## Master research Clinical Psychology:

# The effect of Neurofeedback training on healthy individuals as measured by objective and self report measures of attention and impulsivity, when compared to a sham control condition.

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# Abstract

Current medical treatment of patients with Attention Deficit Hyperactivity Disorder (ADHD) is not entirely satisfactory. An alternative, but not yet fully understood, way to improve attention and impulsivity is neurofeedback training. This procedure is aimed at learning to regulate brain wave activity. Previous studies reported significant benefits of neurofeedback training, using active and passive control groups, but double-blind sham controlled studies are lacking so far. This study attempts to address this problem. Participants in this study were randomly assigned to either a treatment group, or a sham group and eventually received 16 20-minute sessions of neurofeedback. The results indicate several (trends towards) main effects, but no group x time interactions for neurofeedback treatment are found. From these results it can be concluded that 16 sessions of neurofeedback have no effect above placebo on measures of attention and impulsivity.

## Preface

After months of hard work and a lot of stress, we have finally completed our research for our master Clinical Psychology. Throughout the entire project we have worked together intensively. Even though this research has been a source of great stress, we have enjoyed the work, the endless discussions and going through the process of how to conduct a research together. This research is the result of great teamwork, but each of us has also looked deeper into some aspects of the study. Zsófia has dug deeper into the qEEGs, Marieke the self-report questionnaires, and Susanna the computer tasks.

We could not have done this study without the help of a lot of people. First of all we would like to thank Alexander Logemann for asking us to join his very interesting research. Without him it would have been impossible to conduct this study; he has great knowledge on this subject and has (re)written several computer programs used in this study, making it possible to make a truly double blind research. We wish him the best of luck with his PhD. We would also like to thank Drs. G.F.A. Loots from the Neurofeedback Instituut Nederland for enthusiastically teaching us the ins and outs of neurofeedback and of course for letting us use the EEG apparatus and the neurofeedback program. When thanking everyone we cannot possibly leave out are partners, who, with their social support and patience, have supported us throughout this research. Last, but certainly not least, we would like to give our special thanks to Prof. dr. Lorenz van Doornen from the Universiteit Utrecht who has supervised our research and has given us very useful hints and tips for our research and our research paper.

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# Introduction

The treatment of patients with Attention Deficit Hyperactivity Disorder (ADHD) is far from satisfactory. Most treatments consist of pharmacotherapy, of which Ritalin is the most widely used drug. About 70 to 80 % of children with ADHD can benefit from the use of Ritalin, of which about 35 % improve because of the placebo effect (Barkley, 1998).
According to these data it could be argued that children with ADHD are receiving proper treatment, unfortunately this does not seem to be the case. First of all, 20 to 25 % of the children do not respond to Ritalin (Swanson, Sergeant, Taylor, Sonuga-Barke, Jensen & Cantwell, 1998). This percentage may be even higher in adults with ADHD, they are often prescribed anti-depressants as alternative medication for ADHD symptoms (Thompson & Thompson, 2005). Secondly, after cessation of psychopharmacological treatment, the symptoms of ADHD return (Barkley, 1998; Thompson & Thompson, 2005). In addition, pharmacotherapy has serious side effects on short-term such as headaches, stomach aches, irritability, decreased appetite, and sleeping problems (Goldstein & Goldstein, 1990), and negative effects with long-term use such as teeth grinding, cardiac rhythm changes, blood pressure increases, and anxiety/nervousness (Gunkelman & Johnstone, 2005).
As an alternative to sole pharmacological intervention, numerous behavioral programs have been designed to treat ADHD in combination with or without medication. The effectiveness of these treatments is not entirely elucidated, some children respond well whereas some do not (Van der Oord, Prins, Oosterlaan & Emmelkamp, 2008).

## Neurofeedback training

An alternative, but not yet fully understood, way to improve attention and impulsivity is to regulate the brain activity through neurofeedback training. This method is non-invasive, and to this date no negative side effects are documented. In addition to the effects on ADHD symptoms, the individual also gains self-esteem, due to a feeling of personal control (Thompson & Thompson, 2005). Furthermore, whereas the use of pharmacotreatment seems to produce state dependent learning, i.e. the medication only works while it is in the system of the patient, neurofeedback training seems to have a long-lasting effect (Lubar, 1997) because learning is internalized (Gunkelman & Johnstone, 2005). In addition, because of the fact that this method is non-invasive, people without an official ADHD diagnosis, but who do experience impairment because of ADHD characteristics can also profit from training (Thompson & Thompson, 2005). In neurofeedback training an individual

is being fed back the own brain activity, and is instructed to try to regulate his/her brain activity to an appropriate level. When brain wave activity reaches an appropriate level, the participant is rewarded, e.g. by gaining points in a computer game or by being enabled to watch a movie.

