

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800

Open access books available

122,000

International authors and editors

135M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.

For more information visit www.intechopen.com



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 (Nicoletis, 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 (Cortcos 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).

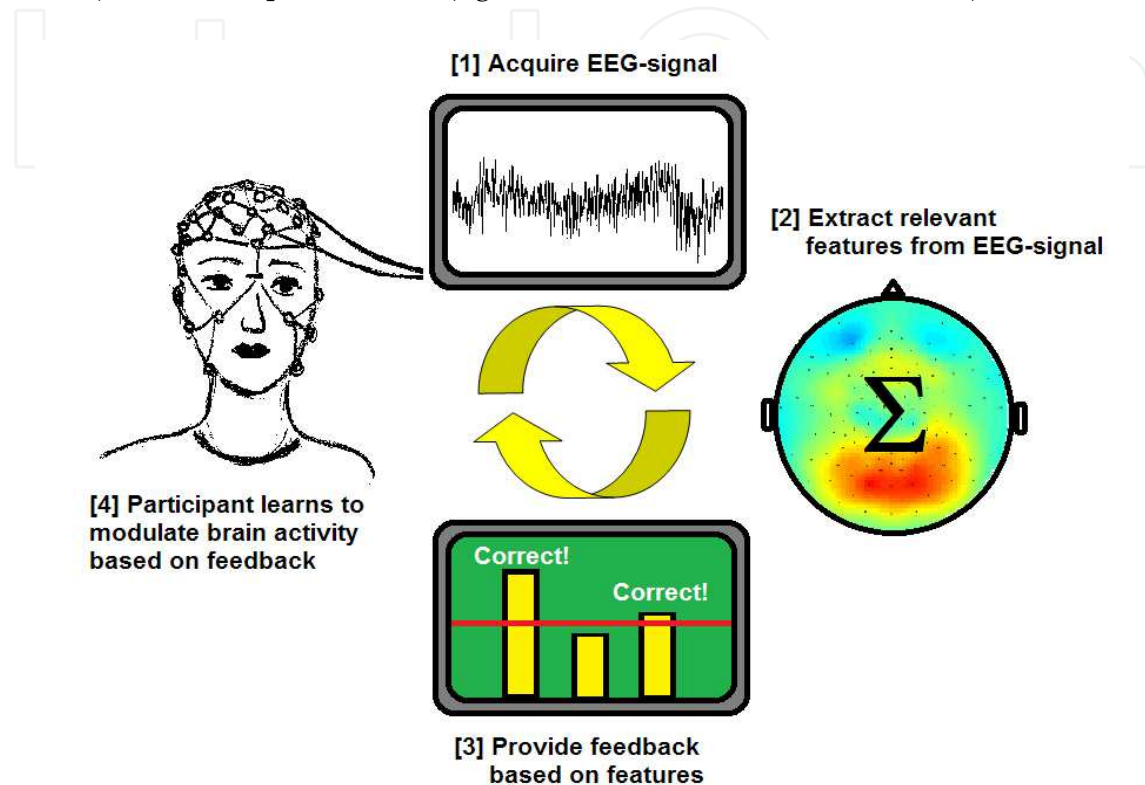


Fig. 1. The concept of EEG-biofeedback. The EEG is recorded [1], a suitable EEG-biomarker is extracted [2] and made available to the participant and correct changes in brain activity are rewarded by, e.g., a visual stimulus indicating success [3]. With repetition, this enables the participant to learn 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 (Serman & Friar, 1972; Serman 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 were 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).

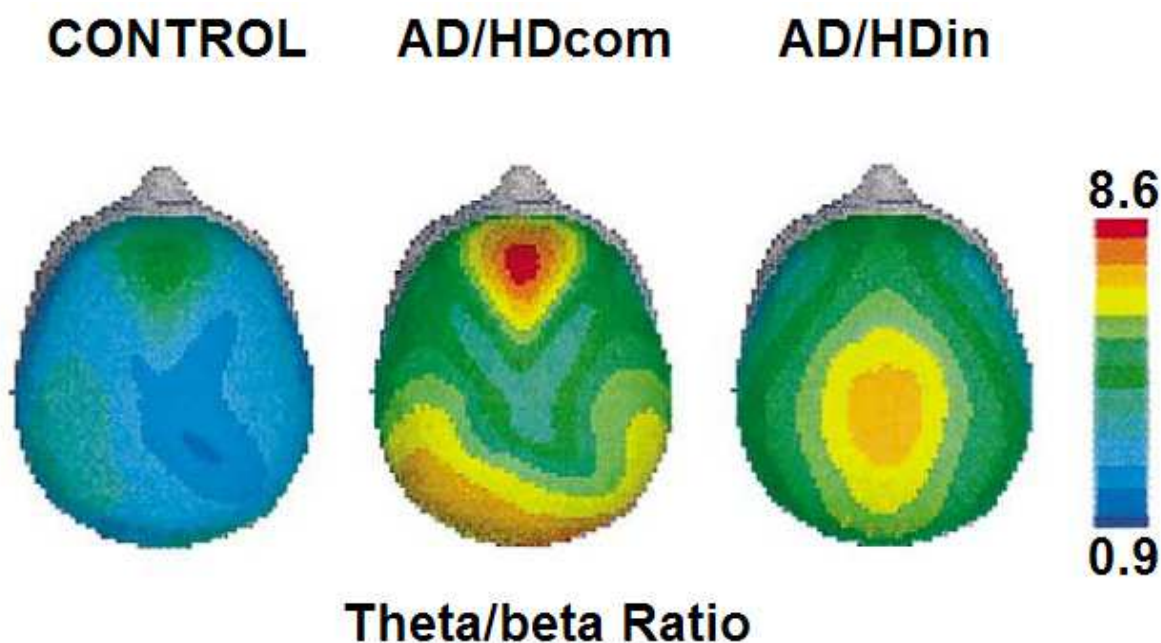


Fig. 2. Brain activity profiles in children with ADHD differ from healthy controls. Theta/beta-band activity ratio is strongly elevated in ADHD, but differs in spatial localization between combined (AD/HDcom) and inattentive (AD/HDin) subtypes. (From: Barry et al., 2003.).

