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SMR NEUROFEEDBACK TRAINING FOR COGNITIVE ENHANCEMENT: THE MEDIATING EFFECT OF SMR BASELINE LEVELS

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ABSTRACT

FACULTY OF SCIENCE AND TECHNOLOGY

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The demands posed by a competitive society and by high life expectancy foster the interest in methods capable of enhancing cognitive abilities, such as neurofeedback training. However, the use of neurofeedback training to improve cognitive performance is still experimental and more studies with appropriate control groups and pre and post training measures are required. Furthermore, it has yet to be established whether individual electrophysiological features can influence the ability to learn to control electrophysiological variables.

In this study, 24 adults without any psychological or neurological disorders participated either in 10 neurofeedback training sessions to increase the amplitude of a frequency band between 12 and 15 Hz (sensorimotor rhythm - SMR) or in ten mock neurofeedback sessions. Pre and post training measures of memory and executive functions were completed, along with quantitative electroencephalography (QEEG) measurements in order to detect changes after the training course. Furthermore, measures of SMR amplitude were taken within and across sessions to determine whether self-regulation of SMR had been achieved.

The data analysis performed shows no significant differences in cognitive performance between the group who underwent neurofeedback training and the group who underwent mock neurofeedback training. The groups did not show electrophysiological changes after the training. Additionally, no significant changes in SMR amplitude or percent time above threshold across or within the 10 sessions were found in the experimental group. Moreover, the data showed a tendency, which indicates that the higher the baseline amplitude and absolute power of SMR the less

time was spent above threshold during the training and the less increase in SMR amplitude between baseline and training periods.

The findings obtained indicate that neurofeedback training did not affect memory, executive functions or the QEEG. The absence of significant changes in SMR amplitude across sessions might reflect failure in learning the neurofeedback task and may account for the lack of cognitive improvement and QEEG changes. The fact that the ability to self-regulate SMR might be dependent on baseline amplitude has important implications in setting thresholds. Setting thresholds according to baseline levels might increase the difficulty in maintaining SMR above threshold when the baseline is higher. Future research should also address whether baseline amplitude has a predictive value in determining successful self-regulation of brain activity.

Keywords: Neurofeedback training, sensorimotor rhythm (SMR), mock neurofeedback training, cognitive enhancement

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CHAPTER ONE
INTRODUCTION

1.1. GENERAL OVERVIEW

The human brain is a complex organ regulated by neurochemical and electrophysiological processes, which are often investigated using neuroimaging methods. These methods are essential for the understanding of brain mechanisms and structures. Techniques such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), single photon emission computed tomography (SPECT), optical imaging, or electroencephalography (EEG) are used to identify the brain's anatomical and functional aspects involved in a normal functioning brain and in psychopathology. Furthermore, these technologies can be used to assess research and treatment results. The EEG is one such neuroimaging method, which is devoted to the study and recording of electrical activity in the brain, the electroencephalogram being the resulting record (Noachtar et al., 1999).

The quantitative EEG (QEEG) is the processing of the EEG signal through mathematical methods in order to emphasize and quantify specific EEG components (Nuwer, 1997). One of the most important developments introduced by the QEEG is that instead of only analyzing EEG activity from a time domain perspective, where variations of amplitude as a function of time are examined, quantitative techniques enable analysis to be made in the frequency domain (Pivik et al., 1993). Analysing the EEG signal in the frequency domain involves decomposing the spectrum of the EEG into frequency ranges (bandwidths) and measuring the amount of energy (amplitude or power) distributed in each frequency band (Cantor, 1999; Pivik et al., 1993). Although bandwidth definitions can vary (Kaiser, 2000, 2006), commonly the frequencies considered are delta (between 0.5 and 4 Hz), theta (4-8 Hz), alpha (8-13 Hz), beta (14-40 Hz) and gamma (> 40 Hz) (Noachtar et al., 1999). Each frequency can be described and measured in terms of hertz (Hz) and microvolts (μV). Hertz describe the rhythms of the wave, where 1 Hz is one cycle per second (Demos, 2005). Amplitude is measured as the height of the wave from peak to peak and is expressed in microvolts (Noachtar et al., 1999). The square of the amplitude is defined as power, expressed in μV^2 (Thatcher, 1999).

The possibilities brought by QEEG surpass diagnostic and assessment purposes and include self-regulation interventions, such as neurofeedback training. Self-regulation strategies can extend themselves beyond behaviour and psychological processes and can also encompass autonomic physiological functions, such as skin conductance or blood pressure. Biofeedback is one of such strategies, which aims to increase awareness of physiological functions and consequently their voluntary control through the use of electronic equipment that measures physiological processes and feeds back that information (Schwartz & Schwartz, 2005). Biofeedback therapies may be a possibility to consider in cases that do not respond to mainstream treatments, when the clients show adverse effects to medication, and also for people who prefer therapies based on principles of self-regulation, which might be considered more natural (Moss & Kirk, 2004).

Neurofeedback is a biofeedback modality specially focused on the regulation of the electrical activity of the brain. During neurofeedback training certain electrophysiological parameters are recorded and when these parameters meet the conditions set at the beginning of the training (for example, when the amplitude of a certain brainwave surpasses a certain threshold), this information is fed back to the individual using visual or audio stimuli. It is expected that this information helps the individual to acquire control over an electrophysiological feature that they were unaware of before. According to the operant learning paradigm, the feedback information works as a reward, increasing the probability of appearance of specific electrophysiological patterns (e.g. Fultz, 2002).

The origins of neurofeedback training go back to the late 1950s and throughout the 1960s, when Dr. Joe Kamiya, at the University of Chicago, trained volunteer participants to recognize the generation of alpha rhythm (8-12 Hz activity), and discovered that when participants were trained to discriminate alpha activity they also could learn to control alpha intentionally (Kamiya, 1976). Sterman, LoPresti and Fairchild (1969) also found that cats trained through operant conditioning to produce SMR showed a later onset of seizures or no convulsions at all, even though they had been exposed to monomethylhydrazine (MMH), which is a convulsive compound. These results led to investigations in humans that demonstrated the potential benefits of SMR training for epilepsy (e.g. Sterman, Macdonald, & Stone, 1974) and afterwards for ADHD (e.g. see review by Monastra et al., 2005), disorders for which neurofeedback training is considered efficacious (Yucha & Montgomery, 2008). These first findings

showed not only that control of electrophysiological activity was possible, but it could also be used in the treatment of clinical disorders.

Research showing symptom reduction after neurofeedback training in several clinical disorders is growing (e.g. Arns, Ridder, Strehl, Breteler, & Coenen, 2009; Rice, Blanchard, & Purcell, 1993; Scott, Kaiser, Othmer, & Sideroff, 2005; Sterman, 2000). However, another focus of interest is also developing: the use of neurofeedback training in healthy people for performance enhancement. This study is particularly focused on the application of neurofeedback training to improve cognitive performance and electrophysiological parameters (as measured by the QEEG) in healthy individuals.

1.2. PROBLEM STATEMENT

Nowadays there has been a growing interest in medication that can improve cognitive functioning, as indicated by the use of prescription drugs in people without diagnosed disorders (Compton & Volkow, 2006; Farah et al., 2004; McCabe, Teter, & Boyd, 2006). However, besides medication other methods might hold the promise of improving cognitive abilities, without the often cited complications associated with the use of medication (e.g. Mehlman, 2004). One of these techniques might be neurofeedback training (NFT).

The applications of neurofeedback training have been studied mostly in the context of clinical disorders, such as Attention Deficit Hyperactivity Disorder (AD/HD), anxiety disorders, autism and brain lesions (see Yucha & Montgomery, 2008).

Neurofeedback training for performance enhancement is also an area of interest with possible applications in the context of arts, sports and cognition (for a review see Vernon, 2005, 2009). However, several limitations have been directed at the studies conducted so far, such as lack of non contingent control groups or the lack of pre and post QEEG measures that make it possible to correlate changes in behaviour to changes in the EEG (Vernon, 2005). Alternatively, one of the problems with the use of placebo conditions in neurofeedback research is that it has been considered easily detected by participants (Kotchoubey et al., 2001).

Another aspect worth exploring is for whom neurofeedback training might work best. The psychopharmacological literature suggests that the improvement in cognitive functioning in healthy individuals may be modulated by baseline performance, in that a

poor baseline performance is associated with significant improvement, while higher baseline performance is associated with less improvement (for a review, de Jongh, Bolt, Schermer, & Olivier, 2008). Following the same line of thought, baseline levels of electrophysiological variables may also influence performance during neurofeedback training. Although previous research has tried to distinguish people who learn to control EEG parameters from the ones who do not (e.g. Dempster & Vernon, 2009), little has been done in order to understand which individual variables might be playing a role in mediating the ability to self control the EEG.

In conclusion, there is an absence of evidence supporting the usefulness of neurofeedback training to improve cognitive performance, which, in part, is caused by the lack of rigorous methodology in some of the studies conducted so far. Moreover, the variables that influence the ability to learn to control the EEG through neurofeedback training are still unclear and in need of further scrutiny.

1.3. AIMS OF THE STUDY

The aims of the study are to test in a controlled manner (comparing an experimental group with a placebo control group):

- whether SMR up-training is able to improve cognition (executive functions and memory);

- whether possible cognitive changes in the experimental group are accompanied by EEG changes.

- if baseline levels of electrophysiological variables influence the ability to control SMR.

1.4. SIGNIFICANCE OF THE STUDY

The proposed study will focus on the application of neurofeedback training for cognitive enhancement purposes, which is an area that still needs empirical support (Gruzelier & Egner, 2005; Vernon, 2005). Even though, previous research has focused on the enhancement of executive functions and memory, through SMR up-training (e.g. Ros et al., 2009; Vernon et al., 2003), a clear association between the training and the cognitive enhancement observed has not been established yet. In addition, to the researcher's knowledge, the analysis of the influence of electrophysiological baseline

levels of performance on learning the neurofeedback task has not been addressed before and it would be important in determining the individual factors that mediate learning.

Finally, the research design and methods will ensure that the aims of the study are met in a controlled and rigorous manner, through the inclusion of a control group, randomized allocation to groups, and neuropsychological and psychophysiological measures (taken before and after neurofeedback training). This controlled methodology has often not been achieved in research concerning neurofeedback. Therefore this study will make an original contribution to the neurofeedback field and will add to existing knowledge.

1.5. OUTLINE OF THE THESIS

Chapter two presents a review of the relevant literature, which informs the research goals and hypotheses. Chapter three outlines the method used in this study and discusses the decisions made. Chapter four presents the description of the statistical analyses employed to analyse the data, along with the results of the study. Chapter five discusses the key findings of the research, draws conclusions and presents future suggestions. Moreover, possible limitations of the study are also discussed.

CHAPTER TWO
LITERATURE REVIEW

2.1. INTRODUCTION

The literature reviewed in this chapter is focused, first of all, on the rationale that underlies the use of neurofeedback training for clinical and non clinical applications. The relevance of using neurofeedback training as a cognitive enhancement technique will be discussed in comparison with other techniques. The rationale for enhancing executive functions and memory will be given. Studies focused on the effects of SMR training on cognition will be reviewed, with particular emphasis and detail being given to research in non clinical populations. A special focus will be given to the relationship between SMR enhancement and executive functions and memory. Moreover, the literature on mediating effects of baseline levels of electrophysiological and cognitive variables on the changes that can result from cognitive enhancement interventions will be overviewed.

Finally, an introduction to the proposed study will be presented and hypotheses will be formulated according to the literature reviewed.

2.2. RATIONALE AND APPLICATIONS FOR NEUROFEEDBACK TRAINING

Neurofeedback can be best understood when considering the neurophysiological mechanisms that underlie learning. Neuronal plasticity, for example, enables the brain to continuously change its structure and its function throughout the life span (Kolb, 1995). Although this characteristic of the brain is most evident during development, the brain remains malleable throughout life (Huttenlocher, 2002; Kolb, 1995; Raymont & Grafman, 2006). This means that the adult brain is still capable of being influenced by experience. The capacity to learn and to remember depends upon the brain modification in response to experience (Kolb, 1995; Maren & Baudry, 1995; Rosenzweig & Bennet, 1996).

Neuromodulation and long-term potentiation (LTP) are two relevant processes for brain plasticity (Abarbanel, 1999). Neuromodulation is a type of neurotransmission distinguished from traditional neurotransmission mostly due to the action of metabotropic receptors which exert a great influence in electrophysiological properties

of a cell (e.g. resting potential, threshold potential). LTP is the increased synaptic transmission efficiency as the result of high-frequency synaptic activation (Andersen, 2004). LTP enables structural and biochemical changes to last, as the strength of synapses is potentiated and persists after synaptic activation (Bliss, Collingridge & Morris, 2004). Therefore, it is thought that during neurofeedback training the relevant neural networks are modified through neuromodulation (Abarbanel, 1999) and that these changes are made persistent during ongoing feedback training through LTP (Abarbanel, 1999; Sterman & Egner, 2006).

It has been widely documented in the scientific literature that the EEG reflects information processing (e.g. Başar, Başar-Eroglu, Karakaş, & Schürmann, 2000; Başar, Başar-Eroglu, Karakaş, & Schürmann, 2001; Petsche, Kaplan, Stein, & Filz, 1997). Klimesch (1996) believes that electrophysiological processes are the foundation for information transmission and that cognitive processes may be explained on the basis of brain oscillations that are present in specific cortical areas. Evidence exists to support the hypothesis that different cognitive processes are reflected by different and narrow frequency bands (e.g. Klimesch, Doppelmayr, Pachinger & Russegger, 1997).

The fact that different bandwidths have distinct functional significance provides a good rationale for the use of neurofeedback training to facilitate control over electrical brain activity. It is expected that neurofeedback training helps to exert control over specific EEG parameters and consequently over the functions associated with them. In the case of clinical applications, it is hoped that neurofeedback training helps to normalize electrophysiological imbalances (e.g. Monastra et al., 2005), whereas in the case of peak performance or non clinical applications it is hoped that EEG patterns associated with optimal functioning are reinforced (Vernon, 2005).

Neurofeedback training has been used in a variety of disorders such as, alcoholism and substance abuse, anxiety, AD/HD, autism, depressive disorders, epilepsy, insomnia, post-traumatic stress disorder (PTSD) and traumatic brain injury (Yucha & Montgomery, 2008). There is also some evidence suggesting that the ability to learn to control the EEG through neurofeedback training and the subsequent behavioural and cognitive improvements can be maintained in the long-term (e.g. Leins et al., 2007; Monastra, Monastra, & George, 2002).

Neurofeedback training can also be used in non-clinical contexts with the aim of improving performance in specific areas. These areas include sports, cognitive and artistic performance (Vernon, 2005, 2009).

2.3. NON-CLINICAL APPLICATIONS OF NEUROFEEDBACK TRAINING: THE CASE FOR COGNITIVE ENHANCEMENT

2.3.1. Techniques to improve cognition

In the 1950's the humanistic theories called for a rethinking of the principles that guided psychological clinical practice and urged for a more positive view of the human being that went beyond psychopathology and its remediation. Abraham Maslow, for example, studied what he considered to be psychologically healthy people and concluded that there is a tendency in the human being to grow towards psychological health, creativity or individuality, just to mention a few aspects, a tendency known as self-actualization (e.g. Maslow, 1987, 1999). This perspective of human beings as constantly evolving and growing is also present in the work of one of the most well known proponents of the humanistic movement, Carl Rogers (e.g. Rogers, 1961).

The ideas proposed by the humanistic movement are also reflected in the Positive Psychology approach. Seligman and Csikszentmihalyi (2000), in a special issue of the *American Psychologist* dedicated to Positive Psychology, stated that Psychology should be a science and practice with a stronger focus on identifying and building strengths, in their words, on "making normal people stronger and more productive and making human potential actual" (p.8). Performance enhancement seems to be in line with these goals, as it aims to promote optimal levels of performance in healthy people whose performance falls within normative levels (Vernon, 2009).

Cognitive abilities are among the areas that are able to be enhanced. Cognition is usually considered a set of skills encompassing attention, memory, language, learning, praxis and executive functions (Reed, 2007; Sternberg, 2009; Whitehouse, Juengst, Mehlman, & Murray, 1997). Factors such as the increasing life expectancy and the competitive society that we live in might explain why enhancing cognition is appealing for so many people. Many drink coffee with the hope that this will boost their attention levels and will keep them awake throughout the day. The desire to improve cognition to meet academic deadlines and to improve grades is, in some cases, the reason why many students misuse stimulants to the detriment of their own health (e.g. Sussman, Pentz, Spruijt-Metz, & Miller, 2006; White, Becker-Blease, & Grace-Bishop, 2006).

The methods used to enhance performance are varied (for reviews, see Bostrom & Sandberg, 2009; Bush, 2006; Vernon, 2009) and some have been used for thousands of years, like herbal extracts or the practice of yoga and meditation (Bostrom & Sandberg, 2009). Education and training also aim to improve cognitive performance, in that its focus is not only concerned with the development and acquisition of specific skills and information but also with improving general cognitive capacities, such as concentration or critical thinking (Bostrom & Sandberg, 2009). Schooling is an important factor for better outcomes on psychometric measures of intelligence (Ceci & Williams, 1997). Other factors such as nutrition, child rearing practices and the development of high technology have been considered some of the causes for the increases observed on intelligence measures over time (Flynn, 1987; Neisser, 1997), a phenomenon commonly referred to as the Flynn effect (Herrnstein & Murray, 1994).

