniston, 1995), anxiety (Angelakis et al., 2007), attention-

deficit/hyperactivity disorder, autism (Coben & Padolsky, 2007; Pineda et al., 2008), depression (Baehr et al., 1997; Hammond, 2005), post-traumatic stress disorder (Peniston & Kulkosky, 1991), and sleep disorders (Cortoos et al., 2009). More recently, research has explored the potential of biofeedback to enhance normal cognition, e.g. to improve attention (Egner et al., 2002; Gruzelier et al., 2006), working memory (Hoedlmoser et al., 2008; Vernon et al., 2003), or athletic performance (Egner & Gruzelier, 2003; Vernon, 2005).

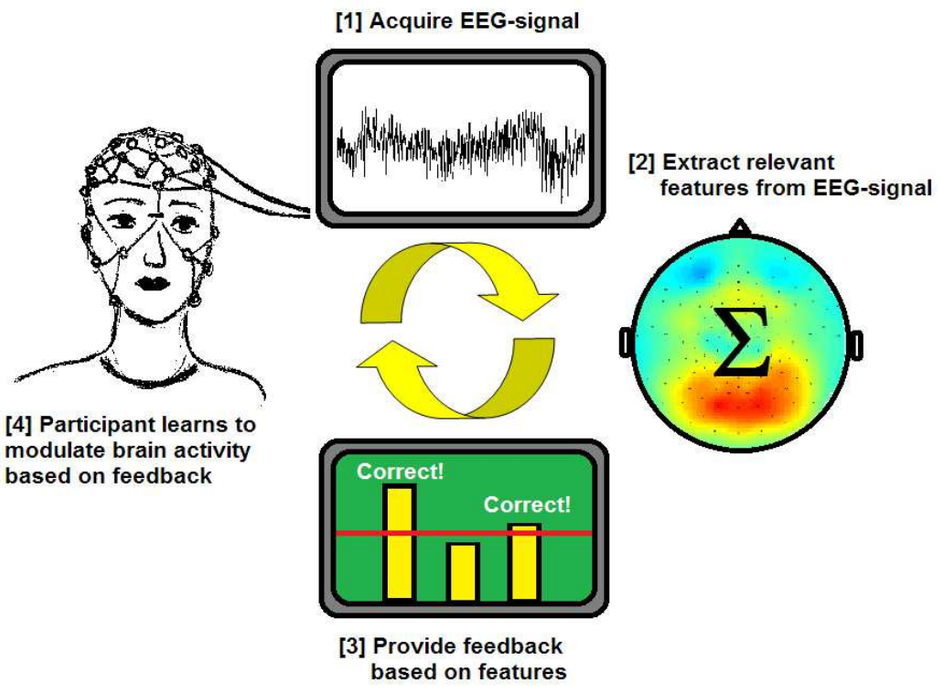

Fig. 1. The concept of EEG-biofeedback. The EEG is recorded [1], a suitable EEG-biomarker is extracted [2] and made available to the participant and correct changes in brain activity are rewarded by, e.g., a visual stimulus indicating success [3]. With repetition, this enables the participant to learn what strategies to employ in order to change brain activity in the desired direction [4].

In spite of the many studies using EBF to improve a clinical condition, the concept awaits a solid theoretical framework and the efficacy of EBF therapy requires further validation to gain widespread acceptance. Nevertheless, EBF holds the prospects to become an alternative to pharmaceutical intervention, where side-effects and dependency are prominent risks. An efficient EBF protocol that enables learning with a moderate number of sessions, will not only be more cost-effective but may bear additional psychological benefits such as avoiding certain stigmata (requiring psychiatric consultation or medication) and giving the participant more control over his/her own treatment. It is also conceivable that the mechanism with which EBF training exerts its therapeutic action is distinct from drug treatment as has been observed, e.g., when comparing neurobiological changes following successful treatment of depression using either cognitive behavioural therapy (CBT) or medication (Kumari, 2006).This would raise the perspective that EBF could be of help to those patients that do not respond to medication.

In this chapter, we focus on two disorders that share a characteristic arousal component, which EEG-biofeedback therapy attempts to modulate: attention-deficit hyperactivity disorder (ADHD) and insomnia.

Band	Frequency range (Hz)	Hallmark
δ	0.1–4	Sleep (stages N3-N4)
θ	4–8	Drowsiness, Sleep (stages N1-N2)
α	8–13	Relaxed wakefulness, cortical idling
σ	12–14	Spindle range (N2)
SMR	12–15	Sensorimotor rhythm
β	13–30	Cognitive effort, alertness

Table 1. All EEG bands from delta to beta have proven relevant for EBF in ADHD and insomnia.