Different frequency bands of the human electroencephalogram (EEG) can be brought under operant control by means of this training process. This process involves the registration of a specific frequency and giving constant feedback on it (EEG-bio/neurofeedback loop) (Egner & Gruzelier, 2001). The principal feasibility of learned self regulation has been demonstrated for slow cortical potentials (SCPs) (Birbaumer, 1984; Strehl, Leins, Goth, Klinger, Hinterberger & Birbaumer, 2006) and EEG frequency components (Kamiya, 1968).

## Theoretical background

### *Differences between people diagnosed with ADHD and non-ADHD people*

Numerous studies over the last decades have identified differences between ADHD and non-ADHD children in brain activity. These differences are found in resting condition as well as when performing specific cognitive tasks. Individuals diagnosed with ADHD are generally found to have a surplus of slow wave activity, mostly in the theta bands (4-7 Hz), and deficiencies in fast wave activity, mostly in low beta bands (12-22 Hz) when compared to people without the diagnosis of ADHD (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy & Selikowitz, 1998, 2001, 2001a, 2001b, 2001c; Clarke, Barry, McCarthy, Selikowitz & Brown, 2002; Fox, Tharpe & Fox, 2005; Mann, Lubar, Zimmermann, Miller & Muenchen, 1992; Satterfield, Cantwell, Lesser, Podosin, 1972). A component of the low beta band is SMR (12-15 Hz sensori motor rhythm). These differences are found mostly in frontal regions of the brain (Mann et al., 1992). These differences are usually interpreted as hypoarousal, having an underaroused central nervous system (Gunkelman & Johnstone, 2005; Lubar, 1991). When an individual with ADHD is presented with an attentional task, such as reading or listening to a story, his/her EEG usually shifts to a low frequency (theta) without any significant increase in frontal fast wave activity which is normally expected (Lubar, 1991; Mann et al., 1992).

It appears that QEEG is able to successfully differentiate between individuals with and without the diagnosis of ADHD (Chabot & Serfontein, 1996). Specificity of QEEG differentiation in this study was found to be 88 %, sensitivity was found to be 93,7 %.

***EEG profiles***

In several neurobiological studies that compared individuals diagnosed with ADHD people to those without this diagnosis, different endophenotypes of ADHD were found. Three different endophenotypes emerged from literature. Most patients diagnosed with ADHD seem to be cortically hypoaroused, which means decreased neurophysiologic activity in frontal regions of the brain is found compared to controls from a healthy population (Ernst, Liebenauer, King, Fitzgerald, Cohen & Zametkin, 1994). On EEG measures, these patients show decreased fast wave beta 1 activity (12-22 Hz) and increased slow wave theta (4-7 Hz) activity as compared to healthy subjects (Chabot & Serfontein, 1996; Clarke et al, 1998, 2001, 2001a, 2001c; Clarke et al., 2002; Egner & Gruzelier, 2004; Monastra, Lynn, Linden, Lubar, Gruzelier & LaVaque, 2005). Another group seems to present EEGs similar to that of much younger subjects and are therefore commonly referred to as a maturational lag endophenotype (Clarke et al., 2001; Magee, Clarke, Barry, McCarthy & Selikowitz, 2005; Mann et al., 1992). On EEG measures, these patients show decreased fast wave activity in the alpha (8-12 Hz) and beta 1 bands. In contrast to the cortically hypoaroused group of ADHD patients, there is also a smaller group of patients who show the opposite pattern of activity, and are commonly referred to as a hyperaroused group (Clarke et al., 2001b). This is represented in the EEG by a surplus of beta 1 activity, decreased alpha activity and a decreased theta/beta ratio (Clarke et al., 2001b; Clarke et al., 2002; Chabot & Serfontein, 1996; Monastra et al., 2005). It is assumed that this group reacts poorly to stimulant medication (Fox et al., 2005; Monastra et al., 2005).