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] [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 (Cortoo 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 (Serman, 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
Cortoo 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

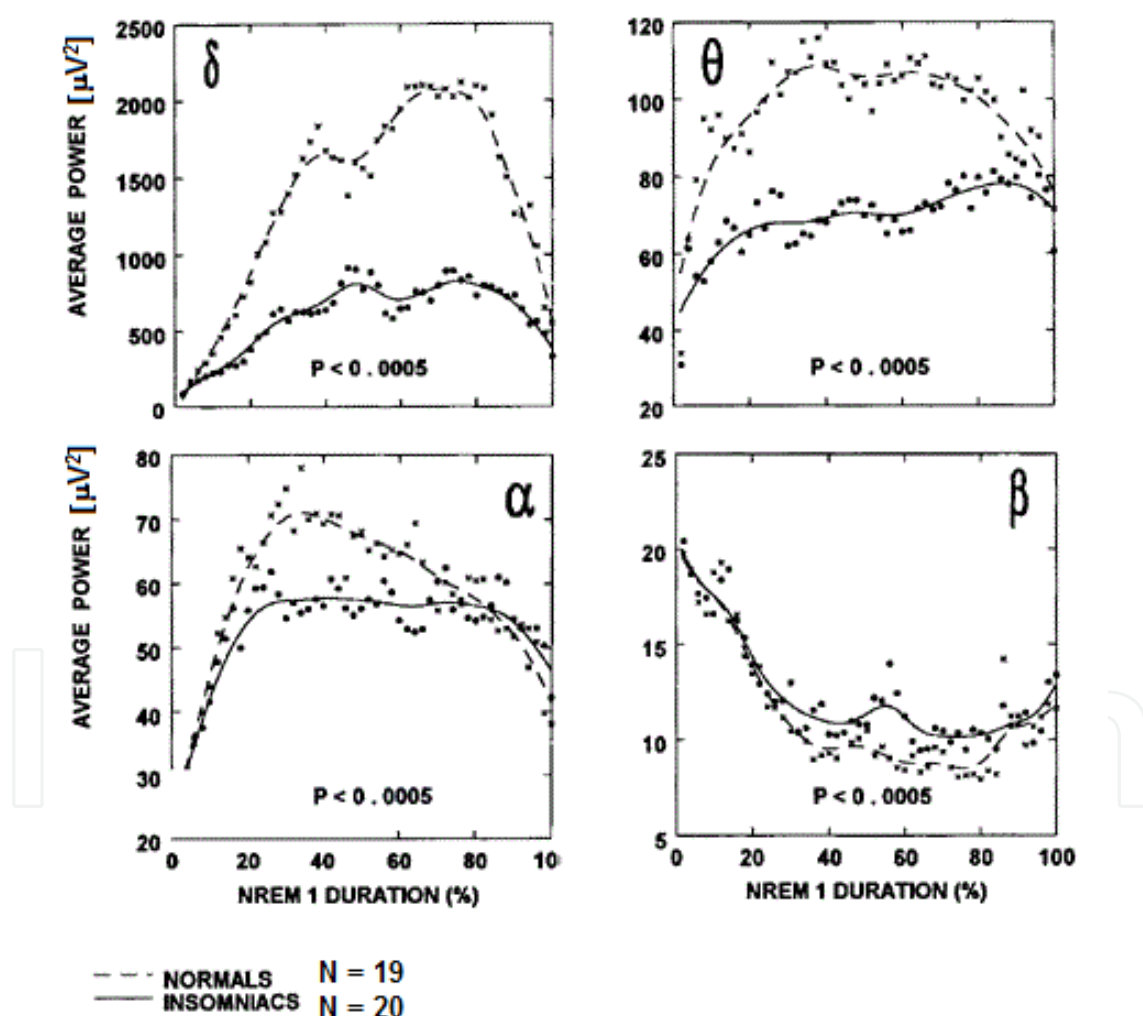


Fig. 3. Insomniacs and normal sleepers have different EEG during stage 1 sleep. Insomniacs (solid line) have reduced delta, theta and alpha activity, but higher levels of beta activity compared to normal sleepers (dashed line) during early stages of sleep. Y-axis: average power over all participants 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.