ADHD has been described as a disorder of decreased CNS arousal and cortical inhibition, partially explaining the symptom normalizing effect psychostimulants have in the treatment of ADHD (Satterfield et al., 1974). These arousal deficits become manifest in lowered skin conductance levels (Barry et al., 2009; Raine et al., 1990; Satterfield et al., 1974), EEG deviations (e.g. increased theta but less beta activity) (Barry et al., 2003a; Barry et al., 2003b; Clarke et al., 2002; Clarke et al., 2001) and are related to CNS dopamine systems and associated genes (Li et al., 2006).

Insomniacs in contrast, exhibit elevated (cognitive) arousal effectively delaying the transition from wakefulness to sleep or resulting in frequent awakenings, oftentimes directly related to persistent (psychological) stressors (Bonnet, 2010; Bonnet & Arand, 1997; Bonnet & Arand, 2005; Cortoos et al., 2006; Drake et al., 2004; Drummond et al., 2004; Jansson & Linton, 2007; Nofzinger, 2004; Perlis, 2001). Brain areas involved in sleep regulation, arousal and attention are closely related (Brown et al., 2001) possibly explaining the observation that 50% of ADHD children also have difficulties falling asleep and 20% report recurring severe sleep problems (Ball et al., 1997; Stein, 1999). The association between arousal and sleep has classically been described using the EEG, where elevated arousal is associated with beta and gamma (>30 Hz) activity, whereas decreases in arousal are associated with enhanced delta and theta band activity (Alkire et al., 2008; Rechtschaffen & Kales, 1968; Steriade et al., 1993).

Here we propose that for EBF to have a therapeutic effect it is required that (1) EEG can index (disease-)relevant states of the brain, (2) one can learn to modulate these brain states, (3) training the modulation of brain states causes (lasting and desired) changes to the brain, and (4) EBF-related changes to the brain have cognitive and/or behavioral correlates. In the following, ADHD and insomnia are treated as case examples of disorders that have been proposed to benefit from EEG-biofeedback therapy. We present the evidence that EBF has a therapeutic effect on these disorders and outline trends and perspectives by reviewing recent progress in the design of EBF for pre-clinical research.

2. EEG-biofeedback in ADHD

Attention deficit/ hyperactivity disorder (ADHD) is a psychiatric disorder, characterized by symptoms of inattention and/or impulsivity and hyperactivity. These symptoms frequently co-exist with emotional, behavioural and learning deficits such as conduct disorder and oppositional defiant disorder, anxiety disorders and major depressive disorder (Barry et al., 2003). Prevalence in school-aged children is fairly high (3–12%) (Brown et al., 2001) and 30–50% of these children will continue to experience symptoms into adulthood (Barry et al., 2003; Monastra, 2005). DSM-IV criteria allow the distinction of three ADHD subtypes: (1) the predominantly inattentive type, (2) the predominantly hyperactive-impulsive type and (3) the combined type, which exhibits symptoms of both inattention and hyperactivity-impulsivity (DSM-IV-TR; American Psychiatric Association, 2000).

Pharmacological intervention based on psychostimulant medication leads to a reduction of ADHD symptoms by increasing CNS arousal (Satterfield et al., 1974), but lacks long-term efficacy (Faraone & Buitelaar, 2010; Faraone & Glatt, 2010; Molina et al., 2009) and introduces adverse effects in 20-50% of the patients (Charach et al., 2004; Efron et al., 1997; Goldstein & Goldstein, 1990). Still, 35–45% of the patients with an "inattentive" type of ADHD and 10–30% of those diagnosed as "combined" type do not respond to medication, limiting the effectiveness of pharmaceutical intervention (Barkley, 1998; Hermens et al., 2006; Swanson et al., 1993). EEG biofeedback therapy for ADHD is one proposed alternative treatment and aims at restoring CNS arousal imbalances by training participants to suppress EEG rhythms associated with underarousal and enhance those rhythms associated with attention (J. F. Lubar & Shouse, 1976; Monastra et al., 2005; Thompson & Thompson, 1998).