In adults with ADHD different endophenotypes of ADHD were found as well. Hypoarousal is found in 80 % of the adult ADHD population, represented by a high theta/beta1 ratio. This group is referred to as 'attentional drifters' by Thompson and Thompson (2005). Hyperarousal is found in the remaining 20 %. This latter group is distracted by concerns or thoughts unrelated to the task at hand, and are referred to as having a busy brain by Thompson and Thompson (2005). This distraction is related to high beta 2 (20-34 Hz) activity, and a decrease in SMR activity. This EEG pattern is also associated with being anxious and with ruminating.

## Effect of neurofeedback training

In this section the effects of neurofeedback training on various measures of effect will be discussed. First, the effect of neurofeedback on EEG measures will be discussed. Effects on EEG measures following neurofeedback training represent an objective measure of the

effect of neurofeedback on brain wave activity. Second, the effects of neurofeedback on computer task performance will be documented. The performance of subjects with ADHD on computer tasks represents an objective measure of performance in tasks requiring attention and response inhibition. Last of all, the effects of neurofeedback on subjective measures will be discussed. This represents the degree to which participants improve on subjective measures of behavior.

## *Objective measures*

### *EEG measures*

The effect of neurofeedback on EEG measures of brain wave activity is documented extensively. Participants were able to successfully learn to regulate brain wave activity associated with attention and impulsivity. This is documented in several case studies (Lubar, Swartwood, Swartwood & 'O Donnell, 1995; Thompson & Thompson, 1998) as well as in controlled group studies (Beauregard & Lévesque, 2006; Monastra et al., 2002; Vernon et al., 2003). This indicates that learning to regulate EEG activity is not due to a placebo effect, as it was also shown in controlled group studies, where participants in the control group did not manage to achieve significant changes in their EEGs.

Neurofeedback training involves reinforcing only appropriate activity and inhibiting inappropriate activity. In line with the abnormalities of power in EEG frequencies in ADHD patients, three protocols have been originally designed. These classical protocols are SMR enhancement and accordingly theta suppression, SMR enhancement and high beta suppression (22-30 Hz), and lastly beta 1 (16-20 Hz) enhancement and theta suppression. Studies done on the effectiveness of these protocols in reducing symptomatology in ADHD have shown results on attention and impulsivity comparable to Methylphenidate with 75% of patients improving (Monastra et al., 2005). A study of Monastra, Monastra and George (2002) suggests the effects last for at least a year after cessation of neurofeedback training. However, none of the studies were placebo-controlled and not all were double-blind, making it impossible to draw conclusions about the effect of the intervention on attentional performance and impulsivity above the placebo effect (Rossiter, 2004; Rossiter & LaVaque, 1995; Monastra et al., 2002; Fuchs, Birbaumer, Lutzenberger, Gruzelier & Kaiser, 2003).

In neurofeedback protocols, a stated assumption is that the use of the SMR protocols addresses problems of hyperactivity and impulse control and that the use of beta1 (15-18 Hz) protocols is held to alleviate symptoms of inattentiveness (Lubar & Lubar, 1984). However, the specific impact of SMR and beta1 training on these domains has not unequivocally been demonstrated (Egner & Gruzelier, 2004). The study of Egner & Gruzelier

(2001), where healthy volunteers received neurofeedback training of the SMR and beta1 band components, led to significant and protocol-specific effects, which argues against a mere placebo effect. Both SMR and beta1 training improved cognitive integration of sensory input, but their effects on the subsequent motor response were counteractive. Whereas SMR training reduced improved inhibitory motor control, beta 1 training led to fast but error prone responding. This study is the only study found reporting dose-response effects, relative success at SMR enhancement correlated positively with attention performance.

A second study of Egner and Gruzelier (2004) solved some methodological difficulties and also found protocol specific effects, i.e. the effects of different protocols on performance were not equal. SMR training was associated with less variability in reaction time and less inattentive errors, as opposed to reduced impulsive errors. This was interpreted as a general attention enhancing effect of SMR uptraining, with varying contributions to reducing inattentive and impulsive errors. These relative contributions may be dependent on the initial error profile. In a study of Vernon, Egner, Cooper, Compton, Neilands, Sheri and Gruzelier (2003) SMR uptraining was investigated. The attention enhancing effect of SMR training found by Egner and Gruzelier (2004) was replicated. These studies do suggest protocol-specific effects and argue against a mere placebo effect of neurofeedback training. However, the magnitude of the effect of regulating EEG on attention and impulsivity above placebo is still unknown on the basis of former research. As Vernon (2005) put it, the role of neurofeedback in enhancing performance has yet to be established.