5. References

- Aldrich, M. S. (1992). Sleep disorders. *Curr Opin Neurol Neurosurg*, 5(2), 240-246.
- Alhambra, M. A., Fowler, T. P., & Alhambra, A. A. (1995). EEG biofeedback: A new treatment option for ADD/ADHD. *Journal of Neurotherapy*, 1(2), 39-43.
- Alkire, M. T., Hudetz, A. G., & Tononi, G. (2008). Consciousness and anesthesia. *Science*, 322(5903), 876-880.
- Angelakis, E., Stathopoulou, S., Frymiare, J. L., Green, D. L., Lubar, J. F., & Kounios, J. (2007). EEG neurofeedback: a brief overview and an example of peak alpha

- frequency training for cognitive enhancement in the elderly. *The Clinical Neuropsychologist*, 21(1), 110-129.
- Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: the effects on inattention, impulsivity and hyperactivity: a meta-analysis. *Clin EEG Neurosci*, 40(3), 180-189.
- Baehr, E., Rosenfeld, J. P., & Baehr, R. (1997). The clinical use of an alpha asymmetry protocol in the neurofeedback treatment of depression: two case studies. *Journal of Neurotherapy*, 2, 10-23.
- Ball, J., Tiernan, M., Janusz, J., & Furr, A. (1997). Sleep patterns among children with attention-deficit hyperactivity disorder: a reexamination of parent perceptions. *Journal of pediatric psychology*, 22(3), 389.
- Barkley, R. A. (1998). *Attention-deficit hyperactivity disorder: a handbook of diagnosis and treatment* (2nd ed.). New York: Guilford.
- Barry, R. J., & Clarke, A. R. (2009). Spontaneous EEG oscillations in children, adolescents, and adults: Typical development, and pathological aspects in relation to AD/HD. *Journal of Psychophysiology*, 23(4), 157.
- Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. *Clin Neurophysiol*, 114(2), 171-183.
- Barry, R. J., Clarke, A. R., Johnstone, S. J., McCarthy, R., & Selikowitz, M. (2009). Electroencephalogram [theta]/[beta] Ratio and Arousal in Attention-Deficit/Hyperactivity Disorder: Evidence of Independent Processes. *Biological Psychiatry*, 66(4), 398-401.
- Barry, R. J., Johnstone, S. J., & Clarke, A. R. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: II. Event-related potentials. *Clinical Neurophysiology*, 114(2), 184-198.
- Bastien, C. H., LeBlanc, M., Carrier, J., & Morin, C. M. (2003). Sleep EEG power spectra, insomnia, and chronic use of benzodiazepines. *Sleep*, 26(3), 313-317.
- Bastien, C. H., Vallieres, A., & Morin, C. M. (2004). Precipitating factors of insomnia. *Behav Sleep Med*, 2(1), 50-62.
- Beauregard, M., & Levesque, J. (2006). Functional magnetic resonance imaging investigation of the effects of neurofeedback training on the neural bases of selective attention and response inhibition in children with attention-deficit/hyperactivity disorder. *Appl Psychophysiol Biofeedback*, 31(1), 3-20.
- Bell, J. S. (1979). The use of EEG theta biofeedback in the treatment of a patient with sleep-onset insomnia. *Biofeedback Self Regul*, 4(3), 229-236.
- Berner, I., Schabus, M., Wienerroither, T., & Klimesch, W. (2006). The significance of sigma neurofeedback training on sleep spindles and aspects of declarative memory. *Appl Psychophysiol Biofeedback*, 31(2), 97-114.
- Besset, A., Villemin, E., Tafti, M., & Billiard, M. (1998). Homeostatic process and sleep spindles in patients with sleep-maintenance insomnia: effect of partial (21 h) sleep deprivation. *Electroencephalogr Clin Neurophysiol*, 107(2), 122-132.
- Birbaumer, N., Ghanayim, N., Hinterberger, T., Iversen, I., Kotchoubey, B., Kübler, A., et al. (1999). A spelling device for the paralysed. *Nature*, 398(6725), 297-298.
- Bonnet, M. H. (2010). Hyperarousal and insomnia. *Sleep medicine reviews*, 14(1).
- Bonnet, M. H., & Arand, D. (1997). Physiological activation in patients with sleep state misperception. *Psychosomatic medicine*, 59(5), 533.