Mental practice (i.e. as the cognitive rehearsal of a task before its performance, in the absence of actual rehearsal) is another method that seems to be effective at enhancing performance, particularly if the task involves cognitive elements (Driskell, Copper, & Moran, 1994). Other potential methods for cognitive enhancement include genetic modification (e.g. Sisodiya et al., 2007; Tang et al., 1999) and transcranial magnetic stimulation (TMS) (e.g. Bütefisch, Khurana, Kopylev, & Cohen, 2004).

Some of the enhancement methods, like some herbal extracts, do not have enough scientific evidence in their favour (e.g. Bent, 2008; Gold, Cahill, & Wenk, 2002; Ness & Sherman, 1999), other methods, such as genetic modification and psychopharmacology, lead to ethical questions mostly related with issues of social equality, safety and moral values (e.g. Bush, 2006; Farah et al., 2004; Greely, Campbell, Sahakian, Harris, & Kessler, 2008; Hall, 2004; Persson & Savulescu, 2008; Turner & Sahakian, 2006). At the moment, these ethical concerns, are more focused on the use of psychopharmacology, which according to Farah et al. (2004) might be due in part to the fact that this method is already being used, while the use of other methods is still hypothetical. Moreover, the risks associated with medication for enhancing performance are still unclear (Turner, 2008), with physical and psychological dependency, and toxicity being possible side effects (Mehlman, 2004).

In addition, Bush (2006) points out that fewer risks can be accepted for cognitive enhancement purposes in healthy people as compared to the risks that result from interventions that aim to treat a disease, where the benefits obtained may surpass the side effects. Wolpe (2002) also claims that methods that have a direct influence in the

brain raise more ethical concerns than methods that are based on the resources of the body or methods that change the external surroundings to indirectly alter brain functioning, such as meditation or biofeedback. Neurofeedback training as a biofeedback modality that relies on learned self-regulation of brain activity, therefore on one's individual resources, falls within this last category.

The appeal of neurofeedback, being a self-regulation technique, may stem from the fact that it empowers the client to be involved in the process of self change instead of being a passive recipient of interventions. The concepts of empowerment, self-regulation and active involvement are in line with the ideas promoted by the humanistic movement (e.g. Criswell, 2001) and more recently by the Positive Psychology movement (e.g. Joseph & Linley, 2004).

The efficacy of neurofeedback training in enhancing cognition has not been established yet, making its use for cognitive enhancement an experimental application (Vernon, 2005). Executive functions and memory are two skills that might be able to be enhanced through neurofeedback training.

2.3.2. Executive functions and memory

Given that executive functions encompass several complex processes, most definitions are supported by examples (Elliott, 2003). Examples of behaviours controlled through executive functions include goal setting, planning, evaluation and choice of alternative behaviours, performance monitoring, sustained attention, working memory and response inhibition (Rabbitt, 1997). In addition, executive control is particularly relevant in novel and complex situations (Rabbitt, 1997).

The existence of executive functions was suggested from the observation of brain lesions, usually in the frontal lobes (Aron, 2008; Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). Overall, people with frontal lobe lesions often show difficulties in regulating behaviour and with normal functioning in everyday life (Miyake et al., 2000). It is often the case that performance on tests of executive function is considered dependent on the frontal lobes, and the terms 'frontal lobe function' and 'executive function' are even used interchangeably (Elliot, 2003). However, the frontal cortex is not the only area responsible for executive functions, as indicated by the fact that executive functioning can be compromised without frontal lesions and frontal impairment does not necessarily lead to deficits in executive functions (Andrés, 2003;

Godefroy, 2003; Royall et al., 2002). Furthermore neuroimaging studies have found that although the prefrontal cortex is an important structure during the performance of executive tasks, other cortical areas are also activated, such as the temporal or parietal cortices (Andrés, 2003). Therefore it has been suggested that this set of functions are probably best explained as being supported by distributed networks in the brain rather than by discrete structures, such as the frontal regions (e.g. Andrés, 2003; Elliot, 2003).

The relevance of executive functions for the normal functioning of any individual is well reflected when deficits in these functions are observed. Some of the problems that may arise when executive functions are impaired are deficits in self-control, greater irritability, more excitability and disinhibition, difficulty in initiating and suppressing action, decreased or lack of motivation, defective planning, difficulty in performing activity sequences that are part of purposive behaviour, difficulty in shifting attention, perseveration and stereotyped behaviour (e.g. Godefroy, 2003; Lezak, Howieson, & Loring, 2004).

People with neurodevelopmental disorders, such as autistic disorders and AD/HD have shown executive deficits (e.g. Ozonoff & Jensen, 1999). In addition, executive function deficits and frontal damage and metabolic deficits have been detected in neuropsychiatric disorders, such as schizophrenia and major depression, (e.g. Elliot, 2003; Royall et al., 2002). Pathologies such as dementia (including vascular dementia, Alzheimer's disease, and Parkinson's disease), stroke and closed head injury are known to compromise executive functions (e.g. Godefroy, 2003).

Besides executive function deficits, memory impairments also appear in cognitive decline associated with aging and pathological conditions, such as dementia (e.g. Blacker et al., 2007; Buckner, 2004; Grober et al., 2008; Hedden & Gabrieli, 2004; Lindeboom & Weinstein, 2004).

Human memory consists of several systems that enable the encoding, storage and retrieval of information (Baddeley, 1997). Declarative memory refers to the ability to consciously recollect facts and events. Non declarative memory is expressed through performances within the systems where learning occurred in the first place (Squire, 2004). Working memory is a control system, with limited capacity, that manipulates and temporally stores information, supporting thought processes (Baddeley, 2003). It is worth noting that the involvement of working memory, so as to manipulate information and coordinate simultaneous mnemonic activities, is often mentioned as being part of executive tasks (e.g. Baddeley, 2003; Phillips, 1997).

Memory has an important survival value by, for example, enabling the recollection of dangerous events and also by promoting a sense of personal identity through the capacity to recollect personal events and experiences (Glannon, 2006). Moreover, the fact that memory enables the execution of learned abilities, as well as the recollection of events, facts and concepts is essential in the performance of many daily activities.

The enhancement of executive function and memory have often been referred as targets for cognitive enhancement (e.g. Farah et al., 2004; Greely et al., 2008; Hall, 2004; Turner & Sahakian, 2006; Whitehouse et al., 1997). Probably, this is due, in part, to the fact that these skills are prone to enhancement as evidenced by research regarding pharmacological enhancement of memory and executive functions (e.g. de Jongh et al., 2008). In fact, pharmacological agents used in the treatment of AD/HD and dementia which are thought to act on the neural substrates of memory and executive functioning are now being studied in healthy individuals (e.g. Grön, Kirstein, Thielscher, Riepe, & Spitzer, 2005; Turner et al., 2003). Alternatively, the undeniable value of memory and executive functions in the performance of everyday activities is a very good reason to promote their enhancement, through neurofeedback training.

Neurofeedback training protocols for cognitive enhancement have been focused on theta suppression, alpha enhancement, alpha/theta training, beta enhancement, sensorimotor rhythm (SMR) enhancement, or a combination of these (Vernon, 2005). The next section will explore the rationale for using SMR training for cognitive enhancement, particularly for the enhancement of executive functioning and memory.

2.4. SMR TRAINING FOR COGNITIVE ENHANCEMENT

Neurofeedback training to increase SMR has been used successfully in the treatment of AD/HD and epilepsy, two of the three disorders for which neurofeedback training has been considered efficacious (Yucha & Montgomery, 2008), according to the guidelines for the evaluation of the clinical efficacy of psychophysiological interventions (La Vaque et al., 2002). As it will be explored in more detail, the amelioration observed in these disorders is often accompanied by improvements in cognition, some of which might indicate better executive functioning and memory abilities.

2.4.1. SMR training in the treatment of clinical disorders

SMR is a rhythm within the range between 12 and 20 Hz, showing a peak activity approximately at 12-14 Hz (Sterman, 1996; Sterman, 1999). This is a frequency band within the beta range but its designation derives from the location where it is detected – the sensorimotor strip.

The appearance of SMR is usually associated with suppression of body movements, suppression of respiratory activity, muscle tonus decrease and alertness (e.g. Howe & Sterman, 1972; Hummel, Andres, Altenmüller, Dichgans, & Gerloff, 2002; Wyrwicka and Sterman, 1968). For example, motor response inhibition in a GO/NO-GO task was related with SMR synchronization (Zhang, Chen, Bressler, & Ding, 2008). Mann, Sterman and Kaiser (1996) found out that body movements led to a selective suppression of 11-15 Hz in the central cortex when compared to visual tasks. The observation of body movements can also lead to significant power decreases in a frequency band from 13 to 18 Hz over the central cortex (Cochin, Barthelemy, Lejeune, Roux, & Martineau, 1998).

However, SMR is not only related to suppression of motor activity but also with cognitive activity. For example, associations between sleep spindles, which are bursts of EEG activity within the range of 12 to 14 Hz occurring in stage 2 sleep (Castronovo & Butkov, 2007) and cognitive abilities have been found. Schabus et al. (2006) found that sleep spindles were associated with general cognitive ability (as measured by the Raven's Advanced Progressive Matrices) and memory (measured by the Wechsler Memory scale-revised). In a study by Rosanova and Ulrich (2005), stimulating spindles patterns induced long-term potentiation, which is a mechanism known to underlie learning and memory (e.g. Andersen, 2004; Bliss et al., 2004). Gais, Mölle, Helms and Born (2002) found that sleep spindle density (mean number of spindles per 30 seconds epoch) was significantly higher after a learning task compared to a non-learning task. A significant positive correlation between sleep spindle activity and recall performance was also found. The association between sleep spindles and memory performance is also suggested in a study by Clemens, Fabó and Halász (2005), where a significant positive correlation between overnight verbal memory retention and number of sleep spindles, was encountered.

As already mentioned in the first chapter, neurofeedback training has been mostly studied in relation to its clinical applications, such as the treatment of AD/HD. The rationale for using neurofeedback training in the treatment of AD/HD stems from the literature showing an abnormal pattern of electrophysiological activity among these patients, as well as from the fact that response to medication is sometimes insufficient and carries side effects (Monastra et al., 2005). Overall, the electrophysiological patterns often found are indicative of hypoactivation, as reflected by the increased slow wave activity and decreased fast wave activity (Lubar, 1991). A decrease in relative and absolute beta power was encountered as one of the most distinctive patterns in people with AD/HD (e.g. Bresnahan et al., 1999; Clarke et al., 1998, 2001a, 2001b, 2001c). Within the beta range, the particular association between sensorimotor inhibition and SMR (already discussed in the beginning of this section) provided the rationale for the introduction of SMR reinforcement in the neurofeedback training protocols of AD/HD patients, for whom overactivity is a main feature (Lubar and Shouse, 1976).

Research aimed at investigating the use of SMR neurofeedback training in the treatment of AD/HD showed improvements in measures of impulsivity and attention (e.g. Carmody, Radvanski, Wadhwani, Sabo, & Vergara, 2001; Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; Kaiser & Othmer, 2000; Rossiter, 2004; Rossiter & La Vaque, 1995), behavioural indices (e.g. Shouse & Lubar, 1979), changes in subcortical areas associated with response inhibition and selective attention (e.g. Beauregard & Lévesque, 2006) and an increase in event-related potentials (ERP) components associated with executive functions during a GO/NOGO task (Kropotov et al., 2007).

In addition, in children with learning disabilities, increases in intelligence (as measured by the Wechsler Intelligence Scale for Children – Revised), were observed after neurofeedback training aimed at increasing the amplitude and frequency of 14 Hz over the central sensorimotor cortex (Tansey, 1984). Tansey (1984) hypothesized that the improvement observed was due to an increased functional symmetry in the interactions between brain hemispheres caused by up-training the SMR.

Kouijzer, Moor, Gerrits, Congedo, and Schie (2009) found that in children with Autisitic Spectrum Disorders (ASD) a course of neurofeedback training, involving the enhancement of SMR (12-15 Hz), resulted in significant changes in tasks involving executive functions. In this study participants (N = 14) were assigned either to an experimental group, who underwent neurofeedback training, or a waiting-list control

group. After neurofeedback training, the experimental group, when compared to the control group, showed significant improvements in auditory selective attention and motor response inhibition (measured by the Test of Sustained Selected Attention), verbal response inhibition (assessed by the Stroop Test), set shifting ability (measured by the Trail Making Test), concept generation and use of feedback (measured by the Milwaukee Card Sorting Test) and planning ability (assed by the Tower of London).

Another major application of neurofeedback training including SMR enhancement in the training protocol is in the treatment of epilepsy. In fact, after the finding that seizures were absent or had higher thresholds in cats as the result of training to enhance SMR (Sterman et al., 1969), the use of SMR training in the treatment of epilepsy was reported in humans with positive results (e.g. for reviews see Monderer, Harrison & Haut, 2002; Tan et al., 2009). Furthermore, improvements in memory and executive functions (assessed by the Stroop-Color Word Test) in participants who were able to control SMR and who showed greater seizure reduction were observed (Lantz & Sterman, 1988; Sterman & Lantz, 2001). Sterman and Lantz (2001) found that participants with left hemisphere lesions showed significant improvements on the Visual Reproduction subtest of the Wechsler Memory Scale, as well as on the Seashore Tonal Memory Test, which are tests mostly involved in right hemisphere processing. Participants with right hemisphere lesions showed significant improvements on the Logical Memory subtest of the Wechsler Memory Scale, and on the Buschke Word List Recall Test, both associated with left hemisphere processing. These results are interpreted as an indication that neurofeedback training might reduce the abnormal electrical activity originated from the impaired hemisphere, which could account for the specific improvement seen only in the unimpaired hemisphere (Sterman & Lantz, 2001).

The knowledge of the origins of the SMR has provided the basis for the understanding of how its operant control may have benefits for some clinical disorders and for healthy individuals (Sterman & Egner, 2006). SMR seems to be originated in the thalamus, specifically in the ventrobasal nuclei, which is the somatosensory relay nuclei of the thalamus that conveys to the cortex signals from sensory pathways (Sterman, 1996). When ventrobasal cells become hyperpolarized due to the attenuation of the sensory input, a bursting discharge initiates and SMR activity appears (Sterman, 1996). Therefore, during inactive behaviour SMR activity initiates. On the other hand, during active behaviour excitatory inputs suppress cellular bursting and reduce EEG

rhythms in the sensorimotor cortex (Sterman, 1996). In addition SMR activity may be influenced by cholinergic and monoaminergic neuromodulation that affects thalamic relay nuclei and the cortical areas that receive the relayed signals (Sterman & Egner, 2006). For example, during waking states, ventrobasal cells are kept depolarized by neuromodulator influences that suppress SMR, which means that neuromodulation might interfere with excitability levels of cell populations (Sterman, 1996; Sterman & Egner, 2006).

Since SMR is the dominant frequency of the integrated thalamocortical somotamotor and somotosensory pathways its operant control through neurofeedback training is thought to increase the ability to control excitation in this system. Therefore disorders characterized by cortical or thalamocortical hyperexcitability seem to improve as a result of SMR training due to increased thresholds for excitation (Sterman & Egner, 2006). Moreover, according to Sterman (1996), motor activity associated to abnormal sensorimotor excitability might interfere with cognitive processing, since motor activity can disrupt perceptual and integrative components of information processing. This could mean that decreasing motor interference through SMR training may result in better cognitive performance.

Although neurofeedback training to enhance SMR for the treatment of clinical disorders has achieved positive results, methodological problems limit the conclusions of some of the studies mentioned above, such as the lack of control groups (e.g. Kaiser & Othmer, 2000), small sample sizes (e.g. Kouijzer et al., 2009), use of case studies (e.g. Tansey, 1984), the employment of other treatment modalities in conjunction with neurofeedback training (e.g. Shouse & Lubar, 1979) and the lack of EEG assessments (e.g. Fuchs et al., 2003). Similar problems are also encountered when using neurofeedback training in healthy people (for a review, see Vernon, 2005).

2.4.2. SMR training for the improvement of cognitive abilities in healthy people

The impact of neurofeedback training in enhancing several cognitive abilities in healthy individuals has been explored in the last decade, leading to some promising results.

Egner and Gruzelier (2001) employed SMR (12-15 Hz) training over C4 and beta training (15-18 Hz) over C3 in a group of 22 healthy individuals, while simultaneously

inhibiting theta (4-7 Hz) and high beta (22-30 Hz). After ten sessions, SMR training was associated with performance enhancement in impulse control (reflected in less commission errors, i.e. decreased response to non target stimulus) and perceptual sensitivity – d´(which is a signal detection measure, expressing the ratio of correct to false detection rate), while the opposite association was found for beta training. Another result coming from this experiment is that the enhancement of both frequency bands was associated with an increase in P300 amplitudes in an auditory oddball task. P300 is a component of event-related potential (ERP), usually generated in tasks requiring attention and discrimination between stimuli, like an oddball paradigm, when an occasional target must be detected among non target stimuli (Picton, 1992; Polich & Kok, 1995). However, Vernon (2005) points out some limitations in this study, such as the lack of a control group or absence of significant EEG changes following neurofeedback training. Furthermore changes in P300 amplitude were not associated with performance on the auditory oddball task, where relevant changes were not found. These limitations raise the question if the effects observed were due to non specific factors, such as practice effects.