Contrary to studies mentioned above Heywood and Beale (2003) found EEG biofeedback to be no more effective than a placebo control condition involving non-contingent feedback. In this pilot study neither condition was found to result in improvements relative to baseline levels of attention and impulsivity.

***Computer tasks***

In the last four decades, the Continuous Performance Test (CPT) has been one of the most widely used tests to measure attention and improvement in attention, both in clinical settings as in research settings (Van Leeuwen et al., 1998). The CPT has been claimed to measure inattention as well as impulsivity. There has been much debate as to what the various parameters of the CPT actually measure in relation to ADHD behaviors. Errors of commission (i.e. responses that occur when no response is required) are believed to reflect impulsivity, whereas errors of omission (i.e. the absence of a response when one is required) are assumed to reflect symptoms of inattention (Epstein, Erkanli, Conners, Klaric, Costello, and Angold, 2003). Furthermore, Epstein et al. (2003) found that subjects

diagnosed with ADHD have more variable mean reaction times. Because of this, the standard deviation of the mean reaction time was used as dependent measure in our study.
The above mentioned measures – errors of omission, errors of commission and variability- have been shown to be significantly different between pretreatment and on-medication conditions when evaluating the effects of stimulant medication on performance (Lubar, Swartwood, Swartwood & O'Donnell, 1995).
Since CPTs have shown sensitivity to medication effects on ADHD, they will also be useful in evaluating the outcomes of alternative treatments (Rossiter, 2004) such as neurofeedback. In accordance, several studies have found positive effects of both stimulant drugs and neurofeedback in treating ADHD on the TOVA, a computerized visual CPT (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; Monastra et al., 2002). Furthermore, Lubar et al. (1995) have demonstrated that changes on TOVA performance following downtraining of theta activity provides evidence that decreasing slow EEG activity leads to more normal performance on a CPT. In addition, these researchers showed that with medication, improvement on the TOVA only exists while a stimulant is still in the participant's system whereas with neurofeedback improvement also occurs whilst the participant does not have a training during the CPT task. Furthermore, reinforcing beta1 and SMR activity and downtraining theta activity was found to improve scores on the TOVA (Fuchs et al., 2003; Linden, Habib, & Radojevic, 1996; Monastra et al., 2002; Rossiter, 2004; Rossiter & LaVaque, 1995; Vernon et al., 2003).
Besides studies that have looked at improvement on CPT performance due to neurofeedback training in individuals with ADHD, the effect of neurofeedback has also been studied in healthy subjects. In their studies, Egner and Gruzelier (2001, 2004) showed that uptraining SMR (12-15 Hz) was associated with better perceptual sensitivity and a decline in commission errors on the CPT. High beta uptraining (15-18 Hz) resulted in the opposite effect.
The Stop Signal paradigm has been used extensively to measure inhibitory motor control (Logan, 1994). The stop signal reaction time (SSRT) is believed to reflect the ability to inhibit responses (Schachar, Mota, Logan, Tannock, and Klim, 1999). Children with ADHD have been shown to have significantly longer SSRTs and thus significantly impaired inhibitory control compared to normal controls or children with conduct disorder.
Beauregard and Lévesque (2006) studied the effect of SMR and beta1 uptraining op the Stroop and on the Stop task. After neurofeedback training, the subjects in the experimental condition scored better at both tasks than subjects in the control condition. Furthermore, as shown on fMRI, whilst doing the STOP task the experimental group showed significant

activation in the right ACcd, right ventrolateral prefrontal cortex, left thalamus, left caudate nucleus, and left substantia nigra, whereas these activations were not seen before the training. These differences were not found in the control group after the same neurofeedback training. This suggests that neurofeedback training does cater for changes in brain activation whilst doing a task that asks for an ability to inhibit responses.