- Bonnet, M. H., & Arand, D. L. (2005). Impact of motivation on Multiple Sleep Latency Test and Maintenance of Wakefulness Test measurements. *Journal of Clinical Sleep Medicine: JCSM: Official Publication of the American Academy of Sleep Medicine*, 1(4), 386.
- Bregman, N. J., & McAllister, H. A. (1982). Motivation and Skin Temperature Biofeedback: Yerkes Dodson Revisited. *Psychophysiology*, 19(3), 282-285.
- Brown, R. T., Freeman, W. S., Perrin, J. M., Stein, M. T., Amler, R. W., Feldman, H. M., et al. (2001). Prevalence and assessment of attention-deficit/hyperactivity disorder in primary care settings. *Pediatrics*, 107(3), E43.
- Budzynski, T. H. (1973). Biofeedback procedures in the clinic. *Semin Psychiatry*, 5(4), 537-547.
- Buysse, D., Hall, M., Strollo, P., Kamarck, T., Owens, J., Lee, L., et al. (2008). Relationships between the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and clinical/polysomnographic measures in a community sample. *Journal of Clinical Sleep Medicine: JCSM: Official Publication of the American Academy of Sleep Medicine*, 4(6), 563.
- Buysse, D. J., Germain, A., Hall, M. L., Moul, D. E., Nofzinger, E. A., Begley, A., et al. (2008). EEG Spectral Analysis in Primary Insomnia: NREM Period Effects and Sex Differences. *Sleep*, 31(12), 1673.
- Carmody, D. P., Radvanski, D. C., Wadhvani, S., Sabo, M. J., & Vergara, L. (2000). EEG biofeedback training and attention-deficit/hyperactivity disorder in an elementary school setting. *Journal of Neurotherapy*, 4(3), 5-27.
- Charach, A., Ickowicz, A., & Schachar, R. (2004). Stimulant treatment over five years: adherence, effectiveness, and adverse effects. *Journal of the American Academy of Child & Adolescent Psychiatry*, 43(5), 559-567.
- Clarke, A., Barry, R., McCarthy, R., Selikowitz, M., & Brown, C. (2002). EEG evidence for a new conceptualisation of attention deficit hyperactivity disorder. *Clinical Neurophysiology*, 113(7), 1036-1044.
- Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001). Age and sex effects in the EEG: differences in two subtypes of attention-deficit/hyperactivity disorder. *Clinical Neurophysiology*, 112(5), 815-826.
- Coben, R., & Padolsky, I. (2007). Assessment-guided neurofeedback for Autistic Spectrum Disorder. *Journal of Neurotherapy*, 11(1), 5-23.
- Cortoo, A., De Valck, E., Arns, M., Breteler, M. H., & Cluydts, R. (2009). An Exploratory Study on the Effects of Tele-neurofeedback and Tele-biofeedback on Objective and Subjective Sleep in Patients with Primary Insomnia. *Appl Psychophysiol Biofeedback*.
- Cortoo, A., De Valck, E., Arns, M., Breteler, M. H., & Cluydts, R. (2010). An exploratory study on the effects of tele-neurofeedback and tele-biofeedback on objective and subjective sleep in patients with primary insomnia. *Appl Psychophysiol Biofeedback*, 35(2), 125-134.
- Cortoo, A., Verstraeten, E., & Cluydts, R. (2006). Neurophysiological aspects of primary insomnia: implications for its treatment. *Sleep Med Rev*, 10(4), 255-266.
- Daley, M., Morin, C. M., LeBlanc, M., Grégoire, J. P., & Savard, J. (2009). The economic burden of insomnia: direct and indirect costs for individuals with insomnia syndrome, insomnia symptoms, and good sleepers. *Sleep*, 32(1), 55.
- Doehnert, M., Brandeis, D., Straub, M., Steinhausen, H. C., & Drechsler, R. (2008). Slow cortical potential neurofeedback in attention deficit hyperactivity disorder: is there neurophysiological evidence for specific effects? *J Neural Transm*, 115(10), 1445-1456.

- Dornhege, G., Blankertz, B., Krauledat, M., Losch, F., Curio, G., & Müller, K. R. (2006). Combined optimization of spatial and temporal filters for improving brain-computer interfacing. *Biomedical Engineering, IEEE Transactions on*, 53(11), 2274-2281.
- Drake, C., Richardson, G., Roehrs, T., Scofield, H., & Roth, T. (2004). Vulnerability to stress-related sleep disturbance and hyperarousal. *Sleep*, 27(2), 285-291.
- Drechsler, R., Straub, M., Doehnert, M., Heinrich, H., Steinhausen, H. C., & Brandeis, D. (2007). Controlled evaluation of a neurofeedback training of slow cortical potentials in children with Attention Deficit/Hyperactivity Disorder (ADHD). *Behav Brain Funct*, 3, 35.
- Drummond, S., Brown, G. G., Salamat, J. S., & Gillin, J. C. (2004). Increasing task difficulty facilitates the cerebral compensatory response to total sleep deprivation. *Sleep*, 27(3), 445-451.
- Ebben, M. R., & Spielman, A. J. (2009). Non-pharmacological treatments for insomnia. *J Behav Med*, 32(3), 244-254.
- Edinger, J. D., Bonnet, M. H., Bootzin, R. R., Doghramji, K., Dorsey, C. M., Espie, C. A., et al. (2004). Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep*, 27(8), 1567-1596.
- Efron, D., Jarman, F., & Barker, M. (1997). Side effects of methylphenidate and dexamphetamine in children with attention deficit hyperactivity disorder: a double-blind, crossover trial. *Pediatrics*, 100(4), 662.
- Egner, T., & Gruzelier, J. H. (2003). Ecological validity of neurofeedback: modulation of slow wave EEG enhances musical performance. *Neuroreport*, 14(9), 1221-1224.
- Egner, T., Strawson, E., & Gruzelier, J. H. (2002). EEG signature and phenomenology of alpha/theta neurofeedback training versus mock feedback. *Applied Psychophysiology and Biofeedback*, 27(4), 261-270.
- Espie, C. A. (1999). Cognitive behaviour therapy as the treatment of choice for primary insomnia. *Sleep Med Rev*, 3(2), 97-99.
- Espie, C. A. (2007). Understanding insomnia through cognitive modelling. *Sleep Med*, 8 Suppl 4, S3-8.
- Faraone, S. V., & Buitelaar, J. (2010). Comparing the efficacy of stimulants for ADHD in children and adolescents using meta-analysis. *Eur Child Adolesc Psychiatry*, 19(4), 353-364.
- Faraone, S. V., & Glatt, S. J. (2010). A comparison of the efficacy of medications for adult attention-deficit/hyperactivity disorder using meta-analysis of effect sizes. *The Journal of clinical psychiatry*, 71(6), 754.
- Felton, E. A., Wilson, J. A., Williams, J. C., & Garell, P. C. (2007). Electrocorticographically controlled brain-computer interfaces using motor and sensory imagery in patients with temporary subdural electrode implants. *Journal of neurosurgery*, 106(3), 495-500.
- Frank, R., & Hargreaves, R. (2003). Clinical biomarkers in drug discovery and development. *Nature Reviews Drug Discovery*, 2(7), 566-580.
- Freedman, R. R. (1986). EEG power spectra in sleep-onset insomnia. *Electroencephalogr Clin Neurophysiol*, 63(5), 408-413.
- Fuchs, T., Birbaumer, N., Lutzenberger, W., Gruzelier, J. H., & Kaiser, J. (2003). Neurofeedback treatment for attention-deficit/hyperactivity disorder in children: a comparison with methylphenidate. *Appl Psychophysiol Biofeedback*, 28(1), 1-12.
- Gani, C., Birbaumer, N., & Strehl, U. (2009). Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with attentiondeficit/hyperactivity disorder (ADHD). *International Journal of Bioelectromagnetics*, 10, 209-232.

- Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., et al. (2010). Neurofeedback training in children with ADHD: 6-month follow-up of a randomised controlled trial. *Eur Child Adolesc Psychiatry*, 19(9), 715-724.
- Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., et al. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. *Journal of Child Psychology and Psychiatry*, 50(7), 780-789.
- Glenn, L. L., & Steriade, M. (1982). Discharge rate and excitability of cortically projecting intralaminar thalamic neurons during waking and sleep states. *J Neurosci*, 2(10), 1387-1404.
- Goldstein, S., & Goldstein, M. (1990). *Managing attention disorders in children: A guide for practitioners*. New York: Wiley.
- Gruzelier, J., Egner, T., & Vernon, D. (2006). Validating the efficacy of neurofeedback for optimising performance. *Progress in Brain Research*, 159, 421-431.
- Hammond, D. C. (2005). Neurofeedback treatment of depression and anxiety. *Journal of Adult Development*, 12, 131-137.
- Harvey, A., & Payne, S. (2002). The management of unwanted pre-sleep thoughts in insomnia: distraction with imagery versus general distraction. *Behaviour Research and Therapy*, 40(3), 267-277.
- Hauri, P. (1981). Treating psychophysiologic insomnia with biofeedback. *Arch Gen Psychiatry*, 38(7), 752-758.
- Hauri, P. J., Percy, L., Hellekson, C., Hartmann, E., & Russ, D. (1982). The treatment of psychophysiologic insomnia with biofeedback: a replication study. *Biofeedback Self Regul*, 7(2), 223-235.
- Heinrich, H., Gevensleben, H., Freisleder, F. J., Moll, G. H., & Rothenberger, A. (2004). Training of slow cortical potentials in attention-deficit/hyperactivity disorder: evidence for positive behavioral and neurophysiological effects. *Biol Psychiatry*, 55(7), 772-775.
- Heinrich, H., Gevensleben, H., & Strehl, U. (2007). Annotation: neurofeedback - train your brain to train behaviour. *J Child Psychol Psychiatry*, 48(1), 3-16.
- Hermens, D. F., Rowe, D. L., Gordon, E., & Williams, L. M. (2006). Integrative neuroscience approach to predict ADHD stimulant response. *Expert Rev Neurother*, 6(5), 753-763.
- Heywood, C., & Beale, I. (2003). EEG biofeedback vs. placebo treatment for attention-deficit/hyperactivity disorder: a pilot study. *J Atten Disord*, 7(1), 43-55.
- Hoedlmoser, K., Pecherstorfer, T., Gruber, G., Anderer, P., Doppelmayr, M., Klimesch, W., et al. (2008). Instrumental conditioning of human sensorimotor rhythm (12-15 Hz) and its impact on sleep as well as declarative learning. *Sleep*, 31(10), 1401-1408.
- Holtmann, M., Grasmann, D., Cionek-Szpak, E., Hager, V., Panzer, N., & Beyer, A. (2009). Spezifische wirksamkeit von Neurofeedback auf die Impulsivitat bei ADHS - Literaturuberblick und ergebnisse einer prospective, kontrollierten Studie. *Kindheit und Entwicklung*, 18, 95-104.
- Huang, H., Wolf, S. L., & He, J. (2006). Recent developments in biofeedback for neuromotor rehabilitation. *Journal of NeuroEngineering and Rehabilitation*, 3(1), 11.
- Jacobs, G. D., Benson, H., & Friedman, R. (1993). Home-based central nervous system assessment of a multifactor behavioral intervention for chronic sleep-onset insomnia. *Behavior Ther*, 24, 159-174.
- Jansson, M., & Linton, S. J. (2007). Psychological mechanisms in the maintenance of insomnia: arousal, distress, and sleep-related beliefs. *Behav Res Ther*, 45(3), 511-521.