In another study by Vernon et al. (2003), neurofeedback training was employed with two groups: a theta-group, i.e. a group that underwent training to enhance theta (4-8 Hz), while inhibiting delta (0-4 Hz) and alpha activity (8-12 Hz); and an SMR-group, trained to enhance SMR (12-15 Hz) and simultaneously inhibiting theta and beta (18-22 Hz). The training lasted 8 sessions and occurred over Cz. A third group of participants was part of a non-training condition and all participants were randomly allocated to one of the three groups. Participants in the SMR-group learned to increase SMR/theta and SMR/beta ratios within sessions, showing limited improvements in the accuracy of attentional processing and improvement in a semantic working memory task (Vernon et al., 2003). It was hypothesized that the improvement produced by SMR training on working memory performance could be related to the fact that training in this frequency band may help to maintain the memory representation used in semantic working memory, which is in line with the work of Haarman and Cameron (2005). Nevertheless, a causal link between neurofeedback training and improvement in the semantic working memory task is not possible to establish because changes in the training parameters only occurred within sessions but not across sessions.

Egner and Gruzelier (2004) compared different neurofeedback training protocols in order to evaluate the differential effects on attention. Twenty five volunteers were

randomly allocated to one of three groups: a non neurofeedback training group that underwent Alexander technique training; an SMR (12-15 Hz) group, trained to enhance SMR and to inhibit theta (4-7 Hz) and high beta (22-30 Hz); and a beta 1 group, trained to enhance the 15-18 Hz frequency band, while simultaneously avoiding increases in theta and high beta activity. Neurofeedback training was carried out at Cz and lasted 10 weekly sessions. Before and after the training participants were tested using the TOVA (Test of Variables of Attention), the divided attention task and the oddball task (only used for the neurofeedback training groups). The results obtained support protocol-specific effects on measures of attentional processing: beta1 training was related with faster reaction times and increments in target P300 amplitudes, but effects on either impulsive or inattentive errors were not found; SMR training, on the other hand, was associated with improved perceptual sensitivity (d´), reduced omission errors and reaction time variability (RTV). These results were interpreted as demonstrating that SMR training does not affect only impulsive aspects of attention and may have an important role in reducing sensorimotor processing interference (Egner & Gruzelier, 2004), as suggested by Sterman (1996). The fact that no EEG pre and post changes were reported for the neurofeedback group and the fact that changes in P300 amplitude were not coincident with the performance in the oddball task limits the results, as a link between electrophysiological measures and behavioural changes cannot be established (Vernon, 2005).

Fritson, Wadkins, Gerdes, and Hof (2007) explored the effects of neurofeedback training on measures of response control and attention. Two groups of healthy participants (n = 32) were randomly assigned to a neurofeedback condition or to a control condition. In the neurofeedback condition participants received SMR training (12-15 Hz), while inhibiting theta (4-7 Hz) and high beta (22-36 Hz), whereas in the control condition participants received placebo or sham neurofeedback training. In the sham condition, participants were initially informed that they were receiving feedback based on their own brain activity, however the feedback received was based on a previously recorded EEG from another person. A bipolar sensor placement was used, whereby C3 was the active site and C4 was the referential site. Sensor placements were similar for both groups and participants attended a total of 20 twice-weekly sessions. The results indicated that significant post training changes were obtained in the experimental group in measures of response control, however no significant changes were found for measures of attention for either the control or treatment group. Although

this study has the advantage of including a control group, electrophysiological measures indicating whether self-regulation of the neurofeedback parameters had been achieved were not reported.

Hoedlmoser et al. (2008) tested whether SMR training had any effect on sleep and declarative memory performance. They recruited 27 participants, which were randomly assigned to either an experimental group (n=16) or a control group (n=11). The experimental group received a course of 10 neurofeedback training sessions in consecutive days, lasting one hour (with 24 minutes of actual training), whereby the increase in SMR amplitude was reinforced at C3. The control group was trained to enhance the amplitude of a different frequency range in every session, with the exception of SMR. Before and after the training the participants performed a declarative word-pair association task that preceded a 90 minutes sleep period. Before and after the nap the participants had to perform a cued recall. The results revealed that, compared to the control group, the experimental group showed an increase in SMR relative amplitude from the first sessions to the last sessions and during sleep. Furthermore, the retrieval scores on the word-pair association task after neurofeedback training increased significantly for the experimental group, indicating improved declarative learning.

Ros et al. (2009) randomly assigned 20 trainee ophtalmic microsurgeons to an SMR-Theta training group (n=10) or an Alpha-Theta training group (n=10). The SMR-Theta group training aimed at enhancing 12-15 Hz activity while at the same time suppressing theta (4-7 Hz) and high beta (22-30 Hz) activity at Cz. The Alpha-Theta group was trained to enhance the ratio of theta (5-8 Hz) over alpha (8-11 Hz), while inhibiting delta activity (1-4 Hz) at Pz. Four participants from each group were randomly selected to be part of a waiting-list control group before their participation in the experimental condition. The course of neurofeedback training consisted of eight neurofeedback sessions that lasted 30 minutes. The results indicated that the SMR training group showed improvements in surgical technique and decrease in total surgery time. According to Ros et al. (2009), the development of surgical skills are thought to involve the retention of strict motor procedures and central executive control processes (attention), therefore the results were interpreted in part as indicating improvements in these skills. However, this study is limited by the small sample size and lack of pre and post EEG assessments. Although this investigation included a wait-list control group and the two experimental protocols used had a different impact in performance, the

study could have benefited from including a sham neurofeedback control group, so as to rule out placebo effects.

Overall, the results obtained up to now suggest there is potential for neurofeedback training to enhance cognitive performance. However, as can be concluded from the studies reviewed, some factors limit the results, such as the absence of control groups, lack of pre and post training measures of the EEG, small sample sizes, or the fact that some electrophysiological measures were not accompanied by behavioural changes in the tasks (for a review, Vernon, 2005).

The demonstration that neurofeedback training is statistically superior to a placebo condition is one of the conditions required for a treatment to be considered "Efficacious and Specific", according to the guidelines for the evaluation of clinical efficacy of psychophysiological interventions (La Vaque et al., 2002). Therefore, its inclusion should also be a condition in neurofeedback research in a non clinical population. However, the inclusion of placebo conditions in which the participants receive feedback that is not based on their own brain activity has been considered inappropriate as it may be easily recognized by participants (e.g. Kotchoubey et al., 2001). However, there is evidence suggesting that providing feedback sounds that have no relation to brain activity does not influence the perception of control of brainwaves, measured by a rating scale, in comparison to a group of participants receiving feedback based on their own brain activity (Raymond, Varney, Parkinson, & Gruzelier, 2005).

The need for pre and post training EEG measures is particularly important given the fact that the rationale behind neurofeedback training is based on the association between particular optimal states or behaviours and certain EEG patterns (see 2.2.). However, this association has not always been well established or addressed in neurofeedback research (e.g. Fritson et al., 2007; Ros et al., 2009). Even when EEG changes are reported as the result of neurofeedback training, sometimes correlations between these changes and behavioural changes are not significant and are mostly attributed to participants' positive expectations (e.g. Pressner & Savitsky, 1977). Plotkin and Rice (1981) reported a decrease in state and trait anxiety in participants regardless of whether they trained alpha enhancement or suppression, when only the group trained to suppress alpha was able to suppress it below baseline levels, whereas in the alpha enhancement group the majority of participants did not show alpha enhancement. Plotkin and Rice (1981) concluded that expectations can be more important in explaining the changes observed as result of EEG biofeedback training, as the

participants were led to believe that the biofeedback task would result in anxiety reduction.

Moreover, sometimes, only changes in EEG parameters, during training, are observed but not after training. For example, Kouijzer et al. (2009) did not find any QEEG changes after training, even though during the training there were changes in EEG parameters related with the training contingencies and even though there were changes in cognitive variables. Analysing the EEG during training can help to determine whether neurofeedback is being successful (whether the participants' brain activity reflects the training contingencies). It is the determination of whether the training is successful or not that enables the establishment of a causal link between neurofeedback training and possible changes in behavioural and electrophysiological dependent variables (Gruzelier & Egner, 2005). Furthermore, comparing pre and post training EEGs enables to investigate any long-term effects of the neurofeedback training in the resting EEG.

Furthermore, training specific frequencies does not necessarily mean that the changes observed are related with the contingencies of training, including frequencies trained and training site (e.g. Barnea, Rassis, & Zaidel, 2005; Egner, Zech & Gruzelier, 2004). However, the lack of correspondence between the training contingencies and EEG post training measures does not attest against the specificity of protocols. For example, alpha/theta neurofeedback training results in replicable frontal beta reduction, which agrees with the training goals in reducing anxiety and agitation (Egner et al., 2004). Furthermore, the specificity of protocols exists in terms of cognitive dimensions, which seem to change according to different contingencies of training (Angelakis et al., 2007; Egner & Gruzelier, 2004). For example, training participants to increase their peak alpha frequency resulted in improvements in executive functioning and speed of processing, whereas training to increase alpha amplitude resulted in memory improvement, and each protocol showed a decrease in the cognitive dimensions improved by the other protocol (Angelakis et al., 2007). Egner and Gruzelier (2004) also found protocol-specific changes in cognitive dimensions, such that, SMR training was associated with improvements in perceptual sensitivity, decreased omissions errors and reaction time variability, and beta1 (15-18 Hz) was associated with increased target P300 amplitudes and faster reaction times.

The specificity of neurofeedback training protocols attests against an explanation of neurofeedback training focused on generic aspects (such as motivational factors).

Furthermore, finding specific effects associated with training contingencies can be useful in developing protocols specific to individual needs. Therefore, more studies including pre and post training QEEG assessments are needed in order to evaluate the relationship between training contingencies, EEG patterns and behavioural and/or cognitive measures.

Moreover, pre and post training measures of EEG and cognitive variables could help determine the possible mediating effect of baseline levels in the changes observed after training.

2.5. THE MEDIATING EFFECT OF BASELINE LEVELS IN THE ABILITY TO CONTROL ELECTROPHYSIOLOGICAL ACTIVITY

The psychopharmacological literature suggests that the use of medication to enhance cognitive performance is dependent on baseline levels of performance, such that low-baseline levels of performance are related with improvement, whereas higher baseline levels of performance are associated with a lack of improvement or worse performance (for a review, de Jongh et al., 2008). For example, the enhancing effect that methylphenidate (which is a psychostimulant medication) has on working memory is greater in participants with a low baseline performance (e.g. Kimberg, D'Esposito, & Farah, 1997; Mehta et al., 2000). Similar results were obtained with other psychopharmacological compounds, such as dextroamphetamine (e.g. Mattay et al., 2000) or modafinil (e.g. Müller, Steffenhagen, Regenthal, & Bublak, 2004), although the opposite result has also been found, i.e. individuals with higher baseline working memory performance have better performances after taking the drug (e.g. Kimberg, Aguirre, Lease, & Esposito, 2001). Similarly, after the administration of Adderal (mixed amphetamine salts) the improvements in a creativity task were observed in participants with low baseline levels of performance, whereas no improvement or impaired performance was observed in participants whose creativity at baseline was higher (Farah, Haimm, Sankoorikal, & Chatterjee, 2008)

The neurofeedback literature has also some examples of how baseline levels of cognitive tests can affect the benefit that is possible to gain after the training. For example, Fritson et al. (2007) reported that in an experimental group that trained to enhance SMR, only measures related with response control showed improvements after training compared with measures of attention, where that the former exhibited higher

baseline levels. Kouijzer et al. (2009) did not observe significant improvements in measures of executive functions (namely tasks that tapped sustained visual attention, and verbal and visual memory) that were performed in a highly efficient manner before neurofeedback training. Recently, Berman and Frederick (2009) also found that general memory ability at baseline was significantly correlated with the improvements observed in cognitive tests (assessing memory and executive functioning) after individualized neurofeedback training (based on QEEG assessment) for people with dementia, in that the higher the baseline memory levels, the higher the standardized mean treatment effect of the cognitive variables that improved.

Lantz and Sterman (1988) also report associations between performance in cognitive tests at baseline and results after training to enhance SMR. On one hand, it was found that a higher performance at baseline in a cognitive test that evaluated problem solving ability was associated with the ability to learn the neurofeedback task (measured by the percentage of change in the number of rewards received for achieving the training contingencies) and greater seizure reduction after neurofeedback training. This fact was interpreted as indicating that higher intellectual ability could facilitate neurofeedback training. Alternatively, a negative association was also found between the baseline performance on tests involving motor components and success at learning the neurofeedback task and subsequent seizure reduction after training, which in turn was interpreted as evidence that the successful use of SMR training for the treatment of epilepsy precluded motor deficits, i.e., motor symptoms during seizures. According to Lantz and Sterman (1988), the normalization of the EEG after SMR neurofeedback training is based on sensorimotor systems, which explains why the training is more beneficial for patients with motor symptoms.

In the psychopharmacological literature the mediating effect of baseline on cognitive enhancement has been interpreted as reflecting individual neurophysiological differences related with neurotransmitter systems (e.g. Mattay et al., 2000). In line with this suggestion, it is reasonable to question whether electrophysiological differences among participants not only impact cognitive performance but also the actual performance in the neurofeedback task, i. e., in the ability to control the training parameters. This suggestion makes sense given that it is assumed that neurofeedback training works, i.e., improves cognitive performance, by changing electrophysiological parameters.

No studies were found in the neurofeedback literature, which address this issue directly but a few results suggest that EEG baseline levels affect the ability to self-regulate brain activity. Shouse and Lubar (1979), for example, in a study whereby the effects of rewarding the production of 12-14 Hz whilst inhibiting 4-7 Hz on four hyperkinetic children were studied, noted that the participants with the lowest amplitudes of SMR at baseline were the ones with the greatest increases in SMR. These observations are obviously limited by the small number of participants.

Rosenfeld, Reinhart and Srivastava (1997) assigned 26 participants to two groups that underwent an acoustic and visual stimulation of either alpha (10 Hz) or beta (22 Hz). It was found that overall, participants with low baseline alpha power exhibited strong entrainment but usually limited to the stimulation period, whereas participants with high level baseline alpha power showed inhibition of alpha power or poor alpha entrainment. Baseline alpha also predicted alpha power in a similar way in the group that underwent beta stimulation, i.e., lower baseline alpha resulted in enhanced entrainment and higher baseline alpha leaded to inhibited entrainment.

It is usual that the threshold levels for rewarding or suppressing certain EEG frequency bands are based on the baseline levels of the parameters being trained (Vernon et al., 2009). Therefore, studying the influence of baseline levels of EEG components on the ability to voluntary control the EEG is highly pertinent.

2.6. CONCLUSIONS AND INTRODUCTION TO THE PROPOSED STUDY

The literature review indicated that neurofeedback training has been mostly used in the treatment of clinical disorders, such as AD/HD, indicating its potential as a cognitive enhancement technique that relies on self-regulation mechanisms. However, the neurofeedback research on cognitive enhancement presents a few methodological shortcomings that prevented the establishment of a clear association between neurofeedback training contingencies, EEG changes and behavioural and cognitive outcomes (see 2.4.2.). The limitations include lack of placebo control conditions and lack of pre and post EEG measures. Therefore, the study described in the next chapter intends to investigate whether neurofeedback training can be used for cognitive enhancement purposes by including in its design pre and post measures of cognitive and EEG variables, as well as including a placebo control condition. Moreover, changes in the training parameters (such as SMR amplitude and percent time above threshold) will

be analysed within and across sessions, so as to investigate the relationship between possible changes in the outcomes measures (cognitive tests and post training EEG) and the contingencies of training.

Moreover, this study intends to be particularly focused on two specific cognitive dimensions: executive functioning and memory (see 2.3.2.). These cognitive abilities have been reported to be impaired in cases of cognitive decline due to aging and disorders, such as dementia, and are crucial skills for the performance of everyday tasks and for the adaptation to the ever changing nature of present society. Moreover, executive functioning and memory have been successfully subjected to change by other cognitive enhancers.

SMR training might be particularly helpful in the enhancement of these cognitive abilities. SMR activity and SMR neurofeedback training is associated with improved executive functioning, as evidenced by the fact that:

- Neurofeedback training involving SMR enhancement in people with AD/HD, ASD and epilepsy leads to improvements in cognitive measures of executive functioning, as well as on ERP changes associated with executive functioning and changes in the neural substrates of inhibition control and selective attention. Furthermore, AD/HD and ASD have been frequently associated with executive functioning deficits. Especially important is the fact that evidence regarding neurofeedback training in the treatment of AD/HD and epilepsy is particularly robust, hence neurofeedback training being considered efficacious in the treatment of these conditions (see 2.4.1.).