2.1 Training duration and feedback

An EBF training session consists of repeated training blocks of typically 3 minutes, each starting with a measure of baseline activity, like 5 minutes eyes-closed rest (J. O. Lubar & Lubar, 1984), within the specified frequency band in order to establish a target threshold value (Table 2). The participant will then attempt to match or exceed this value during a subsequent feedback trial by modulating activity within the set frequency band. The participant need not be aware of the underlying parameter(s) and is merely instructed to meet/exceed the threshold. Participants are encouraged to find their own optimal strategy to alter the brain activity. When the participant successfully exceeds the threshold, e.g., for 0.5 s (Monastra, 2005), a reward signal indicating success (e.g. a bonus point that can be traded for money or toys) is presented to reinforce learning. ADHD patients prefer smaller and immediate rewards to delayed, but larger ones (Loo & Barkley, 2005; Marco et al., 2009; Tripp & Alsop, 2001) and as the ADHD population largely consists of children, feedback protocols often involve video games where success is rewarded instantly (Drechsler et al., 2007; Leins et al., 2007).

2.2 Target brain activity

Spontaneous (resting-state) EEG profiles of ADHD children differ significantly from those of normally developing children, especially increased theta/beta ratio but also lowered alpha band activity has been reported (Barry & Clarke, 2009; Barry et al., 2003; Barry et al., 2009; Barry et al., 2003; Clarke et al., 2002; Clarke et al., 2001).

The increased theta/beta ratio has been proposed as a characteristic biomarker for CNS underarousal (Mann et al., 1992), whereas the SMR has been classically described as reflecting motor inhibition (Sterman & Friar, 1972; Sterman et al., 1970). The vast majority of EBF studies has been inspired by a two-phase protocol of Lubar et al. (1984), in which participants where first trained to increase their SMR and later to inhibit theta activity while simultaneously increasing beta activity (Beauregard & Levesque, 2006; Carmody et al., 2000; Fuchs et al., 2003; Gevensleben et al., 2009; Heywood & Beale, 2003; Holtmann et al., 2009; Kaiser, 1997; Kaiser & Othmer, 2000; Kropotov et al., 2005; La Vaque et al., 2002; Leins et al., 2007; Levesque et al., 2006; Linden et al., 1996; J.F. Lubar et al., 1995; Monastra et al., 2002; Rossiter, 2004; Rossiter, 1998; Rossiter & La Vaque, 1995; Strehl et al., 2006; Thompson & Thompson, 1998).

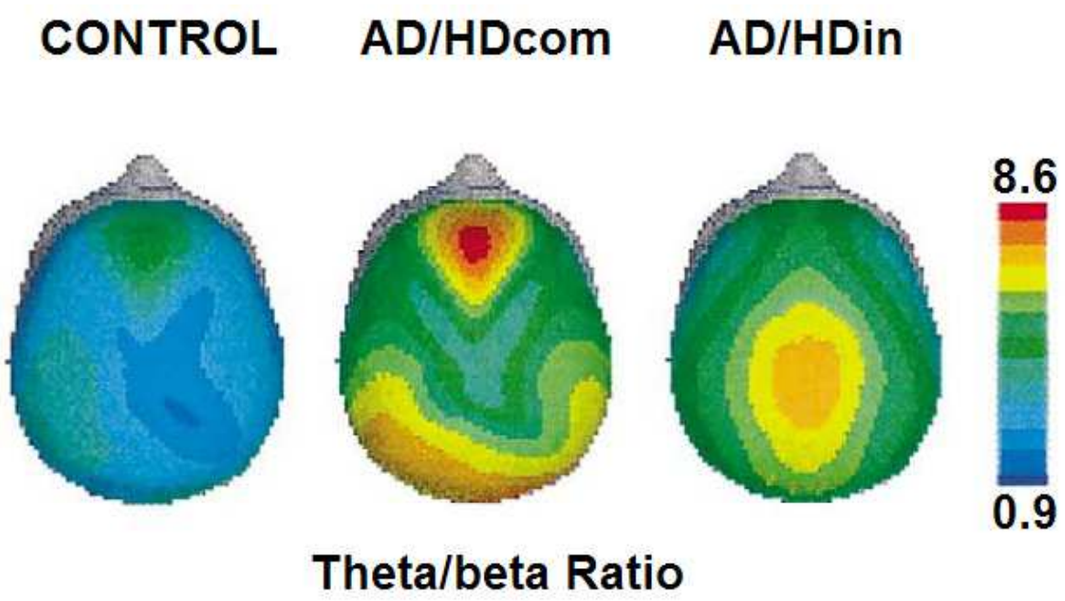

Fig. 2. Brain activity profiles in children with ADHD differ from healthy controls. Theta/beta-band activity ratio is strongly elevated in ADHD, but differs in spatial localization between combined (AD/HDcom) and inattentive (AD/HDin) subtypes. (From: Barry et al., 2003.).