## *Subjective measures*

### *self-report questionnaires*

Prior research using self-report measures is scarce, if not non-existent. This fact can be explained by the fact that research assessing the effects of neurofeedback on ADHD is done almost solely on children. Using self-report questionnaires in research with children is unconventional, most subjective measures used are teacher and parent reports.

Since the current experiment was carried out with healthy college students, two self-report measures were used. Namely, the STIMP, a questionnaire measuring state impulsivity, and the Adult Self-Report questionnaire (ASR), based on DSM–IV criteria (Achenbach & Rescorla, 2003). Since the DSM-IV criteria are also used as the basis of many parent and teacher report scales, it will be assumed that scores on the ASR DSM are comparable to scores on other DSM based subjective report scales.

The effect of neurofeedback training on frequently used report scales will be discussed below.

One of the scales of the IOWA-Conners Behavior Rating Scale is an ADHD scale. This scale consists of 5 items measuring inattentiveness and 10 items measuring overactive and impulsive behaviour. These items are based on DSM criteria for ADHD (Collett, Ohan, & Myers, 2003). Parents and teachers reported progress after neurofeedback training as measured by this scale (Fuchs et al., 2003; Linden et al., 1996; Leins, Goth, Hinterberger, Klinger, Rumpf, & Strehl, 2007).

The ADD Evaluation Scale (ADDES) consists of 46 items, giving an indication of the frequency of ADHD symptoms, and is based on DSM criteria. It is available in both a Home version and a School version (Collett, Ohan, & Myers, 2003). Effect of neurofeedback on the Inattentiveness subscale of the ADDES is consistently reported (Carmody, Radvanski, Wadhwani, Sabo, & Vergara, 2001; Monastra et al., 2002; Lubar et al., 1995). Improvement on the Hyperactivity/Impulsivity Scale is also reported (Monastra et al., 2002; Lubar et al., 1995). When interpreting these results it should be taken into account that in

the study by Lubar and colleagues both the group showing EEG changes as the group not showing EEG changes improved on the ADDES.

The Behavior Assessment System for Children (BASC) is a comprehensive set of rating scales and forms including parent, teacher and self-report ratings (Collett, Ohan, & Myers, 2003). Improvement on scales relevant for ADHD problems is reported (Rossiter, & LaVaque, 1995; Rossiter, 2004).

The Brown ADD Scales (BADDS) can be used by clinicians or significant others. It consists of 40 items measuring attention problems in the absence of hyperactivity (Collett, Ohan, & Myers, 2003). Rossiter (2004) reports significant improvement of the BADDS after neurofeedback training.

The SNAP is a parent or teacher behaviour rating scale based on DSM criteria and closely resembles other DSM based ADHD scales (Collett, Ohan, & Myers, 2003). Improvement after neurofeedback training as measured by this instrument is reported by Linden and colleagues (1996) and Leins and colleagues (2007).

## Conclusion

Neurofeedback is generally found to have positive effects on measures of ADHD symptoms. Positive effects are found with respect to objective measures, that is EEG substrates of attention and impulsivity in the brain, as measured by qEEG, and computer tasks as the CPT and Stop task. Also positive effects of neurofeedback on subjective behavioural measures are reported.

Despite of the general trend of studies documenting positive effects of neurofeedback training, Heywood and Beale (2003) found EEG biofeedback to be no more effective than a placebo control condition involving non-contingent feedback. In this pilot study neither condition was found to result in improvements relative to baseline levels of attention and impulsivity.

Most studies up to this date have not involved double-blind controlled studies involving a sham condition, so it is not entirely possible to rule out that the placebo effect is responsible for these positive results, hence the current study was carried out. Also only one study reported dose-response effects for neurofeedback.

**Objective of this study**

In order to find a solution to these problems with treating ADHD, this master research was carried out to investigate the effect of neurofeedback training on attention and impulsivity on healthy subjects, as measured by subjective self-report measures and objective measures. This research is a follow-up of our previous bachelor research which was conducted as a screening for inclusion of participants. It is hoped that neurofeedback training improves the two major domains in which children with ADHD experience problems, namely impulsivity and (in)attention.
This research was conducted as a cooperation with the Neurofeedback Institution Netherlands (www.neurofeedback.nl). The required equipment such as the qEEG recorder and the neurofeedback apparatus were provided by them.