- Neurofeedback training involving SMR enhancement in healthy people suggests performance enhancement in impulse control and attention. It was also found recently that SMR training improved microsurgical skills and reduced by 26% the time taken on task (Ros et al., 2009), which can be considered as an indication of executive attention enhancement and retention of rigorous motor procedures (see 2.4.2.).

- The increase of SMR is associated with motor response inhibition and SMR neurofeedback training seems to work by inhibiting motor interference in cognitive processing (2.4.1.).

Second, a few studies also suggest that the enhancement of SMR might be related to improvements in memory:

- Neurofeedback training to increase SMR is related to memory enhancement either in healthy people or people with epilepsy, including declarative memory and working memory (see 2.4.1. and 2.4.2.).

- Sleep spindles within the range of SMR are also associated with memory (see 2.4.1.).

Therefore a protocol that aims at increasing SMR amplitude will be used in this study.

Furthermore, in this study, the relationship between baseline levels of SMR and indices of learning (such as, percent time above threshold and amplitude of SMR) during neurofeedback training will be analysed, as the literature has shown that lower baseline levels of electrophysiological and cognitive variables leads to less difficulties in enhancing those variables as a result of training.

In order to test the aims proposed in chapter 1 and, according to the literature reviewed, the following hypotheses were tested:

1. There will be no significant differences on the perception of control of EEG activity between a control condition that uses sham neurofeedback training and an experimental condition that uses real neurofeedback training.
2. During neurofeedback training, the experimental group will show evidence of having learned the neurofeedback task by showing a significant increase in SMR amplitude within and across sessions.
3. A negative correlation between SMR baseline levels and the ability to enhance SMR is expected, as illustrated in figure 1.
4. After neurofeedback training, the experimental group will be more likely to exhibit significant changes in EEG power according to the training protocol in comparison with the pre training period, as measured by the QEEG, than the control group.
5. There will be significant differences between the experimental and control groups in the improvement observed from the pre to the post training period in measures of executive functioning and memory, with the experimental group showing a significantly higher degree of improvement.

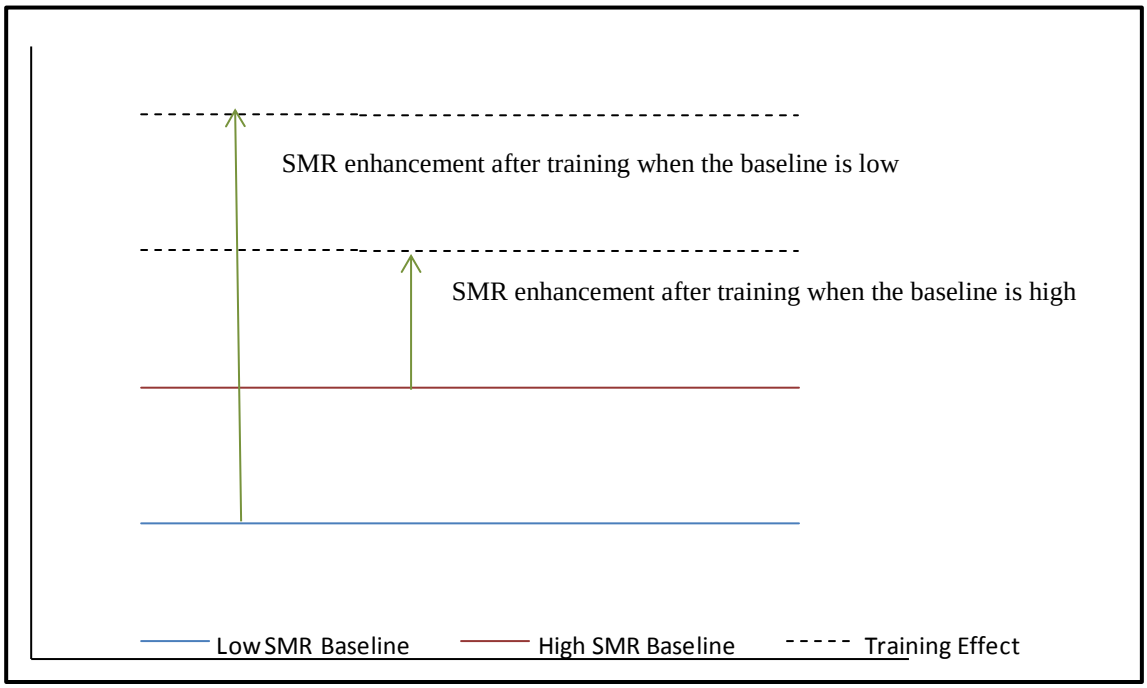

Figure 1 - Illustration of the hypothesis that a negative correlation between SMR baseline amplitudes and SMR enhancement after training is expected.

CHAPTER THREE
METHOD

3.1. PARTICIPANTS

The study sample was self-selecting, as it was formed by volunteer students within Anglia Ruskin University (N = 24). The students were between 18 and 58 years old (M = 26.08, SD = 11.34). 14 participants were female and 10 were male.

In order to screen out participants with a history of mental and neurological conditions, and participants that were taking psychoactive medication, the participants filled a health related questionnaire (see Appendix 5). Only two students could not take part in this research as a result of their answers to this questionnaire.

As a reward for the time and effort involved in taking part in this study all participants received £50 after completion of all sessions.

3.2. DESIGN

A mixed design was used in this research. To explore the use of different neurofeedback protocols in cognitive functioning, and analyze if up training SMR had an impact on cognitive functioning, the independent variable was neurofeedback training. This variable had two levels: SMR training and 'sham' training. The participants who underwent real neurofeedback training to increase SMR activity were part of the experimental group. The participants who underwent 'sham' neurofeedback training were played the feedback from a previously recorded neurofeedback session and were part of the control group.

The dependent variables considered were:

- the electrophysiological measures obtained from the pre and post training QEEG assessments, as well as from the neurofeedback sessions;

- cognitive abilities, with a focus on executive functions and memory, assessed by the BADS (Behavioural Assessment of the Dysexecutive Syndrome), Conceptual Span Task and five subtests of the UK adaptation of the Wechsler Memory Scale – Third Edition (WMS - III^{UK}) (Logical Memory I and II, Family Pictures I and II and Letter-Number Sequencing).

3.1. APPARATUS AND PROCEDURE

3.3.1. General Procedure

Participants were recruited through a general email sent to all the students from the Faculty of Science and Technology (see Appendix 1) and also using the Research Participant Panel (see Appendix 2). An email with the information sheet (see Appendix 3) was sent to some of the students who replied. If after reading the information sheet the students were still interested in taking part in the study the first session was scheduled.

This experiment comprised 12 sessions. The first and last sessions were aimed at obtaining the pre and post training measures, respectively. The remaining sessions were neurofeedback training sessions. The pre and post training assessment sessions happened within two weeks before and after the neurofeedback sessions.

In the first session of this experiment, participants were given the opportunity to read the information sheet one more time and clarify any queries about their participation. After this, all participants signed an informed written consent (see Appendix 4) and filled the health related questionnaire. The first session also included the administration of the cognitive tests and the recording of the EEG.

The participants were randomly allocated to 2 groups: the SMR training group (n = 12) and the control group (n = 12). For both groups the neurofeedback sessions were scheduled twice a week, for 5 weeks, in order to have a total of 10 sessions.

In the last session of the study, the same cognitive tests administered in the pre training assessment were administered for all participants along with another EEG recording.

Given the fact that each participant had to come to two sessions of neurofeedback training per week it was important to ensure that enough timeslots were available for their participation. Therefore, the 24 participants had to take part in the study in two different phases in time. A first group of 11 students participated in this study between October and December 2009, the remaining 13 participants took part in the study between January and March 2010.

The different groups being tested were also tested in different settings due to the room availability in the department. For the second group being tested, all the psychometric tests, neurofeedback sessions and EEG recordings took place in the EEG

laboratory. The EEG laboratory includes two rooms. One of the rooms was where the actual recording of the EEG took place, as well as the neurofeedback sessions. This is a sound proof and electrically shielded room. The other room was where the EEG was being monitored during the recording and where the psychometric tests were administered.

For the first group of participants only the EEG recordings took place in the laboratory, the neurofeedback sessions and the psychometric tests were administered in a different room that was not electrically shielded or sound proofed.

Following completion of post training measures all participants were debriefed (see Appendices 6 and 7) and participants from the 'sham' group were offered the chance to have a course of actual neurofeedback training. Only one participant showed interest in doing a course of neurofeedback training, which was carried out using the same protocol used for the experimental group.

3.3.2. QEEG recording

The EEG was recorded using a Brainproducts (Brainproducts GmbH, Munich, Germany) amplifier system. The software used to record the EEG data was Brain Vision Recorder (Brainproducts GmbH, Munich, Germany). Off-line data processing was carried out using the Brain Vision Analyser version 1.05.0005 (Brainproducts GmbH, Munich, Germany) and NeuroGuide Deluxe (version 2.6.3, Applied Neuroscience, Inc.).

The EEG was recorded from 19 electrode sites (FP1, FP2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, Pz) according to the International 10-20 System (see Figure 2) using a Easycap electrode caps (Easycap GmbH, Herrsching, Germany).

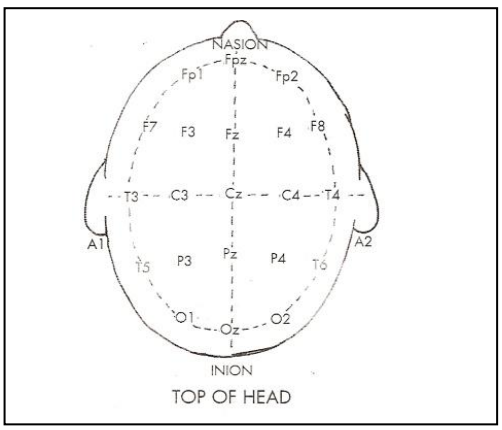

Figure 2 - International 10-20 System (figure originally published in Demos, 2005)

Electro-oculogram (EOG) was recorded using two electrodes placed above and below the left eye and another two electrodes placed about 1 cm beyond the outer canti of the right and left eyes. The EOG enabled the detection of eye blinks and lateral eye movements that contaminated the recording with artifact. All the electrodes were Silver Chloride (AgCl) ring electrodes. The ground electrode was placed on the forehead and the EEG referenced to digitally linked ears. Following the recommendations in the literature, impedance readings were aimed to be kept below 5 kΩ (e.g. Nuwer et al., 1998; Pivik et al., 1993). However, in a few instances, where this was not possible, impedance readings were kept below 10 kΩ, which can be considered an appropriate impedance level when considering the observation that high-quality EEG data can be obtained with impedance below 40 kΩ (Ferree, Luu, Russel, & Tucker, 2001).

EEG was recorded at 250 samples per second, the high band pass filter was 0.1 Hz and the low band pass filter was 70 Hz.

Approximately 30 minutes was spent fitting the electrodes. To make sure that a good connection between the skin and electrodes existed, the skin was cleaned using a cotton bud with an electrolyte gel (Abralyt 2000 chloride free electrolyte). In addition, a small amount of the same gel (which conducts electricity from the scalp to the electrode) was injected through the electrode cap at the electrode sites with a blunt syringe. During the preparation time the participants were familiarized with the procedure and with the equipment.

The EEG was recorded in two resting conditions: eyes open and eyes closed. Each condition was replicated 5 times in an alternating way, each of them lasting approximately 1 minute.

The EEG data was re-referenced to linked ears using the Brain Vision Analyser. The same software corrected EOG artefacts using the Gratton and Coles algorithm (Gratton, Coles & Donchin, 1983). The EEG was also subjected to an automatic artifact rejection procedure in Brain Analyser, which marked EEG segments with an amplitude higher than 100 μv.

After the data had been re-referenced and subjected to ocular correction it was exported to Neuroguide in order to carry out the quantitative analysis. The EEG was filtered to extract data from 1 to 40 Hz and was converted from the time domain to the frequency domain using Fast Fourier Transformation (0.5 Hz resolution). When exporting the data it was noticed that the segments marked as artifact by Brain Analyser

were not marked as such by Neuroguide. Therefore the artifact analysis had to be carried out in Neuroguide.

The first step to artifact the data in Neuroguide was to select at least 10 seconds of artifact free data from the first minute of recording in order to run the automatic drowsiness rejection feature. After this, more artifact free data was selected in order to achieve a template of 60 seconds artifact free data. This template was the basis for the automatic selection carried out by the software. To make sure that the data selected did not include artifacts, every EEG analysed in Neuroguide was compared with the same EEG subjected to an automatic artifact rejection procedure run by the Brain Analyser.

After all the good EEG segments were selected, two separate files were created to include only the eyes open segments or the eyes closed segments. Only the eyes open segments were considered relevant for the analysis because the training was performed with eyes open, therefore any changes in the QEEG were more likely to occur in this condition. A minimum of one minute and a maximum of four minutes of artifact free data were included in the analysis, M = 2.47, SD = 0.66. The split-half reliability ranged from 0.96 and 0.99, and the test-retest reliability ranged from 0.90 to 0.98.

The frequency bands analysed were delta (1- 4 Hz), theta (4-8 Hz), alpha 1 (8- 10 Hz), alpha 2 (10-12 Hz), beta 1 (12-15 Hz), beta 2 (15-18 Hz), beta 3 (18-25 Hz) and high beta (25-30 Hz).

3.3.3 Neuropsychological Assessment

The tests used in this study were administered in the same order for every participant:

1. Logic Memory I (from the WMS – III ^{UK})
2. Family Pictures I (from the WMS – III ^{UK})
3. Letter-Number Sequencing (from the WMS – III ^{UK})
4. Behavioural Assessment of the Dysexecutive Syndrome (BADS)
5. Logic Memory II (from the WMS – III ^{UK})
6. Family Pictures II (from the WMS – III ^{UK})
7. Conceptual Span Task

The time gap between the administration of Logic Memory I and Family Pictures I, and Logic Memory II and Family Pictures II had to be at least 30 minutes. Therefore,

Logic Memory I and Family Pictures I were the first to be administered. These subtests were followed by the Letter-Number Sequencing and the BADS in order to allow 30 minutes to pass before administering Logic Memory II and Family Pictures II. The Conceptual Span Task was the last test to be administered because it was thought to be more engaging.

The standardized procedures proposed in the manuals of each test were followed so as not to reduce the validity of the results. The cognitive tests were aimed at assessing executive functions and memory.

Executive Functions

The assessment of executive functions in this study was made using the BADS. The primary purpose of the BADS is to assess any difficulties that may arise from the Dysexecutive Syndrome (Wilson, Alderman, Burgess, Emslie, & Evans, 1996). This test was created to respond to a problem with the majority of the tests used to access executive functioning, which is only accessing "component skills" of executive functioning (Wilson et al., 1996). Therefore the BADS tries to use a set of tasks that pose a set of problems similar to many everyday activities, thus contributing to the ecological validity of the test (Norris & Tate, 2000; Wilson et al., 1996).

The rationale behind the choice of the BADS is that this is a battery of tests that evaluate several aspects of the executive functions, instead of just focusing in one. Furthermore, individual scores can be obtained from each subtest, which in turn results in a general profile score that measures the level of executive functioning ability as a whole. Furthermore, the tests comprising the BADS have been used extensively in the neuropsychological literature (e.g. Evans, Chua, McKenna, & Wilson, 1997; Hornberger, Piguet, Kipps, & Hodges, 2008; Treitz, Daum, Faustmann, & Haase, 2009; Tyson, Laws, Flowers, Mortimer, & Schulz, 2008; White, Burgess, & Hill, 2009). A brief summary of the six tests comprising the BADS and the procedures for their administration is presented in Appendix 8 .

Memory

For the assessment of memory, five subtests of the WMS - III^{UK} were used along with the Conceptual Span Task.

The WMS - III^{UK} is a battery of tests aimed at measuring memory, working memory and learning (Wechsler, 1997). The WMS –III^{UK} comprises 11 subtests, but for the purposes of this research only five subtests were administered: Logical Memory I and II, Family Pictures I and II and Letter-Number Sequencing.

The WMS - III^{UK} was used in this study because it is a battery that assesses different types of memory, namely immediate memory, delayed memory and working memory, in both visual and auditory modalities, and is a well validated and standardized test (Wechsler, 1997). Even though not all subtests were applied, due to time constraints, the subtests chosen assessed the mentioned memory abilities. In choosing the subtests from the WMS - III^{UK} only the primary subtests were considered. The fact that only these tests contribute to the calculation of the memory indexes of the WMS suggests that they are better measures of the abilities assessed. Among the primary tests only the ones that loaded more on the factor being evaluated were included in the assessment.

Auditory Immediate and Delayed Memory

In order to assess auditory immediate and delayed memory, Logic Memory I and II were used. In Logical Memory I two stories were read to the participant andthe second story was read twice. After listening to each story the participant had to retell from memory the story heard. Logic Memory II was a delayed condition of Logic Memory I that was presented about 30 minutes after Logic Memory I, whereby the participant had to retell the stories heard earlier.

Visual Immediate and Delayed Memory

For the assessment of visual immediate and delayed memory Family Pictures I and II were used. In Family Pictures I the participant was presented with a picture of a family, whose family members were shown doing different activities across four scenes.