In recent years, however, an interesting new target for EBF has been found in the form of slow cortical potentials (SCPs). These slow event-related DC shifts represent excitation thresholds of large neuronal assemblies and training ADHD patients to increase SCPs robustly improves symptoms of ADHD (Doehnert et al., 2008; Drechsler et al., 2007; Gevensleben et al., 2009; Heinrich et al., 2007; Kropotov et al., 2005; Leins et al., 2007; Siniatchkin et al., 2000; Strehl et al., 2006).

Study	Control P/R/B*1)	N (m)	Age	Electrodes /Ref	Freq.	Stim/ Reward	#Ses./ Dur.
Monastra et al., 2002	-/-/-	[1] 49(40) [2] 51 (43)	[1] 10.0± 3.7 [2] 10.0± 3.1	CPz & Cz/A2	β ↑ θ ↓	Visual & auditory /Money	43 (34–50) / 30–40 min
Fuchs et al., 2003	-/-/-	[1] 12(12) [2] 22(21)	[1] 9.6±1.2 [2] 9.8±1.3	C3 or C4/A1+A2	SMR↑ /β ↑ θ ↓	Visual & auditory /Points	36 / 30-60 min
Rossiter, 2004	-/-/-	[1] 31(21) [2] 31(22)	[1] 16.7±12.5 [2] 16.6±12.7	C3/A2 or C4/A1	β ↑ θ ↓	Visual & auditory	40 or >60 /30 or 36 min
Lévesque et al., 2006	+/+/-	[1] 5(5) [2] 14(11)	[1] 10.2±0.8 [2] 10.2±1.3	Cz/A1	SMR↑ θ ↓	Visual & auditory (video game)	40 / 60 min
Drechsler et al., 2007	-/-/-	[1] 13(10) [2] 17(13)	[1] 11.2±1.0 [2] 10.5±1.3	Cz/A1+A2	SCP↑/↓	Visual /Points	2x15 /2x45 min
Leins et al., 2007	-/+/+	[1] 16(13) [2] 16(13)	[1] 9.16±1.43 [2] 9.16±1.53	[1] CF3,CF4/ A1+A2 [2] Cz/A1+A2	[1] β ↑ θ ↓ [2] SCP↑/↓	Visual /Points	30 /60 min
Doehnert et al., 2008	-/-/-	[1] 12(10) [2] 14(12)	[1] 11.4±0.9 [2] 10.8±1.3	Cz/A1+A2	SCP↑/↓	Visual /Points	2x15 /2x45 min
Gevensleben et al., 2009	-/+/-	[1] 35(26) [2] 59 (51)	[1] 9.3± 1.16 [2] 9.8± 1.25	Cz/A1+A2	β ↑ θ ↓ SCP↑/↓	Visual	2x9 /2x 50 min

*1) **P/R/B= Placebo/Randomized/Blind (- = no, + = yes). N(m):** Number of participants (males), **Freq.:** Target frequency ↑/↓ (increase/decrease) of EBF condition(s).

Table 2. EBF therapy focused at treating ADHD is an active field of research.

2.3 Efficacy of EEG-biofeedback in the treatment of ADHD

The first study of EBF in ADHD (J. F. Lubar & Shouse, 1976) reported improved attention and normalized levels of arousal, together with improved grades and achievement scores for the (eight) children under treatment. Subsequent studies have reported similarly positive results, showing improvements of behaviour, attention and impulsivity (Alhambra et al., 1995; Carmody et al., 2000; Drechsler et al., 2007; Gevensleben et al., 2010; Gevensleben et al., 2009; Heinrich et al., 2004; Kaiser & Othmer, 2000; Kropotov et al., 2005; Leins et al., 2007; Linden et al., 1996; J.F. Lubar et al., 1995; J. F. Lubar, 1991; Rossiter, 1998; Rossiter & La Vaque, 1995; Strehl, et al., 2006; Thompson & Thompson, 1998; Doehnert et al., 2008). Efficacy of EBF is comparable to psychostimulant medication and group (CBT) therapy programs with effects lasting 6 months and longer (Fuchs et al., 2003; Gani et al., 2009; Gevensleben et al., 2010; Kaiser, 1997; Leins et al., 2007; Linden et al., 1996; J.F. Lubar et al., 1995; Monastra et al., 2002; Rossiter & La Vaque, 1995; Thompson & Thompson, 1998). Overall, EBF treatment results in clinical improvement in about 75% of the cases, without any reported adverse effects so far (Leins et al., 2007; Monastra et al., 2005).