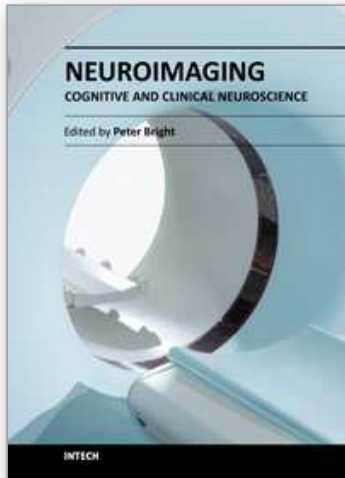
- Kaiser, D. A. (1997). Efficacy of Neurofeedback on adults with Attention Defecit and Related disorders: EEG Spectrum Inc.
- Kaiser, D. A., & Othmer, S. (2000). Effects of neurofeedback on variables of attention in a large multi-center trial. *Journal of Neurotherapy*, 4(1), 5-15.
- Kline, J. P., Brann, C. N., & Loney, B. R. (2002). A cacophony in the brainwaves: a critical appraisal of neurotherapy for attention-deficit disorders. *The Scientific Review of Mental Health Practice*, 1, 44-54.
- Kropotov, J. D., Grin-Yatsenko, V. A., Ponomarev, V. A., Chutko, L. S., Yakovenko, E. A., & Nikishena, I. S. (2005). ERPs correlates of EEG relative beta training in ADHD children. *Int J Psychophysiol*, 55(1), 23-34.
- Krusienski, D. J., Sellers, E. W., Cabestaing, F., Bayouth, S., McFarland, D. J., Vaughan, T. M., et al. (2006). A comparison of classification techniques for the P300 speller. *Journal of Neural Engineering*, 3, 299.
- Krusienski, D. J., Sellers, E. W., McFarland, D. J., Vaughan, T. M., & Wolpaw, J. R. (2008). Toward enhanced P300 speller performance. *Journal of neuroscience methods*, 167(1), 15-21.
- Krystal, A. D., Edinger, J. D., Wohlgemuth, W. K., & Marsh, G. R. (2002). NREM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. *Sleep*, 25(6), 630.
- Kübler, A., Kotchoubey, B., Kaiser, J., Wolpaw, J. R., & Birbaumer, N. (2001). Brain-computer communication: Unlocking the locked in. *Psychological Bulletin*, 127(3), 358.
- Kumari, V. (2006). Do psychotherapies produce neurobiological effects? *Acta neuropsychiatrica*, 18(2), 61-70.
- La Vaque, T. J., Hammond, D. C., Trudeau, D., Monastra, V. J., J., P., & Lehrer, P. (2002). Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological interventions. *Applied Psychophysiology and Biofeedback*, 27, 273-281.
- Lamarche, C. H., & Ogilvie, R. D. (1997). Electrophysiological changes during the sleep onset period of psychophysiological insomniacs, psychiatric insomniacs, and normal sleepers. *Sleep(New York, NY)*, 20(9), 724-733.
- Leins, U., Goth, G., Hinterberger, T., Klinger, C., Rumpf, N., & Strehl, U. (2007). Neurofeedback for children with ADHD: a comparison of SCP and Theta/Beta protocols. *Appl Psychophysiol Biofeedback*, 32(2), 73-88.
- Leuthardt, E. C., Miller, K. J., Schalk, G., Rao, R. P. N., & Ojemann, J. G. (2006). Electrocoricography-based brain computer interface-the Seattle experience. *Neural Systems and Rehabilitation Engineering, IEEE Transactions on*, 14(2), 194-198.
- Levesque, J., Beauregard, M., & Mensour, B. (2006). Effect of neurofeedback training on the neural substrates of selective attention in children with attention-deficit/hyperactivity disorder: a functional magnetic resonance imaging study. *Neurosci Lett*, 394(3), 216-221.
- Li, D., Sham, P. C., Owen, M. J., & He, L. (2006). Meta-analysis shows significant association between dopamine system genes and attention deficit hyperactivity disorder (ADHD). *Human molecular genetics*, 15(14), 2276.
- Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Applied Psychophysiology and Biofeedback*, 21(1), 35-49.
- Logemann, H. N. A., Lansbergen, M. M., Van Os, T. W. D. P., Bocker, K. B. E., & Kenemans, J. L. (2010). The effectiveness of EEG-feedback on attention, impulsivity and EEG: A sham feedback controlled study. *Neuroscience Letters*, 479, 49-53.

- Loo, S. K., & Barkley, R. A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Appl Neuropsychol*, 12(2), 64-76.
- Lubar, J., Swartwood, M., Swartwood, J., & O'Donnell, P. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in TOVA scores, behavioral ratings, and WISC-R performance. *Applied Psychophysiology and Biofeedback*, 20(1), 83-99.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Applied Psychophysiology and Biofeedback*, 16(3), 201-225.
- Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR). *Applied Psychophysiology and Biofeedback*, 1(3), 293-306.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. *Biofeedback Self Regul*, 20(1), 83-99.
- Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Applied Psychophysiology and Biofeedback*, 9(1), 1-23.
- Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, C. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: controlled study with clinical implications. *Pediatr Neurol*, 8(1), 30-36.
- Marco, R., Miranda, A., Schlotz, W., Melia, A., Mulligan, A., Müller, U., et al. (2009). Delay and reward choice in ADHD: An experimental test of the role of delay aversion. *Neuropsychology*, 23(3), 367.
- Meichenbaum, D. (1976). Cognitive factors in biofeedback therapy. *Applied Psychophysiology and Biofeedback*, 1(2), 201-216.
- Merica, H., Blois, R., & Gaillard, J. M. (1998). Spectral characteristics of sleep EEG in chronic insomnia. *Eur J Neurosci*, 10(5), 1826-1834.
- Merica, H., & Gaillard, J. (1992). The EEG of the sleep onset period in insomnia: a discriminant analysis. *Physiology & behavior*, 52(2), 199-204.
- Molina, B. S., Hinshaw, S. P., Swanson, J. M., Arnold, L. E., Vitiello, B., & Jensen, P. S. (2009). The MTA at 8 years: prospective follow-up of children treated for combined-type ADHD in a multisite study. *Journal of American Academy of Child and Adolescent Psychiatry*, 48, 484-500.
- Monastra, V. J. (2005). Electroencephalographic biofeedback (neurotherapy) as a treatment for attention deficit hyperactivity disorder: rationale and empirical foundation. *Child Adolesc Psychiatr Clin N Am*, 14(1), 55-82, vi.
- Monastra, V. J., Lynn, S., Linden, M., Lubar, J. F., Gruzelier, J., & LaVaque, T. J. (2005). Electroencephalographic biofeedback in the treatment of attention-deficit/hyperactivity disorder. *Appl Psychophysiol Biofeedback*, 30(2), 95-114.
- Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, 27(4), 231-249.
- Morgenthaler, T., Kramer, M., Alessi, C., Friedman, L., Boehlecke, B., Brown, T., et al. (2006). Practice parameters for the psychological and behavioral treatment of insomnia: an update. An American Academy of Sleep Medicine report. *Sleep*, 29(11), 1415-1419.