After all four scenes were shown the participant was asked to recall who was in each scene, where the characters were and what they were doing. In Family Pictures II the participant had to recall this information, about 30 minutes after the first test was administered, without the scenes being shown again.

Working Memory

Two working memory tasks were chosen for this study: the Conceptual Span Task and the Letter-Number Sequencing subtest from the WMS – III^{UK}.

The Conceptual Span Task used in this study is an adaptation from the Conceptual Span Task developed by Haarman, Davelaar and Usher (2003). This is a category cued-recall test that assesses the semantic short-term component of working memory. The reason for using the Conceptual Span Task is related with the study of Vernon et al. (2003), which found that after neurofeedback training to increase SMR amplitude and inhibit theta and beta activity, there was a significant improvement in semantic working memory performance as measured by this task.

This test is composed of 21 trials. On each trial nine nouns were presented on a computer screen at a rate of one word per second. After the presentation of the last word of each trial, the name of one category appeared (e.g. Animal) and the participants had five seconds to recall aloud the three nouns that belonged to that category. The same 21 trials were administered after the neurofeedback training but they were presented in a different order. The number of correctly recalled words were recorded, along with the number of intrusions (number of incorrect words recalled).

For the Letter-Number Sequencing subtest the participants were presented orally with a string of numbers and letters, which they were asked to repeat, saying the numbers first in ascending order and then the letters in alphabetical order. The rationale for the inclusion of this working memory test is to explore if training to increase SMR activity has a different impact in the performance of tasks that require semantic processing (Conceptual Span Task) compared to tasks that require the mental manipulation of abstract elements (Letter-Number Sequencing). Furthermore both tasks are presented in different modalities – visual and auditory.

3.3.4. Neurofeedback training

Neurofeedback training was administered employing the Nexus-10 Biofeedback system (Mind Media B.V., Netherlands). The software used to digitize the signal and to design the training protocol was Biotrace (Mind Media B.V., Netherlands).

Neurofeedback training was scheduled twice a week for five consecutive weeks for every participant. Nine participants (five participants from the experimental group

and four participants from the control group) did not follow the training schedule strictly, due to health or personal reasons. The maximum amount of time between two sessions was 12 days, which only happened for one participant from the experimental group.

Each session lasted approximately between 50 minutes and one hour: 10 minutes of preparation (cleaning scalp, placing electrodes and setting up equipment), 2 minutes baseline period, 30 minutes of neurofeedback divided in 5 minutes periods separated by approximately 1 minute breaks.

In the first training session the neurofeedback training process and the role of each piece of equipment were explained. The participants were told that the feedback received would reinforce the increase in amplitude of the frequency band trained. Participants were also informed about sources of artifact, such as body movements. During the baseline period participants were instructed to rest with their eyes open.

Participants were seated comfortably in an armchair in front of the feedback monitor. The scalp and mastoids were cleaned using a cotton bud with NuPrep and the conductive paste used on each electrode was Ten20 paste.

A referential montage was used, whereby a reference electrode was attached to the right mastoid and a ground electrode was attached to the left mastoid, the active electrode was placed at Cz, according to the 10-20 system. Referential and bipolar montages have both been used successfully in neurofeedback training (for discussions regarding this matter see Putman, 2001, and Vernon, Frick, & Gruzelier, 2004). The referential montage provides a measure of SMR amplitude at a single site, i.e. Cz, instead of measuring the relationship between different sites, as is the case with the bipolar montage. Therefore, for the purposes of this study a montage that results in unambiguous data was preferred. The ongoing EEG at Cz was band-pass filtered to extract SMR.

Although it is still unclear which criteria to use to set a training threshold, it seems to be important to make sure that enough feedback information is provided and that the threshold is based in the individual EEG resting activity (Vernon et al., 2009). Therefore, in this study, the reward threshold was set for each session according to a 2 minute baseline recording without feedback. After Egner, Zech and Gruzelier (2004) and Ros et al. (2009) the threshold was set at 0.8 times SMR baseline mean amplitude, so the participants could receive feedback approximately 60% of the time. Providing

feedback 60% of the time ensured that the neurofeedback task would not be too easy or too difficult, so as not to demotivate or frustrate participants.

Although the majority of the training protocols that reward increases in SMR amplitude also include the inhibition of theta, this was not included in this study. The reasons for excluding theta from the training protocol are twofold. First, training just one frequency band means that any effects observed are only due to the manipulation of that variable. Second, in previous studies with healthy participants, the inclusion of a component of theta inhibition in the training protocol, although not explained (e.g. Barnea, Rassis, & Zaidel, 2005; Egner & Gruzelier, 2004; Egner, Zech & Gruzelier, 2004; Vernon et al., 2003) seemed to be based on the association between slow wave activity and certain disorders, such as AD/HD (e.g. Bresnahan, Anderson, & Barry, 1999; Chabot, Michele, Prichep, & John, 2001; Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 1998; Clarke et al., 2003; Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995; Lazzaro et al., 1999). Because the sample for this study only included healthy participants this did not seem necessary.

Visual feedback was provided whenever SMR amplitude increased above the threshold. The feedback was provided in the form of a ball moving and a bargraph changing from the colour red to the colour green when the amplitude of SMR surpassed the threshold (see Figure 3). When the amplitude of SMR was below the threshold the ball stopped moving and the bargraph turned red (Figure 4).

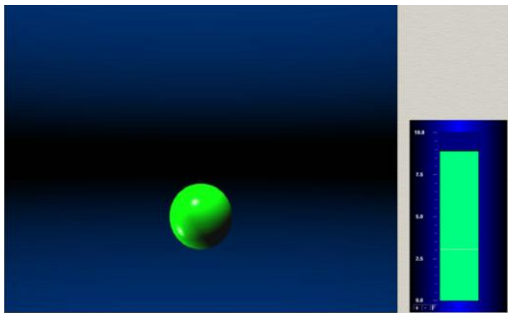

Figure 3 - Feedback provided when SMR is above the threshold

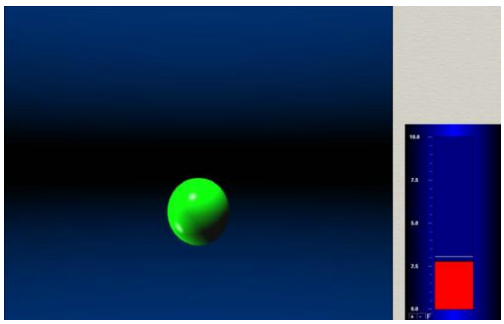

Figure 4 –Feedback provided when SMR is below the threshold

The inclusion of a control group that underwent sham neurofeedback training seemed essential so as to rule out expectations as the cause of any change in the dependent variables. Although the lack of controlled studies in neurofeedback training research is criticized in the literature (Vernon, 2005), there are some claims that the placebo condition is easily recognizable in neurofeedback training (e.g. Kotchoubey et al., 2001). Therefore, after Raymond et al. (2005) participants were asked at the end of each session to what extent they felt they had controlled their brainwaves on a scale from 1 (no control) to 5 (complete control) (see Appendix 9). This question aimed to assess if the sham neurofeedback condition actually worked as a placebo.

None of the participants were aware of the group they belonged to, or that there were experimental and control conditions. They were told that different training protocols would be compared and for that reason they would be randomly assigned to different groups. Gevensleben et al. (2009) suggested that the awareness of the existence of a placebo condition could cause a sense of uncontrollability, loss of motivation and less effort. Therefore, all participants were debriefed about the control condition only after training.

During the sham neurofeedback sessions participants from the control group were played back the sessions of one of the participants from the experimental group, who was chosen randomly. Therefore, the participants in the control group received the same amount of feedback received by the participant in the experimental group on the corresponding session.

The EEG data obtained during the neurofeedback sessions were artifacted, after the sessions, using the Automatic Artifact Rejection feature available in Biotrace. Because scalp EEG activity in the waking adult usually lies below 100μv (Fisch, 1999; Niedermeyer, 2005) the data was rejected when SMR amplitude was above 100μv.

Specific guidelines for setting a threshold for EMG artifact were not found in the literature, therefore different thresholds were tested (10μv, 15μv, 20μv) and the percentage of rejected data was considered. With a 15μv threshold the percentage of data rejected ranged from 0.66% to 3%, M = 1.50, SD = 0.88, when considering 11 participants. A smaller threshold would result in a greater data loss, whereas a higher threshold could possibly include more EMG artifacts than desired. However, it must be noted that the percentage of data rejected fell outside the aforementioned range for one participant (for a detailed explanation see section 4.3.). When considering all participants, the mean percentage of data rejected was 2.50 (SD = 3.56).

3.4. STATISTICAL ANALYSES

All Statistical analyses were performed using the software SPSS 16.0 (SPSS Inc, Chicago, Illinois). Significance level was set to $p \leq 0.05$. When repeated measured ANOVAs were carried out and the Mauchly's test of sphericity was significant, Greenhouse-Geisser correction was used. When main effects were found, pairwise comparisons used Bonferroni adjustment. The specific statistical methods employed in this study will be described in detail in the next chapter.

CHAPTER FOUR
RESULTS

4.1. DIFFERENCES BETWEEN GROUPS IN THE PRE TRAINING PERIOD

In order to detect if the groups were significantly different before the neurofeedback training in terms of age, unrelated t-tests were performed.

To test whether there were significant numbers of females or males in each group a Fisher exact test was used. Although the chi-square procedure was used in SPSS, the Fisher exact test was computed because there was at least one cell with an expected frequency of less than 5.

The groups were not significantly different in terms of age (t = -.79, df = 22, two tailed p = .44) or gender (two-tailed Fish exact p = .68).

4.2. PERCEPTION OF CONTROL OF BRAINWAVES

In order to test the hypothesis that the perception of control of the EEG would not differ significantly between the groups , the answers to the question "How well do you feel you have been able to control your brainwaves?" were used to compare them (see Appendix 9). The mean scores of the two groups were compared using a Mann-Whitney *U*-test.

The mean perception of control for the experimental group was 3.16 and the mean perception of control for the control group was 3.23. It was found that the scores of perception of control from the two groups were not significantly different (z = 0.0001, two-tailed p = 1.00).

4.3. SELF-REGULATION OF SMR ACROSS AND WITHIN SESSIONS

As the control group did not go through an actual course of neurofeedback sessions, statistical analyses for the data concerning the neurofeedback sessions were carried out only for the experimental group.

To test the hypothesis that the neurofeedback task had been learned (see 2.6.), indices of learning included measures of amplitude and percent time above threshold within and across sessions along with comparisons to baseline. Dempster and Vernon

(2009) considered that comparing amplitude and percent time above threshold with baseline was more relevant than measures of amplitude and percent time above threshold alone because it enables the evaluation of changes that occur during training, which exceed the natural levels produced during baseline. However, in the absence of more research addressing this issue, it was considered important to be inclusive in this study and incorporate several indices of learning.

Because it was noted by Dempster and Vernon (2009) that any changes across sessions, as a result of neurofeedback training, might be confounded by increasing baselines across sessions, comparisons between SMR amplitude and percent time above threshold and baseline in session 1 were also made.

It is important to note that one participant exhibited particularly high amplitudes (exceeding 100 μv) across the whole EEG spectrum in some neurofeedback sessions. In session 2 this was worse, and even though all the standard procedures were followed (checking leads, changing the electrodes, checking for other electronic devices that might interfere with recording, switching off mobile phones and advising the participant to stay relaxed) the amplitudes remained high. For this reason, it was decided to stop the session early, as the feedback was not reflecting increases in the amplitude of SMR but instead increasing amplitudes in the whole spectrum. Therefore, for session 2 there was a participant who had 10 minutes less neurofeedback training than the other participants. Because the values missing for this participant were part of variables that were averaged into new variables (for example the mean SMR amplitude in session 2 segment 6 was used to create a variable that averages SMR amplitude across all sessions in segment 6) it was decided not to substitute them using estimation methods, which in turn, might have resulted in biased data.

As it was suspected that for this participant the percentage of data rejected in each session would be higher than for the other participants, box plots were made for each session revealing extreme values (over 3 times the interquartile range) for sessions 1, 2 and 10 and one outlier for session 8 (value over 1.5 times the interquartile range). All of these outliers corresponded to the same participant that exhibited high amplitudes across the EEG. For this reason, statistical analyses on the neurofeedback sessions data were carried out with and without data from this particular participant. Both analyses were only reported when different results were obtained.

4.3.1. Indices of learning across sessions

Changes in SMR amplitude and percent time above threshold across the neurofeedback sessions were analyzed using a one-way repeated measures ANOVA, with "Session" as a factor with ten levels. In order to compare SMR amplitude and percent time above threshold during the training phase to amplitude and percent time above threshold during the baseline phase, a 2 (baseline versus training) x 10 (neurofeedback sessions), phase by session two-way repeated measures ANOVA was used.

To compare SMR amplitude and percent time above threshold across sessions with baseline in session one, ten new computed variables were calculated, subtracting each measure during baseline from the same measure during the neurofeedback training sessions (e.g. SMR amplitude session 5 – SMR amplitude baseline session 1) and one - way repeated measures ANOVAs were conducted on this 'difference' score.

SMR amplitude

The one-way repeated measures ANOVA did not reveal a main effect of Session, F (9, 99) = 0.79, p = 0.627, partial η^2 = 0.067.

SMR amplitude across sessions compared to baseline

A 2 (baseline versus training) x 10 (neurofeedback sessions), Phase by Session two-way repeated measures ANOVA did not reveal a significant main effect of phase, F (1, 11) = 0.15, p = .70, η^2 = .01, nor a significant main effect of training session, F (9,99) = 1.13, p = .35, η^2 = .09. Moreover, the phase by session interaction was also non significant, F (3.70, 40.70) = 0.93, p = .45, partial η^2 = .08.

SMR amplitude across sessions compared to baseline in session 1

A one-way repeated measures ANOVA performed on the computed variables (amplitude during training – amplitude during baseline) did not show a main effect of Session, F (9, 99) = 0.79, p = .63, partial η^2 = .07.

Percent time above threshold

A repeated measures ANOVA did not reveal a main effect of Session, F (9, 99) = 0.979, p = .462, partial η^2 = 0.082.

Percent time above threshold across sessions compared to baseline

A 2 (baseline versus training) x 10 (neurofeedback sessions), Phase by Session two-way repeated measures ANOVA did not show a significant effect of Phase (F [1, 11] = 0.32, p = .58, η^2 = .03) nor a significant effect of training Session (F [9, 99] = 1.17, p = .32, η^2 = .10). There was not a significant Phase by Session interaction, F (4.46, 49.04) = 1.03, p = .41, partial η^2 = .09.

Percent time above threshold across sessions compared to baseline in session 1

A repeated measures ANOVA did not reveal a main effect of Session, F (9, 99) = 0.98, p = .46, partial η^2 = .08.

4.3.2. Indices of learning within sessions

For the purposes of the within session analyses the training sessions were divided into 6 five minutes periods. A division in shorter periods of time, such as 30 one minute periods, would have resulted in less loss of information. However, this could be problematic when performing ANOVAS because it would lead to insufficient degrees of freedom due to a higher number of factors (30) than cases (n = 12).

One-way repeated measures ANOVAs were conducted to analyse the amplitude of SMR and the percent time above threshold across the training periods.

To compare the measures during the training period to baseline, new variables were computed, subtracting mean amplitude or percent time above threshold during baseline to its mean during training. One-way repeated measures ANOVAs were conducted on the computed variables.

SMR amplitude across 6 five minute periods

A marginally significant main effect of training period was found, F (5, 55) = 2.34, p = .05, partial η^2 = .18. Pairwise comparisons showed that the amplitude of SMR was not significantly different between segments. When the same analysis was conducted without the case considered an outlier, there was not a significant main effect of period, F (5, 50) = 2.01, p = .09, partial η^2 = .17.

SMR amplitude within sessions (6 five minute periods) compared to baseline

A marginally significant main effect of period (F [5, 55] = 2.32, p = .06, partial η^2 = .17) was found. Pairwise comparisons did not reveal significant differences between the computed scores.

When the same analysis was conducted without the outlier there was no significant main effect of period F (5, 50) = 2.01, p = .09, partial η^2 = .17.

Percent time above threshold across 6 five minute periods

A one-way repeated measures ANOVA did not reveal a main effect of training period, F (5, 55) = 1.83, p = .12, partial η^2 = .14.

Percent time above threshold within sessions (6 five minute periods) compared to baseline

No main effect of training period was found F (5, 55) = 1.86, p = .12, partial η^2 = .15.

4.4. INFLUENCE OF BASELINE AMPLITUDE IN THE ABILITY TO CONTROL SMR

To determine if SMR baseline power recorded during the QEEG pre training assessment and SMR baseline amplitude at the beginning of the sessions was associated with the ability to control SMR, Pearson's correlations were carried out between these variables and the following indices of learning: mean percent time above threshold,

mean difference between time spent above threshold during training and during baseline and mean difference between SMR amplitude during training and baseline. Given the fact that a negative relationship between the variables was hypothesized (see 2.6.), one-tailed *p* values were used.

The correlation between baseline during neurofeedback training and mean SMR amplitude during training was positive and highly significant (r = .93, p < .01). This result was expected, given that no significant differences were found between training and baseline periods.