It should be noted, however, that the use of the theta/beta ratio as marker of general arousal has been questioned, because it does not correlate with skin conductance level (R.J. Barry & Clarke, 2009; R.J. Barry et al., 2009). Similarly, SCPs are no direct correlates of arousal but rather represent attentional processes (Siniatchkin et al., 2000). This raises the interesting notion that in ADHD, EBF may not restore or modulate arousal systems per se, but compensate underarousal by strengthening cognitive functions that have been negatively affected by the arousal dysfunction.

3. EBF as treatment of insomnia

Insomnia is a most pervasive disorder, affecting about 15% of the general population while 6% meet clinical (DSM-IV) criteria (Ohayon, 2002) and interferes with cognition, quality of life, job performance and represents a multi-billion dollar burden on healthcare providers (Daley et al., 2009; Ebben & Spielman, 2009; Edinger et al., 2004). Insomnia can be subdivided into primary and co-morbid insomnia with the most salient symptoms being difficulty initiating and/or maintaining sleep (Espie, 2007). Causes of primary insomnia include physiological, cognitive and behavioural factors (Espie, 2007). Symptoms and duration are related to severity and persistence of stressors (Morin et al., 2006).

To better understand the possible therapeutic targets of insomnia, the so-called "3P model" has been proposed (Ebben & Spielman, 2009). This model specifies three categories of factors influencing the risk at developing or worsening insomnia: predisposing, precipitating and perpetuating factors. The first category constitutes genetic factors or personality traits, such as increased basal level of anxiety or hyperarousal (Drake et al., 2004), whereas precipitating events represent work and educational stress together with health and emotional problems (Bastien et al., 2004). Finally, perpetuating factors, such as continuous stress and poor sleep hygiene, may cause the actual transition to chronic insomnia and complete the vicious circle.

Pharmacological treatment of insomnia with sedative-hypnotic agents has seen a steady decline over the past (Aldrich, 1992; Walsh & Schweitzer, 1999), because of side effects, discontinuation discomfort, and the risk of developing drug tolerance or dependency (Ebben & Spielman, 2009; Walsh & Schweitzer, 1999). Alternative treatment options that have been met with success are cognitive-behavioural therapy (CBT) (Ebben & Spielman, 2009; Espie, 1999; Morin et al., 1999; Morin et al., 1994; Murtagh & Greenwood, 1995; Siebern & Manber, 2010) or treatments

increasing body temperature (e.g., physical exercise, hot bath before bed), which has recently been shown to hasten sleep onset (Van Someren, 2006). Whereas CBT causes sustained improvements and reduces sleep complaints, one fifth of the patients does not respond to the intervention (Cortoos et al., 2010; Harvey & Payne, 2002; Morin, 2006). EBF therapy for insomnia could be a safer alternative to medication and may offer treatment where CBT fails.
The EEG profile of insomniacs (Fig. 3) consists of increased levels of beta activity especially during the sleep-onset period and early sleep stages (Merica et al., 1998). These observations may be interpreted as evidence of cognitive hyperarousal, which is in line with the often reported 'racing thoughts' of insomniacs (Bastien et al., 2003; Buysse et al., 2008; Buysse et al., 2008; Freedman, 1986; Harvey & Payne, 2002; Jacobs et al., 1993; Lamarche & Ogilvie, 1997; Merica, et al., 1998; Merica & Gaillard, 1992; Nofzinger et al., 1999; Perlis et al., 2001). In addition, elevated levels of alpha activity at sleep onset (Besset et al., 1998; Krystal et al., 2002) as well as a decrease in delta activity during non-REM sleep (Merica et al., 1998; Merica & Gaillard, 1992) have been reported. Furthermore, it has been demonstrated that insomniacs produce less spontaneous waking SMR activity than controls (P. Hauri, 1981; Krystal et al., 2002). One interesting aspect about the SMR is that it lies in the same frequency range as sleep spindles (Sterman, et al., 1970). Spindles are the hallmark waveform of stage 2 sleep, and their occurrence is reduced in insomniacs (Besset et al., 1998), possibly resulting in lighter and more fragmented sleep (Glenn & Steriade, 1982; Perlis et al., 2001).