- Morin, C., Hauri, P., Espie, C., Spielman, A., Buysse, D., & Bootzin, R. (1999). Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. *Sleep*, 22(8), 1134-1156.
- Morin, C. M. (2006). Combined therapeutics for insomnia: should our first approach be behavioral or pharmacological? *Sleep Med*, 7 Suppl 1, S15-19.
- Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004). *Sleep*, 29(11), 1398-1414.
- Morin, C. M., Culbert, J. P., & Schwartz, S. M. (1994). Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *Am J Psychiatry*, 151(8), 1172-1180.
- Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A. J., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia. *Sleep(New York, NY)*, 22(8), 1134-1156.
- Murtagh, D. R., & Greenwood, K. M. (1995). Identifying effective psychological treatments for insomnia: a meta-analysis. *J Consult Clin Psychol*, 63(1), 79-89.
- Nicolelis, M. A. L. (2003). Brain-machine interfaces to restore motor function and probe neural circuits. *Nature Reviews Neuroscience*, 4(5), 417-422.
- Nofzinger, E. A. (2004). What can neuroimaging findings tell us about sleep disorders? *Sleep Medicine*, 5, S16-S22.
- Nofzinger, E. A., Nowell, P. D., Buysse, D. J., Vasco, R. C., Thase, M. E., E., F., et al. (1999). Towards a neurobiology of sleep disturbance in primary insomnia and depression: a comparison of subjective, visually scored, period amplitude, and power spectral density sleep measures. *Sleep*, 22(1), S99.
- Ohayon, M. M. (2002). Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev*, 6(2), 97-111.
- Passini, F. T., Watson, C. G., Dehnel, L., Herder, J., & Watkins, B. (1977). Alpha wave biofeedback training therapy in alcoholics. *Journal of Clinical Psychology*, 33(1), 292-299.
- Pelham, W. E., & Waschbusch, D. A. (2006). Attention-deficit hyperactivity disorder (ADHD). *Practitioner's Guide to Evidence-Based Psychotherapy*, 93-100.
- Peniston, E. G., & Kulkosky, P. J. (1989). Alpha-theta brainwave training and beta-Endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Research*, 13(2), 271-279.
- Peniston, E. G., & Kulkosky, P. J. (1991). Alpha-theta brainwave neurofeedback for Vietnam veterans with combat-related post-traumatic stress disorder. *Medical Psychotherapy*, 4, 47-60.
- Perlis, M. L. (2001). Response to "Do increases in beta EEG activity uniquely reflect insomnia?" (C. H. Bastein and M. H. Bonnet). *Sleep Med Rev*, 5(5), 379-383.
- Perlis, M. L., Merica, H., Smith, M. T., & Giles, D. E. (2001). Beta EEG activity and insomnia. *Sleep Med Rev*, 5(5), 363-374.
- Perlis, M. L., Smith, M. T., Andrews, P. J., Orff, H., & Giles, D. E. (2001). Beta/Gamma EEG activity in patients with primary and secondary insomnia and good sleeper controls. *Sleep(New York, NY)*, 24(1), 110-117.
- Pineda, J. A., Brang, D., Hecht, E., Edwards, L., Carey, S., Bacon, M., et al. (2008). Positive behavioral and electrophysiological changes following neurofeedback training in children with autism. *Research in Autism Spectrum Disorders*, 2, 557-581.

- Raine, A., Venables, P. H., & Williams, M. (1990). Relationships between central and autonomic measures of arousal at age 15 years and criminality at age 24 years. *Archives of General Psychiatry*, 47(11), 1003.
- Ramirez, P. M., Desantis, D., & Opler, L. A. (2001). EEG Biofeedback Treatment of ADD. *Annals of the New York Academy of Sciences*, 931(1), 342-358.
- Rechtschaffen, A., & Kales, A. (1968). A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects.
- Rickles, W. H., Onoda, L., & Doyle, C. C. (1982). Task force study section report. *Applied Psychophysiology and Biofeedback*, 7(1), 1-33.
- Rossiter, T. (2004). The effectiveness of neurofeedback and stimulant drugs in treating AD/HD: part II. Replication. *Appl Psychophysiol Biofeedback*, 29(4), 233-243.
- Rossiter, T. R. (1998). Patient-directed neurofeedback for AD/HD. *Journal of Neurotherapy*, 2(4), 54-64.
- Rossiter, T. R., & La Vaque, T. J. (1995). A comparison of EEG biofeedback and psychostimulants in treating attention deficit/hyperactivity disorders. *Journal of Neurotherapy*, 1(1), 48-59.
- Roth, S. R., Sterman, M. B., & Clemente, C. C. (1967). Comparison of EEG correlates of reinforcement, internal inhibition, and sleep. *Electroencephalography and Clinical Neurophysiology*, 23, 509-520.
- Satterfield, J. H., Cantwell, D. P., & Satterfield, B. T. (1974). Pathophysiology of the hyperactive child syndrome. *Archives of General Psychiatry*, 31(6), 839.
- Saxby, E., & Peniston, E. G. (1995). Alpha-theta brainwave neurofeedback training: an effective treatment for male and female alcoholics with depressive symptoms. *Journal of Clinical Psychology*, 51(5), 685-693.
- Schalk, G., Kubanek, J., Miller, K., Anderson, N., Leuthardt, E., Ojemann, J., et al. (2007). Decoding two-dimensional movement trajectories using electrocorticographic signals in humans. *Journal of Neural Engineering*, 4, 264.
- Siebern, A. T., & Manber, R. (2010). Insomnia and its effective non-pharmacologic treatment. *Med Clin North Am*, 94(3), 581-591.
- Siniatchkin, M., Kropp, P., & Gerber, W. D. (2000). Neurofeedback--the significance of reinforcement and the search for an appropriate strategy for the success of self-regulation. *Appl Psychophysiol Biofeedback*, 25(3), 167-175.
- Sittenfeld, P. (1972). *The control of the EEG theta rhythm*. Chicago: Aldine.
- Stein, M. A. (1999). Unravelling sleep problems in treated and untreated children with ADHD. *Journal of Child and Adolescent Psychopharmacology*, 9(3), 157-168.
- Steriade, M. (2003). The corticothalamic system in sleep. *Front Biosci*, 8, d878-899.
- Steriade, M., Contreras, D., Curro Dossi, R., & Nunez, A. (1993). The slow (< 1 Hz) oscillation in reticular thalamic and thalamocortical neurons: scenario of sleep rhythm generation in interacting thalamic and neocortical networks. *Journal of Neuroscience*, 13(8), 3284.
- Sterman, M., & Friar, L. (1972). Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroencephalography and Clinical Neurophysiology*, 33(1), 89-95.
- Sterman, M. B. (1977). *Effects of sensorimotor EEG feedback training on sleep and clinical manifestations of epilepsy*. New York: Plenum.
- Sterman, M. B., Howe, R. D., & MacDonald, L. R. (1970). Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. *Science*, 167, 1146-1148.