When correlating neurofeedback training sessions' baseline amplitude and indices of learning, individual analyses were also performed so as to evaluate for each participant the influence of these variables across the course of training.

4.4.1. Neurofeedback sessions' SMR baseline amplitude

When analysing the influence of SMR baseline levels on indices of learning for the experimental group as a whole, new computed variables were created that averaged these variables across sessions. Therefore, the mean baseline across sessions was correlated with the mean percent time above threshold, the mean difference between percent time above threshold during training and during baseline, as well as with the mean difference between SMR amplitude during training and during baseline. The correlations for each pair of variables considered (mean baseline across sessions and each index of learning) will be presented next, along with the respective individual analysis. All individual analyses are presented on table 1.

In the group analyses, a significant correlation was found between mean baseline amplitude across sessions and mean percent time above threshold, r = -.56, one-tailed p = .03. Individual analyses revealed that this relationship was negative for all participants but only significant for 6 (see table 1), suggesting that the higher the baseline less time is spent above threshold.

In the group analyses, the results showed a negative non significant relationship between mean baseline and mean difference between percent time above threshold and baseline, r = -.419, one tailed p = .09. The individual analyses showed that for all participants, except two, this relationship was negative, which indicates that for 10 participants the higher the baseline, the less time spent above threshold during training

compared to baseline. However, this relationship was significant for only five participants (see table 1).

Also, a negative significant correlation was found between mean baseline and mean difference between amplitude of SMR during training and during baseline, in the group analyses, r = -.524, one-tailed p = .04. Individual analyses showed that this correlation was negative for all participants, revealing that the higher the baseline the smaller the increase in amplitude between baseline and training periods. Correlations reached significance for six participants (see table 1).

Table 1

Pearson's correlations between baseline amplitude during neurofeedback training sessions and indices of learning

	% time		***% time_baseline***		***MeanAmp_baseline***	
Participants	*R*	*p*	*R*	*P*	*r*	*p*
A	-.21	.30	.30	.21	-.17	.32
B	-.37	.15	.09	.40	-.40	.13
C	-.65*	.02	-.75**	<.01	-.57*	.05
D	-.86**	<.01	-.85**	.01	-.86**	<.01
E	-.86**	<.01	-.92**	<.01	-.89**	<.01
F	-.71*	.01	-.38	.14	-.73**	.01
G	-.64*	.03	-.65*	.02	-.60*	.04
H	-.22	.27	-.22	.30	-.32	.18
I	-.89**	<.01	-.87**	<.01	-.89**	<.01
J	-.52	.07	-.51	.07	-.44	.10
K	-.30	.20	-.34	.17	-.18	.31
L	-.47	.09	-.24	.26	-.42	.12

r *–Pearson correlation coefficient,* ***p*** *– p-value of significance,* ***% time*** *– percent time above threshold,* ***%time_baseline*** *– difference between percent time during training and baseline periods,* ***MeanAmp_Baseline*** *– difference between SMRamplitude during training and baseline*

******* *correlation significant at the 0.05 level (one-tailed)*

******** *correlation significant at the 0.01 level (one-tailed)*

4.4.2. QEEG SMR pre training power

The relationship between SMR absolute power at Cz at the pre training period with mean percent time above threshold was negative and significant, r = -.55, one-tailed p = .03. This indicates that higher SMR power before training was associated with less time above threshold.

The same pattern of results was found for the correlation between SMR absolute power with the mean difference between percent time above threshold during training and during baseline (r = -.53, one-tailed p = .037), as well as with mean difference in SMR amplitude between training and baseline (r = -.59, p = .02). These results suggest that when SMR absolute power is higher at the pre training assessment there is a smaller increase in amplitude or percent time above threshold from baseline to training periods.

No significant correlations were found between SMR relative power at Cz and the aforementioned indices of learning (one-tailed $p < .05$).

4.5. CHANGES IN THE QEEG

Although the neurofeedback training goal in this study was to increase SMR amplitude only, it was important to investigate any changes in other frequency bands, as previous research has suggested EEG changes after neurofeedback training not related with the training contingencies (Egner et al., 2004). Therefore, differences between groups in terms of absolute and relative power from the pre-training to the post-training period were investigated by 2 (pre-training versus post training) x 2 (experimental versus control group) x 6 (electrode groups), Time x Group x Electrode Group three-way mixed ANOVAs for each frequency band. Power values were averaged over groups of electrodes, corresponding to the prefrontal (FP1, FP2, F7 and F8), frontal (F3, FZ and F4), central (C3, CZ and C4), parietal (P3, PZ and P4), occipital (O1 and O2) and temporal (T3, T4, T5 and T6) areas of the brain.

Delta

There was not a significant Time x Group x Electrode Group interaction, either considering absolute power F (1.29, 28.30) = 0.34, p = .62, partial η^2 = 0.02, or considering relative power, F (2.09, 46.02) = 0.55, p = .59, partial η^2 = 0.03.

Theta

When considering theta absolute power, it was not found a significant Time x Group x Electrode Group interaction, F (1.22, 26.73) = 0.45, p = .55, partial η^2 = 0.02. The three-way interaction is also not significant for theta relative power, F (2.21, 48.59) = 0.57, p = .58, partial η^2 = 0.03.

High Beta

There was not a significant Time x Group x Electrode Group interaction, either considering absolute power F (1.12, 24.61) = 1.09, p = .31, partial η^2 = 0.05, or considering relative power, F (1.67, 36.65) = 0.83, p = .42, partial η^2 = 0.04

Alpha 1

The analysis of variance did not reveal a significant Time x Group x Electrode Group interaction, when considering absolute power F (2.28, 50.22) = 0.51, p = .63, partial η^2 = 0.02, or when considering relative power, F (2.00, 43.99) = 0.40, p = .67, partial η^2 = 0.02.

Alpha 2

There was not a significant Time x Group x Electrode Group interaction, for alpha 2 absolute power F (1.55, 33.99) = 1.15, p = .32, partial η^2 = 0.05, or alpha 2 relative power, F (1.83, 40.18) = 1.83, p = .18, partial η^2 = 0.08.

Beta 1

It was not found a significant Time x Group x Electrode Group interaction, either considering absolute power F (1.49, 32.81) = 0.26, p = .71, partial η^2 = 0.01, or relative power, F (2.62, 57.57) = 0.83, p = .47, partial η^2 = 0.04.

Beta 2

For beta 2 absolute power a significant Time x Group x Electrode Group interaction was not found, F (1.65, 36.20) = 1.29, p = .28, partial η^2 = 0.06. Also, for beta 2 relative power a three-way interaction was not found, F (1.91, 42.10) = 0.84, p = .44, partial η^2 = 0.04.

Beta 3

There was not a significant Time x Group x Electrode Group interaction, either considering absolute power F (1.27, 27.92) = 0.90, p = .38, partial η^2 = 0.04, or considering relative power, F (2.14, 47.15) = 1.12, p = .34, partial η^2 = 0.05.

4.6. CHANGES IN COGNITION

The mean scores for all the cognitive tests administered at the pre training and post training periods for both groups are presented in table 2.

To test the hypothesis that a higher degree of improvement in the scores from the cognitive tests would be observed in the experimental group (see 2.6.), a 2 (pre training versus post training) x 2 (experimental versus control group), Time by Group two-way mixed ANOVA was employed for each cognitive test. The results are presented next.

Logic Memory I

There was not a significant Time by Group interaction, F (1, 22) = 0.03, p = .86, partial η^2 = 0.001.

Logic Memory II

The analysis of variance did not reveal a significant Time by Group interaction, F (1, 22) = 0.01, p = .94, partial η^2 < 0.001.

Family Picture I

It was not found a significant Time by Group interaction, F (1, 22) = 2.76, p = .11, partial η^2 = 0.11.

Family Pictures II

There was not a significant Time by Group interaction, F (1, 22) = 1.13, p = .30, partial η^2 = 0.05.

Letter-Number Sequencing

A significant Time by Group interaction was not found, F (1, 22) = 0.23, p = .64, partial η^2 = 0.01.

Rule Shift Cards (BADS)

The analysis of variance did not reveal a significant Time by Group interaction, F (1, 22) < 0.001, p = 1.00, partial η^2 < 0.001.

Action Program (BADS)

Both groups presented the same mean scores in the pre and post training periods, therefore there was no need to perform an analysis of variance.

Key Search (BADS)

There was not a significant Time by Group interaction, F (1, 22) = 2.57, p = .12, partial η^2 = 0.11.

Temporal Judgement (BADS)

It was not found a significant Time by Group interaction, F (1, 22) = 0.15, p = .71, partial η^2 = 0.01.

Zoo Map (BADS)

There was not a significant Time by Group interaction, F (1, 22) = 0.16, p = .70, partial η^2 = 0.01.

Modified Six Elements (BADS)

The analysis of variance did not reveal a significant Time by Group interaction, F (1, 22) = 0.36, p = .56, partial η^2 = 0.02.

BADS – Total Score

A significant Time by Group interaction was not found, F (1, 22) = 0.10, p = .76, partial η^2 = 0.004.

Conceptual Span Task – words correctly recalled

There was not a significant Time by Group interaction, F (1, 22) = 0.03, p = .86, partial η^2 = 0.001.

Conceptual Span Task - intrusions

There was not a significant Time by Group interaction, F (1, 22) = 1.09, p = .31, partial η^2 = 0.05.

Table 2

Scores for cognitive tests at the pre and post training periods for the experimental and control groups

	Experimental group		Control Group	
	Pre training (*M*, *SD*)	Post training (*M*, *SD*)	Pre training (*M*, *SD*)	Post training (M, *SD*)
Logical Memory I	9.17(0.84)	11.83 (0.72)	9.25 (0.84)	11.75 (0.72)
Logical Memory II	9.67 (3.28)	12.58 (2.64)	10.08 (2.64)	13.08 (2.31)
Family Pictures I	8.42 (2.27)	10.50 (2.94)	8.25 (2.14)	11.75 (2.30)
Family Pictures II	7.83 (2.41)	10.50 (2.97)	8.17 (1.70)	11.91 (2.47)
Letter-Number Sequencing	10.75 (2.42)	11.00 (3.28)	9.75 (3.05)	10.42 (3.12)
BADS	97.75 (16.13)	104.5 (10.09)	101.25 (8.25)	106.83 (5.54)
Conceptual Span Task –words correctly recalled	33.17 (5.62)	34.92 (7.39)	29.25 (4.86)	31.58 (6.97)
Conceptual Span Task – intrusions	11.75 (8.14)	11.75 (5.15)	7.33 (5.96)	9.92 (8.32)

4.7. DIFFERENCES IN TESTING SETTING

As already pointed out in the "General Procedure" (section 3.1.), the participants were tested at two different points in time (a first group of 11 participants took part in the study between October and December 2009 and a second group of 13 participants took part in the study between January and March 2010), which also meant different physical settings (due to the rooms availability) for the cognitive assessments and neurofeedback sessions. To rule out the influence of the physical setting in the participants' performance, the groups were compared in terms of their scores in the cognitive tests and in terms of their SMR amplitude and percent time above threshold in the neurofeedback sessions.

To test for differences in the cognitive tests, 2 (pre-training versus post-training) x 2 (first group or second group tested), Time by Group two-way mixed ANOVAs were carried out. Both groups tested at different points in time were constituted by participants from the experimental and the control group. The 2 (pre-training versus post-training) x 2 (first group or second group), Time by Group two-way mixed ANOVA revealed that for the Key Search subtest of the BADS there was a significant interaction time by group, F (1, 22) = 6.637, p = 0.017, partial η^2 = 0.232. Independent t-tests revealed that the mean score in the Key Search subtest was significantly higher only at pre training for the second group being tested (M = 3.39, SD = 1.04) than for the first group (M = 2.36, SD = 1.29), t = -2.15, df = 22, p = 0.043. This result might indicate that for the second group being tested, the environmental setting might have benefited the scores in the Key Search subtest before the neurofeedback training.

Differences in SMR amplitude and percent time above threshold across sessions were tested using 2 (first group being tested versus second group being tested) x 10 (neurofeedback sessions), group by session two way mixed ANOVAs.

2 (first group being tested versus second group being tested) x 2 (baseline versus training) x 10 (neurofeedback sessions) three way mixed ANOVAs were used to compare the groups in terms of SMR amplitude and percent time above threshold compared to baseline across sessions.

For the within sessions analysis 2 (first group being tested versus second group being tested) x 6 (training periods) two way mixed ANOVAs were used to analyse the amplitude of SMR and the percent time above threshold across the training periods. The

same method was used to compare the computed variables that subtracted mean amplitude or percent time above threshold during baseline to its mean during training.

No significant differences between the groups were detected in terms of SMR amplitude and percent time above threshold across and within sessions.

CHAPTER FIVE
DISCUSSION

The present study investigated whether neurofeedback training to enhance SMR could improve cognitive functioning in healthy people, specifically executive functioning and memory. It was also a goal of this investigation to determine if EEG changes, reflecting the training contingencies, could be observed during and after the neurofeedback training course. Furthermore, the role of baseline SMR levels in the ability to learn SMR was explored.

The results did not support the hypothesis that the experimental group would show electrophysiological changes according to the training protocol. Moreover, the hypothesis that the experimental group would show an increase in cognitive performance, relative to the control group, after neurofeedback training was not supported. However, the results did suggest that SMR baseline levels influenced subsequent ability to self-regulate SMR.

5.1. USING PLACEBO CONDITIONS IN NEUROFEEDBACK TRAINING STUDIES

The results of this study support the prediction that it is possible to use a placebo condition in neurofeedback training research without being recognized by participants, as shown by the lack of significant differences between groups in the perception of control of brainwaves. This result is similar to previous research using sham neurofeedback (e.g. Raymond et al., 2005).

The inclusion of placebo groups in neurofeedback training studies seems to be an important condition in order to rule out unspecific effects. Given that ethical implications are at stake when using placebo conditions with clinical populations when other treatments exist (e.g. La Vaque & Rossiter, 2001), it is relevant to include sham feedback when studying neurofeedback training with healthy participants.

5.2. LEARNING TO MODIFY BRAIN ACTIVITY DURING TRAINING

No evidence of learning was detected when using indices of learning that evaluated changes in amplitude of SMR and percent time above threshold, within and across sessions and compared to a resting baseline period.

These results are not consistent with previous studies showing the ability to control SMR during a neurofeedback training course, not only in people with diagnosed disorders but also in healthy people (e.g. Egner et al., 2004; Vernon et al., 2003).

It is still unclear which methodological choices, regarding protocols, enhance the ability to learn to control EEG parameters (for reviews, Vernon et al., 2009; Vernon et al., 2004). These methodological choices include type of feedback, threshold, number, duration and frequency of sessions, and frequency band rewarded.

It is possible that the number and frequency of sessions of this study was not sufficient to facilitate learning. Previous studies using a similar training schedule to the one employed in this study showed mixed results, with research showing EEG changes following training (e.g. Egner et al., 2004), and other studies demonstrating a lack of cognitive and EEG changes (e.g. Logemann, Lansbergen, Van Os, Böcker, & Kenemans, 2010). However, there are methodological differences between studies which make any comparisons difficult. For example, in the study by Logemann et al. (2010), a double-blind design and individualized protocols were used, whereas in the study by Egner et al. (2004) standardized protocols were employed. Therefore, only future research can determine the training schedule that facilitates self-regulation of brain activity.

In terms of the type of feedback used, two sources of visual information (bargraph changing colour and ball moving) were shown. Whether participants could have benefited from an auditory source of feedback is debatable. Vernon et al. (2009) suggest that providing information in two different sensorial modalities may benefit participants whose attention to one modality decreases. However, it is also noted that Breteler, Manolova, Wilde, Caris and Fowler (2008) did not find a beneficial effect of auditory and visual feedback over visual feedback alone when up training SMR (12-15 Hz).

As mentioned by Breteler et al. (2008) a possible reason for thinking that auditory feedback would be of benefit is that auditory feedback can be of the discrete type, whereas visual feedback is continuous. According to Sterman and Egner (2006) the incorporation of rewards in discrete trials can lead to successful operant learning. In this study, no discrete rewards were given to participants, like points, for example. A reward system based on points could have been more useful and the information more meaningful to participants.

The study by Breteler et al. (2008) also concluded that consonant sounds (which are usually considered more pleasant) had a positive impact on neurofeedback training

compared to dissonant sounds. These results are in line with previous investigations demonstrating that the human brain shows a tendency to prefer certain stimuli instead of others. It has even been suggested that EEG can be modified in order to elicit external stimuli that are preferred by the brain or more comfortable for the brain (Kaplan, Kildani, Minikes, & Bandler, 2008; Kaplan, Lim, Jin, Park, & Byeon, 2005). Kaplan et al. (2005) showed that in spite of the lack of instructions and of any explanation regarding the neurofeedback loop, the participants could control their brain activity in the direction that reinforced their colour preferences. These studies imply that the stimuli used for feedback are important influences that can mediate learning.