3.1 Training duration and feedback

Protocols for EEG-biofeedback in insomnia are quite similar in many respects to the ones used in the treatment of ADHD, e.g. patients usually receive feedback and reward in the form of auditory and/or visual stimuli and are encouraged to search for their own

Study	Conds.	Control P/R/B*1)	N (m)	Age	Electrodes /Ref	Freq.	Stim/ Reward	#Ses./ Dur.
Hauri,1981	[1] EBF+EMG [2] EBF [3] EMG [4] Control	-/+/-	[1]12 [2]12 [3]12 [4]12	Total: 41.3±14.6	C3 /A2	θ ↑ SMR↑	Visual	24.8 (15-62) / 60 min
Hauri et al.,1982	[1] EBF(θ) [2] EBF(SMR)	-/+/-	[1]8(5) [2]8(5)	50.1 47.4	T7&C3/A2	θ ↑ SMR↑	Visual	25.4/ 60 min 27.8/60 min
Berner et al., 2006	EBF/Sham	+/+/+	11(4)	20.8±2.8	Cz / FCz	σ ↑	Visual & Auditory	1 / 4x10 min
Hoedlmoser et al., 2008	[1] EBF [2] Sham	+/+/+	[1]16(?) [2]11(?) Total: 27(13)	Total: 23.6±2.7	C3 /A2	SMR↑	Visual & Auditory	10/ 24 min
Cortoos et al., 2009	[1] EBF [2] EMG [3] Control	-/+/-	[1] 9(6) [2] 8(5) [3]12(7)	41.5±9.5 43.8±9.5 44.4±7.8	FPz & Cz /A2	SMR↑ θ ↓ β ↓	Visual	20/ 20 min

*1) **P/R/B**= Placebo/Randomized/Blind (- = no, + = yes). **N(m)**: Number of participants (males), **Freq.**: Target frequency ↑/↓ (increase/decrease), **?** = data unavailable

Table 3. Overview of EBG group studies aimed at improving sleep.

individual strategies (Berner et al., 2006; Cortoos et al., 2009; Hauri et al., 1982; Hoedlmoser et al., 2008). Training sessions (Table 3) are usually blocked (e.g., 3 minute intervals) during which a threshold of activity expressed as a percentage of, or within a predefined band around the baseline, must be maintained for 250–500 ms (Berner et al., 2006; Cortoos et al., 2010; Hoedlmoser et al., 2008).

3.2 Target brain activity

Insomniacs differ from good sleepers in terms of their EEG profile (Fig. 3), especially exhibiting large spectral decreases in the lower frequency bands (delta, theta) (Merica et al., 1998) and attenuated sigma activity, corresponding to less occurrences of sleep spindles (Besset et al., 1998). These findings have led to the design of EBF therapies aimed at either increasing theta activity, due to its close relationship with drowsiness and early sleep stages, or SMR activity, as this rhythm overlaps with the sigma range and is believed to stimulate sleep spindle occurrence which in turn is key to further progression into deeper sleep stages