- Sterman, M. B., LoPresti, R. W., & Fairchild, M. D. (1969). Electroencephalographic and behavioral studies of monomethylhydrazine toxicity in the cat. *Technical Report AMRL-TR-69-3, Wright-Patterson Air Force Base, Ohio, Air Systems Command.*
- Sterman, M. B., & Wyrwicka, W. (1967). EEG correlates of sleep: Evidence for separate forebrain substrates. *Brain Research, 49*, 558-576.
- Strehl, U., Leins, U., Goth, G., Klinger, C., Hinterberger, T., & Birbaumer, N. (2006). Self-regulation of slow cortical potentials: a new treatment for children with attention-deficit/hyperactivity disorder. *Pediatrics, 118*(5), e1530-1540.
- Swanson, J., McBurnett, T., Wigal, T., Pfiffner, L., Lerner, M., & Williams, L. (1993). Effect of stimulant medication on children with attention deficit disorder: A "review of reviews". *Exceptional Children, 60*, 154-162.
- Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: effectiveness in students with ADD. *Appl Psychophysiol Biofeedback, 23*(4), 243-263.
- Tripp, G., & Alsop, B. (2001). Sensitivity to reward delay in children with attention deficit hyperactivity disorder (ADHD). *Journal of Child Psychology and Psychiatry, 42*(5), 691-698.
- Van Someren, E. J. (2006). Mechanisms and functions of coupling between sleep and temperature rhythms. *Prog Brain Res, 153*, 309-324.
- Vernon, D. (2005). Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. *Applied Psychophysiology and Biofeedback, 30*(4), 347-364.
- Vernon, D., Egner, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., et al. (2003). The effect of training distinct neurofeedback protocols on aspects of cognitive performance. *International Journal of Psychophysiology, 47*(1), 75-85.
- Vernon, D., Frick, A., & Gruzelier, J. (2004). Neurofeedback as a treatment for ADHD: A methodological review with implications for future research. *Journal of Neurotherapy, 8*(2), 53-82.
- Walsh, J. K., & Schweitzer, P. K. (1999). Ten-year trends in the pharmacological treatment of insomnia. *Sleep, 22*(3), 371-375.
- Weinert, F. E., Schrader, F. W., & Helmke, A. (1989). Quality of instruction and achievement outcomes. *International Journal of Educational Research, 13*(8), 895-914.
- Wyrwicka, W., & Sterman, M. B. (1968). Instrumental conditioning of sensorimotor cortex EEG spindles in the waking cat. *Physiology and Behavior, 3*, 703-707.



Neuroimaging - Cognitive and Clinical Neuroscience

Edited by Prof. Peter Bright

ISBN 978-953-51-0606-7

Hard cover, 462 pages

Publisher InTech

Published online 16, May, 2012

Published in print edition May, 2012

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).

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

B. Alexander Diaz, Lizeth H. Sloom, Huibert D. Mansvelder and Klaus Linkenkaer-Hansen (2012). EEG-Biofeedback as a Tool to Modulate Arousal: Trends and Perspectives for Treatment of ADHD and Insomnia, *Neuroimaging - Cognitive and Clinical Neuroscience*, Prof. Peter Bright (Ed.), ISBN: 978-953-51-0606-7, InTech, Available from: <http://www.intechopen.com/books/neuroimaging-cognitive-and-clinical-neuroscience/eeg-biofeedback-as-a-tool-to-modulate-arousal-trends-and-perspectives-for-treatment-of-adhd-and-inso>

INTECH
open science | open minds

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

www.intechopen.com

www.intechopen.com

IntechOpen

IntechOpen

© 2012 The Author(s). Licensee IntechOpen. This is an open access article distributed under the terms of the [Creative Commons Attribution 3.0 License](#), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

IntechOpen

IntechOpen