It is also plausible that the lack of learning to self regulate brain activity in the experimental group might be related with the lack of attention to the feedback information during training. Meichenbaum (1976) notes that during training it is not possible to know whether the participant is actually focused on the feedback information or if the cognitions produced are irrelevant to the training. Therefore, it is reasonable to question if using task relevant statements and imagery would be useful in directing the participant's attention to the task at hand (Meichenbaum, 1976).

In fact, it is accepted that learning, classical conditioning and operant conditioning included, is a process influenced by cognitions (e.g. Kirsch, Lynn, Vigorito, & Miller, 2004). For this reason, not only the cognitions that the participants have during training can influence the results, but the cognitions that precede training: the expectations about training, the expectations about the ability to control physiological responses and the conceptualization of the participants' difficulties (Meichenbaum, 1976). Regarding the last point, it is worth noting that training, in this study, was not targeting any specific difficulties or areas that were important for the participants to improve. Although the possibility of cognitive improvement was mentioned before training, this was presented as an exploratory study and no expectations of improvement regarding specific cognitive abilities were given. This lack of specificity in the instructions had the goal of not influencing the participants' expectations and consequently the results. However, the fact that the training contingencies and goals were not tailored to the participants' individual needs may have led to a lack of motivation and a decrease in attention during training. In an overview of the factors that might influence self-regulation failure, Baumeister and Heatherton (1996) mention the importance of considering the long-term consequences and goals of self-regulation. The participants of this study probably did not have a specific motivation or long-term goal that could facilitate learning.

5.3. THE MEDIATING EFFECT OF SMR BASELINE AMPLITUDE IN THE ABILITY TO LEARN THE NEUROFEEDBACK TASK

The results obtained support the hypothesis that a lower baseline SMR power (measured during the QEEG assessment) or amplitude (measured across the 10 neurofeedback sessions) facilitated SMR control. This was evidenced by the fact that the lower the baseline during neurofeedback sessions more time was spent above threshold and a greater improvement in amplitude was observed from baseline to training periods. Furthermore, absolute SMR power (measured through the QEEG) was also negatively correlated with the mean difference in percent time spent above threshold from baseline to training periods. It is important to note that in the individual analyses, correlations between baseline amplitude during training and indices of learning did not reach significance for all participants.

These results are consistent with the research reviewed in section 2.5. regarding the mediating effect of baseline levels of cognitive performance or electrophysiological variables on the improvements that can be gained after a performance enhancement intervention. Given the literature in this area, it would also make sense to investigate directly the mediating effects of baseline cognitive performance on the degree of cognitive change that can be achieved after training. However, this question was not addressed in the data analysis as no differences in the degree of improvement between the groups were found. Nevertheless, the mediating effect of baseline levels on the control of EEG variables suggests that a similar relationship might be found for cognitive performance, which is likely to be reflected on electrophysiological functioning (e.g. Klimesch, 1999).

These findings suggest that neurofeedback training in healthy people might be more useful for people functioning at the lower end of the spectrum, in terms of the amplitude of the frequency band to be reinforced. This further suggests that future studies should address this issue by pre selecting the sample of the experiment on the basis of baseline levels of healthy people. This means that healthy people with lower baseline levels should be compared with healthy people with higher baseline levels in terms of their ability to increase the amplitude of the desired frequency band. Future research could address whether baseline amplitude has a predictive value in determining successful self-regulation of brain activity.

In addition, the fact that the ability to learn to control SMR might be dependent on baseline amplitude across sessions might have important implications in terms of setting thresholds. The individual analyses revealed that, for six participants, baseline amplitudes across sessions were negatively correlated with the amount of time above threshold and with the increase in SMR amplitude from baseline to training periods. Thresholds are usually decided according to baseline levels so as to adjust the degree of difficulty to the natural levels shown by the individual before each training session. However, given that the sessions with higher baselines result in less time above the threshold and less increase in SMR amplitude from baseline to training periods, the present results suggest that adjusting the threshold level according to a higher baseline, for example, might increase the difficulty in maintaining SMR amplitude above threshold.

The fact that individual differences in baseline SMR amplitude or power influence neurofeedback training suggests that individualized training protocols that consider these differences might increase successful self-regulation of brain activity. Hammond (2010) argues that individualized protocols based on QEEG assessments (i.e. comparing QEEG parameters with normative databases) are more likely to be successful in the treatment of clinical disorders than standardized protocols that do not rely on objective individual data. In reality, improvements in clinical populations after employing individualized protocols have been demonstrated (e.g. Breteler, Arns, Peters, Giepmans, & Verhoeven, 2010; Walker, 2011; Walker, Norman, & Weber, 2002), and the superiority of individualized compared with standardized protocols has also been observed (e.g. Coben & Myers, 2010). However, individualized protocols aimed at improving performance in healthy people have not resulted in changes in cognitive and behavioural measures (e.g. Logemann et al., 2010). Therefore, more studies with larger sample sizes, control groups and more outcome measures are warranted to determine the efficacy of individualized as compared to standardized protocols.

5.4. EEG CHANGES AFTER NEUROFEEDBACK TRAINING

The results do not support the hypothesis that SMR power would increase significantly from the pre to the post training phase for the experimental group. Moreover, no significant changes in EEG power were found for any frequency bands analysed, either in the control group or in the experimental group.

Given that the experimental group did not show any evidence of having learned to control SMR during training, it was not expected to find any EEG changes after the training.

It is also possible that any EEG changes, after a course of neurofeedback training, might be more likely to occur when performing a task. Egner et al. (2004) did not find EEG changes after neurofeedback training to increase SMR. This finding was interpreted as suggesting that neurofeedback training did not lead necessarily to changes in EEG during resting periods and perhaps an increased SMR activity would be observed during active inhibition of sensorimotor processing (during the performance of a task). Arns et al. (2009) also suggest that changes in the EEG should be studied during the performance of tasks instead of resting eyes open or eyes closed conditions due to the stability of the EEG over time. This suggestion is also supported by the observation that only EEG reactivity to certain stimuli has changed after neurofeedback training but not the resting EEG (Kropotov et al., 2007).

5.5. THE EFFECT OF NEUROFEEDBACK TRAINING ON COGNITION

One of the main goals of this research was to explore the effect of neurofeedback on cognition in healthy people. Contrary to the prediction that neurofeedback training would improve memory and executive functions, the current study did not detect significant changes in cognition in the experimental group.

The most obvious reason for a lack of cognitive improvement in the experimental group is the absence of any evidence of learning to self-regulate SMR.

It seems unlikely that these results are due to the fact that the cognitive skills studied are not able to be enhanced. In chapter two it was mentioned that medication has been used to improve cognition, namely executive functions and memory. It was also shown that previous studies suggest that changes in cognitive abilities can occur after neurofeedback training, executive functioning and memory included.

Another explanation is related with the possibility that the specific tests used in this study might not have been sensitive to changes possibly caused by this particular protocol. In fact, the BADS is a measure designed for people with executive deficits, therefore it might not be sensitive to changes that might occur in healthy people.

Also, the fact that the sample was composed of healthy individuals only, could mean that their executive and memory abilities might have not left any space for

improvement. White (1998), for example, claims that improvements in memory should not be expected in young healthy individuals functioning close to their maximum level. This probably indicates that, in future research with healthy participants, it may be appropriate to carry out a preliminary assessment on potential participants and only include in the study the ones who have the lowest scores in the cognitive assessment, so as to enable room for improvement, as already done in other investigations (e.g. Logemann et al., 2010). Alternatively, it has also been suggested that cognitive enhancement in healthy participants might be dependent on higher levels of task difficulty, in that tasks that are too easy might not leave the possibility of cognitive improvement, whereas increased task-difficulty enhances the probability of observing cognitive improvement in healthy individuals (Korol, 2002). Therefore, increasing the degree of difficulty of the tasks might also leave more room for improvement.

The results obtained with the Conceptual Span Task are particularly surprising, as this test had already been used in a neurofeedback study to increase SMR in healthy participants and a clear improvement in performance was observed (Vernon et al., 2003). Nonetheless, the present study failed to replicate these findings. However, in the study by Vernon et al. (2003) there was evidence of learning to control SMR amplitude within the sessions, which did not happen in the present study.

5.6. LIMITATIONS OF THE STUDY

There are several limitations in this study that can influence the results obtained.

First of all, not all participants followed the training schedule (two sessions a week) strictly. Therefore, different participants were subjected to different training conditions and this introduces an unwanted source of variance. The different environmental conditions (two different rooms were used for testing) are another source of variance, already mentioned in chapter 3. However, data analysis does not suggest that this factor has had any impact on the results, as no significant differences were found between the participants who were tested in one room compared to the participants that were tested in the other room.

Another limitation of this study is the small sample used. Considering an effect size of 0.50 and the usually recommended power of 0.80, the ideal sample size needed would have been 64 participants. However, due to the time consuming nature of

neurofeedback training, this was an impossible endeavour to take within the time course of this research.

This study is also limited by the absence of any EEG recording from the control group during the neurofeedback sessions. It would have been useful if the EEG resting baselines of the control group from the ten neurofeedback sessions had been recorded, as it would have been possible to compare EEG variables during training between groups.

This study would have benefited from a double blind placebo design. However, this would require more than one researcher, which was not possible to accomplish due to the fact that this research was performed within an MPhil course. Although it has been suggested that a double blind sham controlled design might decrease the effectiveness of neurofeedback training (Logeman et al., 2010), this issue needs to be further explored.

In addition and, as already mentioned throughout this thesis, there are several methodological choices that possibly influence the results, such as the frequency, duration and amount of sessions, the frequency band rewarded or the type of feedback information. These methodological choices are limited by the lack of specific guidelines in the literature indicating what works best and for whom.

5.7. CONCLUSIONS AND FUTURE DIRECTIONS

This study indicates that there were no QEEG changes in the experimental group after a course of neurofeedback training and no evidence of any enhancement in cognition compared to the control group. These results agree with the lack of any evidence that the experimental group learned to control SMR.

The absence of evidence that participants learned to control SMR might be related with specific aspects, such as frequency of training, duration of training, type of feedback provided, just to mention a few. The evaluation of whether neurofeedback training is a good way to promote cognitive enhancement can only be made when more research is available about aspects of the training protocol that facilitate learning the neurofeedback task. These questions have been addressed in the literature (e.g. Vernon et al., 2009; Vernon et al., 2004) but more studies and guidelines are needed.

Another key finding from this study is that it is possible to use sham neurofeedback training without it being detected by participants. This means that

neurofeedback research can benefit from the use of rigorous research designs that rule out unspecific effects. Future research could even include double blind placebo designs.

The possible dependence of self-regulation of SMR on SMR baseline levels indicates that there are individual electrophysiological characteristics that influence the ability to learn the neurofeedback task. Exploring the role that natural levels of electrophysiological activity have in controlling EEG variables could clarify for whom neurofeedback training works best and whether baseline amplitudes are predictive of successful neurofeedback training.

Furthermore, the fact that learning to control SMR is dependent upon baseline raises the question of whether in healthy people it is more difficult to achieve control over electrophysiological variables whose natural levels are supposedly within optimal or at least normative values.

This finding also raises another important question related with how thresholds have been set in training protocols. The results indicate that higher thresholds, based on higher baseline amplitudes in the beginning of the sessions, lead to more difficulty in spending time above threshold. Therefore, the assumption that setting the threshold session by session based on the same proportion of the baseline amplitude will result in the same degree of difficulty of the neurofeedback task is questionable. Future studies should address this issue directly by comparing different ways of setting thresholds (for example, comparing variable thresholds based on baseline amplitude with fixed thresholds).

LIST OF FIGURES

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LIST OF APPENDICES

APPENDIX 1

EMAIL SENT TO ALL THE STUDENTS FROM THE FACULTY OF SCIENCE AND TECHNOLOGY TO RECRUIT PARTICIPANTS

Dear all

We are looking for people to participate in a research project about neurofeedback training for cognitive enhancement.

This research will involve the administration of psychometric tests, the recording of brain activity and brain wave training through neurofeedback. We will pay £50 for participation.

For further information feel free to contact berta.pacheco@student.anglia.ac.uk

Thank you

Berta Pacheco

APPENDIX 2

ADVERT PUBLISHED IN THE PSYCHOLOGY RESEARCH INFORMATION SYSTEM

The influence of neurofeedback training on executive functioning: Is performance level a mediator?	
Abstract: This research aims to explore the use of neurofeedback training to enhance cognitive abilities. Neurofeedback is a non invasive technique that involves the use of technology to help people self-regulate their own brain electrical activity.	
Description	This project involves comparing the effect of training different brainwaves; therefore, participants will be divided into groups receiving different types of training. You will be randomly allocated into one of these groups and participate in three stages: a pre-assessment stage, where you will complete some psychometric tests and have your electrical brain activity measured; a training stage, where you will participate in ten neurofeedback sessions; and a post training assessment, where similar procedures to the pre assessment stage will be undertaken. First stage and last stage will last approximately 3 hours each. Second stage will last 10 training sessions, of approximately 1 hour each over 5 weeks. After participating in this research project you will be rewarded 50 pounds as a compensation of your time and effort. If you decide to take part in this research a questionnaire will be provided to ensure that you do not have any neurological disorder or mental disorder, or are taking any psychoactive medication. This measure prevents the inappropriate use of a neurofeedback training programme that is not adapted to individual needs. If you want more information about participating in this research please contact Berta Pacheco (berta.pacheco@student.anglia.ac.uk).
Eligibility Requirements	You will be aged 18+. You will not have been diagnosed with a psychological or neurological disorder, and not be taking psychoactive medication.
Duration	180 minutes
Pay	50 Pounds
Researcher	Berta Pacheco Email: berta.pacheco@student.anglia.ac.uk

APPENDIX 3

PARTICIPANT INFORMATION SHEET

Anglia Ruskin University
Cambridge & Chelmsford
Cambridge Campus
East Road, Cambridge, CB11PT
Chelmsford Campus
Bishop Hall Lane, Chelmsford, CM1 1SQ
Call: 0845 271 3333
International: +44 1245 493131
Email: answers@anglia.ac.uk

PARTICIPANT INFORMATION SHEET

You are being invited to take part in a research project. The following information will explain the aims of this study and the procedures that will be undertaken. Please take time to read it carefully before you make any decision. If you have any queries or if you need more information feel free to ask.

SECTION A: THE RESEARCH PROJECT

Title of the research project: The influence of neurofeedback training on executive functioning: Is performance level a mediator?

What is neurofeedback training?

Neurofeedback training is a non-invasive technique that helps people to self regulate their own brain electrical activity.

Neurofeedback uses technology to measure the electric activity of the brain and feeds back that information to the individual in the form of a sound or an image. It is known that different brain waves are associated with different mental states. Therefore, the equipment is set so as to give feedback only when the individual is in a state where certain brain waves are enhanced and/or other brain waves are suppressed. In this way the feedback information acts like a reward and enhances the likelihood of that specific pattern of brain waves reoccurring, enabling people to have control over bodily processes that they are usually unaware of.

For example, if the training goal is to improve alertness, a sound may occur every time the brain wave patterns move into a focused state and this reinforcement will hopefully increase the ability to stay focused even without the neurofeedback equipment.

Purpose and value of the study

This study aims to a) determine if neurofeedback training influences cognitive performance and to b) analyze if different levels of performance on cognitive tasks influence the possible impact of neurofeedback training on cognitive functioning.

Neurofeedback training has been used extensively to ameliorate symptoms of several disorders such as Attention Deficit/Hyperactivity Disorder (AD/HD).

However the use of this technique in healthy individuals who want to improve their cognitive abilities is less studied. Therefore neurofeedback training is still an experimental method and a clear association between this kind of training and cognitive improvements has not been established. Thus, this study intends to

investigate this association, by comparing the effects of enhancing different brain waves.

Furthermore this project will analyze for whom neurofeedback training works best, by examining in what way the level of performance on cognitive tasks affects the results that might be obtained with neurofeedback training.

It is anticipated that this study will help to understand the applications and effects of neurofeedback training and therefore it is expected that the results obtained will add to existing knowledge in this field.

Who is organizing the research

The researcher is Berta Pacheco, an MPhil/PhD student from the Science and Technology Faculty.

What will happen to the results of the study

The results of this study will be written up in the form of a thesis. It is also intended that the results will be included in journal articles and presented at conference or scientific meetings.

Once the study is complete participants will be provided with written information about the overall outcomes.

Contact for further information

berta.pacheco@student.anglia.ac.uk

SECTION B: YOUR PARTICIPATION IN THE RESEARCH PROJECT

Why you have been invited to take part

To carry out this research, the participation of volunteer participants is needed to undergo neurofeedback training sessions and analyze its effect on cognitive performance.

This study aims to recruit 24 people within Anglia Ruskin University. The recruitment of participants has been made through adverts placed at the Cambridge Campus. Students are being recruited as their understanding of the value of research projects to the development of knowledge, makes more likely their participation in academic research.

If you have been diagnosed with a neurological disorder (such as epilepsy, attention deficit disorder, brain tumour, infection or injury), a mental disorder or are taking psychoactive medication your participation in this study is not recommended. Neurofeedback training has been successfully applied in the treatment of several disorders (including epilepsy and attention deficit disorder), through the use of training programmes that help to ameliorate specific symptoms. However this type of training requires treatment programmes that are designed on an individual basis, which is not the case in this study.