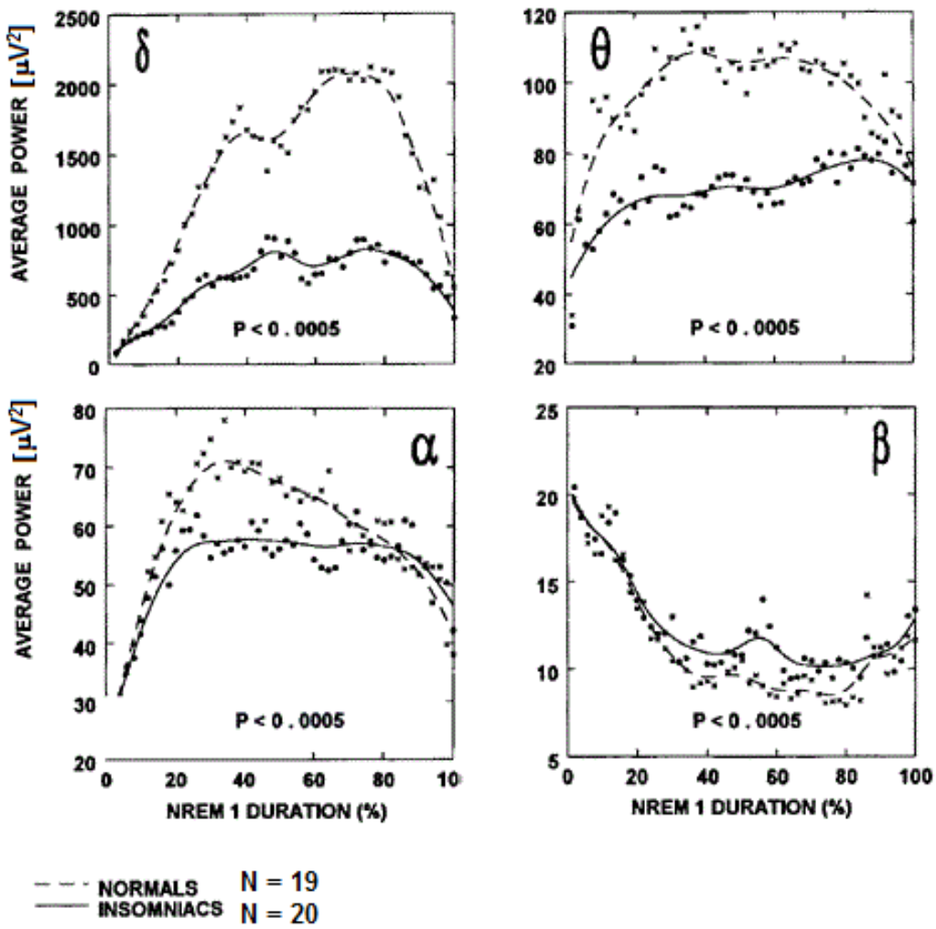

Fig. 3. Insomniacs and normal sleepers have different EEG during stage 1 sleep. Insomniacs (solid line) have reduced delta, theta and alpha activity, but higher levels of beta activity compared to normal sleepers (dashed line) during early stages of sleep. Y-axis: average power over all participants in specific frequency band. X-axis: normalized duration of sleep stage 1, each of the 50 dots marks a 2% interval. From: Merica et al., 1998.

(Berner et al., 2006; Budzynski, 1973; Hauri, 1981; Sittenfeld, 1972; Steriade, 2003). The application of either protocol depends on the insomnia sub-population: theta feedback (enhancement training) is used for patients with difficulty initiating sleep, whereas SMR/sigma feedback is best used on patients that have problems maintaining sleep. The importance of disentangling insomnia subtypes is further illustrated by the studies of Hauri et al. (1981,1982). Even though all participants showed a trend towards improvement, the experimental groups (i.e. theta feedback, SMR feedback) did not differ, which could be attributed to participants having received treatment unsuitable to the underlying symptoms (Hauri, 1981; et al., 1982).

3.3 Efficacy of EEG-biofeedback in the treatment of insomnia

A pioneering case study used theta training to treat an insomnia patient and observed a near doubling of theta activity by the end of the 11-week (one session per week) EBF training, together with vastly decreased sleep-onset latency (from 54 to 16 minutes), an increase in total sleep time and a halving in intrusive thoughts (Bell, 1979). Recent studies have compared SMR training with pseudo-EBF training and reported positive results with respect to the total sleep time and the sleep latency (Berner et al., 2006; Hoedlmoser et al., 2008). Cortoos et al. (2009) compared electromyography (EMG) biofeedback, aimed at reducing muscle tension and relaxation, with an EBF protocol of SMR increase and simultaneous theta-, and beta-band suppression. Both groups showed decreases in sleep latency (-8.5 and -12.3 minutes respectively) and time awake after sleep onset. It is noteworthy that participants were trained to apply electrodes and initiate training in their home environment and experimental control was established remotely through the internet, making this "tele-neurofeedback" protocol an interesting example of fusing established knowledge with advanced technology.

In contrast to the case of ADHD where subjective ratings largely define outcome measures (Table 2), efficacy and validity of EBF-therapy for insomniacs is easier to assess through objective measures such as total sleep time, sleep-onset latency and the number of nightly awakenings. In 1998, the American Academy of Sleep Medicine recommended biofeedback in general, including EMG-biofeedback, as treatment for insomnia and classified it as "probably efficacious", based on the Guidelines for Evaluation of Clinical Efficacy of Psychophysiological Interventions (Table 3). In the update of 1999–2004, this rating was maintained (Morgenthaler et al., 2006; Morin et al., 2006; Morin et al., 1999).