If you have been diagnosed with a neurological or mental disorder and are interested in the applicability of neurofeedback training in these cases, feel free to contact the researcher using the email provided in this information sheet for more information and reading resources.

Whether you can refuse to take part

The participation in this study is voluntary. So it is your decision whether you want to take part. If you decide to participate you will be asked to sign a consent form.

Whether you can withdraw at any time

If you decide to participate and change your mind, you are free to discontinue your participation in this project at any time, without giving a reason and without any consequences. Also your data may be excluded from the study if you request this at any time even after participation is complete.

What will happen if you agree to take part

If you decide to take part in this research a questionnaire will be provided to ensure that you do not have any neurological disorder or mental disorder, or are taking any psychoactive medication. As mentioned before, excluding participants with a history of neurological or mental illnesses is necessary because this study is not employing the use of a neurofeedback training programme that is adapted to individual needs.

This project involves comparing the effect of training different brainwaves, therefore participants will be divided into groups receiving different types of training. You will be put into one of these groups by chance.

After that you will be asked to participate in the three stages of this project:

Stage I – Pre training measures

This stage comprises one session that will last approximately 3 hours. First, psychometric tests will be used to assess cognitive performance. The tasks that you will be asked to do will be explained carefully.

A quantitative electroencephalogram (QEEG) will also be performed. The QEEG is an assessment tool that uses technology to collect information about the brain electrical activity. In order to obtain this information electrodes are placed on the scalp and ears, using a cap with 19 electrodes built into it. To make sure that a good connection between the skin and electrodes exists, the skin will be cleaned with a gel. In addition a small amount of a conductivity paste (which conducts electricity from the scalp to the electrode) will be injected through the electrode sites with a blunted needle. After this the researcher will make sure that a good contact exists at each electrode site. The recording of electric brain activity will occur during eyes closed and eyes open conditions. At the end of the session the cap is removed and electrode sites are cleaned with a tissue to remove residues of the conductivity paste.

Stage II – Neurofeedback training

At this stage you will be asked to attend 10 neurofeedback sessions during 5 weeks, twice a week. Each neurofeedback training session will last approximately 1 hour.

The beginning of a neurofeedback session is similar to a QEEG session: the electrode sites (one scalp electrode and two ear electrodes) will be cleaned with a gel, electrodes will be filled with conductivity paste before being placed and the quality of the contact between electrode and scalp or ear will be checked.

After that the recording of your brain electrical activity starts. When the predefined goals are met (for example enhancing a certain brainwave) a specific feedback information appears (for example the image of a square becomes bigger), thus informing you when you reach the desired state. Finally, electrodes are removed and remains of the conductivity paste are cleaned with a tissue.

Stage III – Post training measures

The exact same procedures undertaken in stage I will be used here.

After participating in the three stages of this research project you will be rewarded 50 pounds as a compensation of your time and effort.

Whether there are any risks involved

The QEEG assessment and the neurofeedback training are both non invasive methods that have no known risks.

Agreement to participate in this research should not compromise your legal rights should something go wrong

Whether there are any special precautions you must take before, during or after taking part in the study

To make sure that a good connection between electrode and scalp or ear exists it is advisable to not use any hair products (like hair spray, mousse, gel or conditioner) on the day of the QEEG assessment and on the days of neurofeedback sessions.

What will happen to any information/data that are collected from you

All information obtained will be analyzed to meet the aims of this project and will not be used for any other purposes. Paper documents will be stored in a locked filing cabinet at the University and computer files will be password protected.

What are the benefits from taking part

Usually people find it interesting to have the opportunity to control their own brain electrical activity and see recordings of that activity. Also, being part of this research means you are actively contributing to improve knowledge in the neurofeedback field.

How your participation in the project will be kept confidential

The information obtained from all participants will only be accessible to the researcher and supervisory team. The names of participants will be removed from the results and substituted by numbers. Paper documents will be stored in a locked filing cabinet at the University and computer files will be password protected.

Any publication or presentation that includes this research will present the results without identifying any participant.

YOU WILL BE GIVEN A COPY OF THIS TO KEEP, TOGETHER WITH A COPY OF YOUR CONSENT FORM

APPENDIX 4

PARTICIPANT CONSENT FORM

Anglia Ruskin University
Cambridge & Chelmsford
Cambridge Campus
East Road, Cambridge, CB11PT
Chelmsford Campus
Bishop Hall Lane, Chelmsford, CM1 1SQ
Call: 0845 271 3333
International: +44 1245 493131
Email: answers@anglia.ac.uk

PARTICIPANT CONSENT FORM

Name of participant:

Title of the project: The influence of neurofeedback training on executive functioning: Is performance level a mediator?

Main investigator and contact details

Berta Pacheco
Email: berta.pacheco@student.anglia.ac.uk

Supervisory team:

Dr. Neil Rutterford, Dr. Peter Bright and Dr. Bettina Mohr.

1. I agree to take part in the above research. I have read the Participant Information Sheet which is attached to this form. I understand what my role will be in this research, and all my questions have been answered to my satisfaction.
2. I understand that I am free to withdraw from the research at any time, for any reason and without prejudice.
3. I have been informed that the confidentiality of the information I provide will be safeguarded.
4. I am free to ask any questions at any time before and during the study.
5. I have been provided with a copy of this form and the Participant Information Sheet.

Data Protection: I agree to the University[1] processing personal data which I have supplied. I agree to the processing of such data for any purposes connected with the Research Project as outlined to me

Name of participant (print)…….Signed……..….Date……

Name of witness (print)……..Signed……..….Date……

YOU WILL BE GIVEN A COPY OF THIS FORM TO KEEP

If you wish to withdraw from the research, please complete the form below and return to the main investigator named above.

Title of Project:

I WISH TO WITHDRAW FROM THIS STUDY

Signed: _____ Date: _____

[1] "The University" includes Anglia Ruskin University and its partner colleges

APPENDIX 5

HEALTH RELATED QUESTIONNAIRE

SCREENING QUESTIONNAIRE

Research Project title: The influence of neurofeedback training on executive functioning: Is performance level a mediator?

Main investigator and contact details

Berta Pacheco
Email: berta.pacheco@student.anglia.ac.uk

Thank you for taking time to answer this questionnaire. The answers to the following questions about your medical history will be used to identify if the participation in this study is appropriate for you. Your responses will be treated with the strictest confidentiality.

1. Have you ever been diagnosed with a mental disorder (for example, anxiety disorders, depression, eating disorders, attention deficit disorder)?

   Yes ☐ No ☐

2. Are you currently taking prescribed medication for mental health needs?

   Yes ☐ No ☐

3. Have you ever been diagnosed with a neurological disorder (for example, brain tumour, brain infections, epilepsy/seizure disorders)?

   Yes ☐ No ☐

4. Have you ever had a serious brain injury?

   Yes ☐ No ☐

I certify that the above information provided by me is true

Signature:_____ Date:_____

APPENDIX 6

DEBRIEFING FORM GIVEN TO THE CONTROL GROUP

Anglia Ruskin University
Cambridge & Chelmsford
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East Road, Cambridge, CB11PT
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Email: answers@anglia.ac.uk

DEBRIEFING FORM

Thank you for being part of the present study as a research participant. The following information will explain the nature and design of this research project. If you would like more information feel free to ask.

Title of the research project: The influence of neurofeedback training on executive functioning: Is performance level a mediator?

Aims of the study

This research is concerned with studying the effects of neurofeedback training on cognitive performance of healthy individuals. This study is also concerned with identifying if the level of performance on cognitive tests before neurofeedback influences the impact that neurofeedback training could have on training person's cognitive abilities.

If neurofeedback training is found to be capable of enhancing cognitive performance, it could be offered as a peak performance training technique.

Research Design

To study these questions the participants were assigned randomly to two different groups. One of these groups was the experimental group, which underwent neurofeedback sessions that involved training to enhance one brainwave called sensorimotor rhythm (SMR). This brainwave is usually associated with attention and impulse control.

Higher levels of this brainwave have shown to be relevant for a type of cognitive processing that is usually referred to as executive function. Executive functions are involved in activities such as planning, facing new situations and solving problems. Therefore, SMR training was included in this project so as to compare its influence on this type of cognitive process.

The second group was a control group that received 'sham' neurofeedback. In this condition participants are played the feedback from a previously recorded neurofeedback session instead of feedback based on their own brain activity. Thus, this group received everything that the other two groups received except the correct feedback information.

The lack of studies that include control groups is one of the reasons why neurofeedback training has not been clearly associated with cognitive improvement. This type of control element is important to help rule out the influence of unspecific effects, such as expectations of improvement. So if the experimental group shows

greater enhancement in cognitive performance than the control group, it is likely that this is due to neurofeedback training.

You were part of the control group and as such you received 'sham' neurofeedback training. For the reasons explained before, the existence of a control group is important to determine if neurofeedback training can actually influence cognitive performance. Therefore, your participation in this group was highly relevant to clarify the nature of this association.

Your participation is very much appreciated and we understand if you feel disappointed because you did not receive actual neurofeedback training. For this reason you have the opportunity to take part in a neurofeedback course and receive real neurofeedback training if you wish. To arrange this you can use the contact information given at the end of this form.

How your participation in the project will be kept confidential

As stated earlier, when you decided to participate in this study, all the information obtained from all participants will only be accessible to the researcher and supervisory team. The names of participants have been removed from the results and substituted by numbers. Paper documents have been stored in a locked filing cabinet at the university and computer files have been password protected.

Any publication or presentation that includes this research will present the results without identifying any participant.

Information about the research

If you are interested, once this study is complete you can be sent the overall results. Feel free to use the contact information in this form if you have any queries about this project and if you need more information.

If you are interested in this area of research you may wish to read the following references:

- Norris, S. L., & Currieri, M. (1999). Performance enhancement training through neurofeedback. In J. R. Evans & A. Abarbanel (Eds.), Introduction to quantitative EEG and neurofeedback (pp. 223-240). San Diego: Academic Press.

- Vernon, D. J. (2005). Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. *Applied Psychophysiology and Biofeedback*, *30* (4), 347-364

Contact information

If you have any questions or concerns about this research feel free to contact Berta Pacheco.

Email address: berta.pacheco@student.anglia.ac.uk.

Thank you very much for participating!

APPENDIX 7

DEBRIEFING FORM GIVEN TO THE EXPERIMENTAL GROUP

Anglia Ruskin University
Cambridge & Chelmsford
East Road, Cambridge, CB11PT
Chelmsford Campus
Bishop Hall Lane, Chelmsford, CM1 1SQ
Call: 0845 271 3333
International: +44 1245 493131
Email: answers@anglia.ac.uk

DEBRIEFING FORM

Thank you for being part of the present study as a research participant. The following information will explain the nature and design of this research project. If you would like more information feel free to ask.

Title of the research project: The influence of neurofeedback training on executive functioning: Is performance level a mediator?

Aims of the study

This research is concerned with studying the effects of neurofeedback training on cognitive performance of healthy individuals. This study is also concerned with identifying if the level of performance on cognitive tests before neurofeedback influences the impact that neurofeedback training could have on training person's cognitive abilities.

If neurofeedback training is found to be capable of enhancing cognitive performance, it could be offered as a peak performance training technique.

Research Design

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Higher levels of this brainwave have shown to be relevant for a type of cognitive processing that is usually referred to as executive function. Executive functions are involved in activities such as planning, facing new situations and solving problems. Therefore, SMR training was included in this project so as to compare its influence on this type of cognitive process. You were part of the experimental group that underwent SMR training.

The second group was a control group that received 'sham' neurofeedback. In this condition participants are played the feedback from a previously recorded neurofeedback session instead of feedback based on their own brain activity. Thus, this group received everything that the other two groups received except the correct feedback information.

The lack of studies that include control groups is one of the reasons why neurofeedback training has not been clearly associated with cognitive improvement. This type of control element is important to help rule out the influence of unspecific effects, such as expectations of improvement. So if the experimental group shows greater enhancement in cognitive performance than the control group, it is likely that this is due to neurofeedback training.

How your participation in the project will be kept confidential

As stated earlier, when you decided to participate in this study, all the information obtained from all participants will only be accessible to the researcher and supervisory team. The names of participants have been removed from the results and substituted by numbers. Paper documents have been stored in a locked filing cabinet at the university and computer files have been password protected.

Any publication or presentation that includes this research will present the results without identifying any participant.

Information about the research

If you are interested, once this study is complete you can be sent the overall results. Feel free to use the contact information in this form if you have any queries about this project and if you need more information.

If you are interested in this area of research you may wish to read the following references:

- Norris, S. L., & Currieri, M. (1999). Performance enhancement training through neurofeedback. In J. R. Evans & A. Abarbanel (Eds.), Introduction to quantitative EEG and neurofeedback (pp. 223-240). San Diego: Academic Press.

- Vernon, D. J. (2005). Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. *Applied Psychophysiology and Biofeedback*, *30* (4), 347-364

Contact information

If you have any questions or concerns about this research feel free to contact Berta Pacheco.

Email address: berta.pacheco@student.anglia.ac.uk.

Thank you very much for participating!

APPENDIX 8

DESCRIPTION AND PROCEDURES FOR ADMINISTERING THE BADS

Test1 – Rule shift cards

In this test the participants were presented with a booklet of 21 playing cards, which were turned one at a time. For the first trial, participants were instructed to say "Yes" when they saw a red card and "No" to a black card. In the second trial the rule changed and the participants were asked to say "Yes" only if the card shown was the same colour as the previous and "No" if the card shown was a different colour from the previous one.

The goal of this test is simply to assess the ability of the participant to shift from the first rule to the second rule, therefore measuring the ability to adapt behaviour to a new situation and at the same time inhibiting a previous behaviour.

Test 2 – Action program

This test is aimed at measuring the ability to solve new problems by formulating a plan of action to solve a problem and to achieve an end.

The participants were presented with a stand, which had in on end a beaker that was two thirds full of water and had a lid with a small hole in it. In the other end of the stand there was a tall transparent tube with a cork in the bottom. On the left side of the stand there was an L-shaped wire hook (not long enough to reach the cork), a container and the container screw top (placed next to the container). The participants were asked to get the cork out of the tube without lifting the stand, the beaker or the tube, and without touching the beaker's lid with the fingers.

To successfully complete this task the participants had to follow 5 steps:

1. Removing the lid with the metal hook
2. Attaching the screw top to the container
3. Filling the container with water taken from the beaker
4. Pouring the water into the tube
5. Repeating last step so that the cork could float to the top of the tube.

What was being evaluated was the ability of the participant to complete each stage independently, therefore there was not a time limit for this task.

Test 3 – Key search

This test evaluates the ability to plan a strategy to solve a problem, in this case finding a key that is lost in a field.

The participants were presented with an A4 piece of paper with a 100 mm square in the center and a 50 mm dot below it. The participants were then asked to imagine that the square was a field where they had lost their keys. They were told to draw a line starting from the dot to show where they would walk to make sure that they would find the keys, no matter where they were.

The scoring criteria for this test comprised the evaluation of different components of the line drawn that made possible to judge the effectiveness of the strategy. The most effective strategies involve:

– Entering the field and finishing the search in a corner.

- Drawing a continuous line.
- Using search patterns made of parallel lines and vertical or horizontal lines
- Using systematic search patterns that are single planned
- Covering all ground
- Higher probability of finding the keys.

Test 4 – Temporal Judgment

For this test participants had to answer four questions that required an accurate estimation of times for commonplace events. According to Wilson et al. (2003), this test is apparently used to assess abstract thinking and judgment.

Test 5 – Zoo map

In this test participants were asked to show how they would visit a number of locations on a map of a zoo according to certain rules. This test has two conditions. The first condition is a high demand condition, which involves being able to create and execute a plan. The second condition is a low demand condition that requires following a plan that has already been created. Both versions measure the ability of the participant to regulate his own behaviour by modifying his course of action when a rule is broken.

Test 6 – Modified six elements

This test aims to measure the ability to plan, organize and monitor behaviour.

The participants were presented with the following test materials: 2 booklets containing arithmetic problems, 2 booklets containing pictures, a timer, a digital recorder, a sheet of paper, a pencil and an eraser. The test materials were presented in the exact same way for all participants and as suggested in the manual. The participants were told that they would get ten minutes to complete the task. The task consisted in writing down the names of the pictures shown in the booklets (Pictures A and Pictures B), writing down the answers to two sets of arithmetic problems (Arithmetic A and Arithmetic B) and describing two events (Dictation A and Dictation B). The participants were told that they had to try to complete at least some of each of the six individual parts, although they did not have to complete everything and/or any one task. The participants were also instructed not to move on to the second part (part B) of a task immediately after attempting the first part (part A) and to not move on to the first part of task after attempting the second part.

The order in which the tasks were attempted was recorded, so as to evaluate if the rule was followed. Also, the time that the participant started to engage in any given part of a subtask and the time the participant stopped performing that task was recorded, so as to calculate afterwards the time spent on each sub task.

APPENDIX 9

ASSESSMENT OF PERCEPTION OF CONTROL OF BRAIN ACTIVITY

How well do you feel you have been able to control your brainwaves?

Session _____

1	2	3	4	5
Not at all	Not very well	Reasonably well	Very well	Extremely well