4. Conclusion

The methodology of EBF studies has often been subject to criticism (Kline et al., 2002; Loo & Barkley, 2005; Pelham & Waschbusch, 2006; Ramirez et al., 2001; Rickles et al., 1982). While some concerns are undoubtedly warranted, much effort has been put in establishing strict guidelines for EBF therapy and this has been met with positive results (Arns et al., 2009; La Vaque et al., 2002). Double-blind, randomised and placebo controlled experiments are unfortunately not always an option. Blinding requires a control condition that is indistinguishable from the treatment condition, which is often technically not feasible. Randomisation, while powerful, is only useful when the target sample is either well-known or homogenous to avoid samples being treated with inadequate protocols (Hauri, 1981; Hauri et al., 1982). Finally, a placebo condition, especially in the case of ADHD, is problematic from an ethical viewpoint, as denying patients a standard and efficacious

treatment (i.e., medication) is in conflict with the Declaration of Helsinki (Vernon et al., 2004). Employing sham (random frequency) feedback (Hoedlmoser et al., 2008; Logemann et al., 2010) is therefore not always an option when treating patients. Thus, apart from reaching certain endpoints of treatment, the further validation of EBF therapy is likely to depend on the observation of complimentary physiological changes, e.g., obtained from neuroimaging experiments or other biomarker assays (Frank & Hargreaves, 2003).

Motivation and cognitive strategies are also important aspects to consider (Bregman & McAllister, 1982; Meichenbaum, 1976). If participants are motivated and rewarded for their success they will put effort into the therapy, whereas lack thereof leads to frustration and possibly resignation (Huang et al., 2006). Good methodology can compensate for possible expectancy effects, i.e., improved symptoms like decreases in sleep onset latency induced by the sheer hope of becoming better through therapy (Hauri et al., 1982). However, providing sham feedback, which lacks obvious rewards, bears the risk of the participant becoming unmotivated, ceasing effort and thus confounding the comparison between control and experimental condition (Logemann et al., 2010). In addition, the instructions given to participants in the EBF studies reviewed here do not go beyond the direction to meet some specified criterion, i.e., increasing an onscreen bar towards a target value. The general idea is that participants need to search for their own strategies to modulate their brain activity. In our view, this is unfortunate, because good instructions/guidance can increase participant compliance and speed of learning (Weinert et al., 1989). While individual strategies are likely to vary greatly, an opportunity for future research presents itself in the collection of these strategies and finding patterns that may be useful to guide participants towards success more efficiently. Interestingly, Gevensleben et al. (2009) report on having queried individual strategies of their participants (albeit without further analysis), making future compilation of strategies feasible.

Technological advances have made it possible to record high-density EEG data from several hundred electrodes at once (Dornhege et al., 2006). However, current EBF studies seldom record from more than two active electrodes (Tables 2 and 3). With ongoing developments towards ever more powerful and cost-effective computational equipment, it is feasible that future research should focus on the opportunities these advances can offer EBF, possibly in combination with tools from the field of BCI (e.g., more sophisticated algorithms, spatial filtering allowing feedback on localized anatomical structures and less artefacts). Despite some (methodological) issues that have subjected the field to scepticism, recent developments give rise to optimism, as stricter guidelines are increasingly being adhered to and new avenues continue to be explored (e.g., SCP feedback and tele-neurofeedback as in Cortoos et al., 2009). Overall, from the studies reviewed here we conclude that EBF is a promising tool for treating disorders of arousal, which offers many opportunities for future research.

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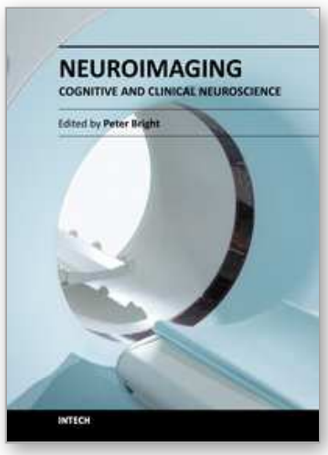

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The rate of technological progress is encouraging increasingly sophisticated lines of enquiry in cognitive neuroscience and shows no sign of slowing down in the foreseeable future. Nevertheless, it is unlikely that even the strongest advocates of the cognitive neuroscience approach would maintain that advances in cognitive theory have kept in step with methods-based developments. There are several candidate reasons for the failure of neuroimaging studies to convincingly resolve many of the most important theoretical debates in the literature. For example, a significant proportion of published functional magnetic resonance imaging (fMRI) studies are not well grounded in cognitive theory, and this represents a step away from the traditional approach in experimental psychology of methodically and systematically building on (or chipping away at) existing theoretical models using tried and tested methods. Unless the experimental study design is set up within a clearly defined theoretical framework, any inferences that are drawn are unlikely to be accepted as anything other than speculative. A second, more fundamental issue is whether neuroimaging data alone can address how cognitive functions operate (far more interesting to the cognitive scientist than establishing the neuroanatomical coordinates of a given function - the where question).

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

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