**On the basis of previous research the following research questions and hypothesis have been formulated:**

1. Are there any EEG abnormalities in healthy subjects with attention/impulsivity problems? This question can be investigated by correlating the scores of the computer tasks at baseline with the patterns of the qEEG (SMR); a negative correlation is expected (high score on the computer tasks; low SMR activity).
2. Whilst investigating the effect of neurofeedback training on EEG measures, it is investigated whether subjects are able to achieve significant changes in their EEGs. Subjects receiving SMR uptraining are expected to show significant changes in their EEG corresponding to their training protocol.
3. Investigating the effect of neurofeedback treatment on attention and impulsivity on the objective level (computer tasks). Subjects receiving neurofeedback training are expected to improve on the objective measures of attention and impulsivity, the CPT and the Stop task.
4. Investigating the effect of neurofeedback treatment on attention and impulsivity on the subjective level (questionnaires). Subjects receiving neurofeedback training are expected to improve on subjective measures of attention and impulsivity.

# Materials and methods

## Participants

### *Participants, first selection*

Although this research is to aid the search for proper treatment for ADHD, non-diagnosed subjects were used for this study. This was done for several reasons. First of all, it is not always convenient to use people diagnosed with ADHD as subjects for research on alternative treatments for ADHD. Because of the high costs involved, the difficulties in recruitment (i.e. getting enough ADHD diagnosed subjects), and ethical problems. If alternative treatments are to be tested experimentally, patients should abstain from medication during testing in order to prevent too many differences between subjects because of the variety in treatments they would be receiving.

Ideally, to test the efficacy of a new treatment one would want the treatment to be tested in a double-blind design, in which one of the groups would be a placebo control group. This placebo control group would ideally consist of people with the diagnosis ADHD, who will get a sham treatment, but are not on medication. Unfortunately, as mentioned above, it was not possible to use ADHD diagnosed subjects for this study. Therefore, in accordance with the two domains of functioning in which people diagnosed with ADHD experience the most problems, we have chosen to use impulsive healthy subjects, and healthy subjects with problems in sustaining attention.

Psychology students of the University of Utrecht (n = 455) filled out the Barrat Impulsivity Scale (Barrat, 1983), and the Cognitive Failure Questionnaire (Broadbent, Cooper, Fitzgerald & Parkes, 1982), during breaks of lectures. Students who scored higher than 1,5 sd below mean performance on both inattention and impulsivity measurements were asked to participate in the computer tasks. Students who showed relatively little problems with impulsivity or sustaining attention were excluded from the study. For mean scores and standard deviation scores see table 1.

### *Participants, second selection*

Students who were the most impulsive and had the most problems with attention according to the measurements of computer tasks and questionnaires, were asked to participate in the neurofeedback training study.

History of epilepsy was an exclusion criterion, since neurofeedback can trigger seizures in epileptic patients. The use of drugs at time of testing and during the sessions was not

allowed. Students who were diagnosed with any kinds of psychological problems could not take part of the research.

***Participants, matching***

29 participants were willing to participate in the definitive neurofeedback training process. Two students were excluded on the basis of their qEEG measurement since signs of possible epilepsy and drug use were seen in their EEG recordings. The remaining 27 students were randomly assigned either to the treatment or to the sham condition. The subjects in the two conditions were not significantly different on subject characteristics (see table 2). In the sample (n=27) the mean age was 20.92 (SD=2.46), 3 participants were left handed and 3 participants were male. The BIS mean score for the selected NFT population was 66.14 (SD=6.66) and the CFQ mean score was 50.52 (SD=9.08). For the BIS score 65.5 % of included NFT participants scored higher than the population's average en 76 % scored above the median. 62 % of the NFT participants scored higher on the CFQ than the total average and median scores. One student dropped out halfway the experiment due to personal reasons; her data were excluded from analysis.

**Table 1**

*Descriptive Statistics selection and baseline. Mean score and standard deviation of the questionnaires BIS (score) and CFQ (score), and the computer tasks STOP-task (SSRT in ms) and the CPT (SDRT in ms) for all participants and the two matched groups.*

| Time and Group | Test | N | Mean | SD |
|---|---|---|---|---|
| First selection | BIS | 455 | 62.06 | 8.61 |
| | CFQ | 455 | 44.28 | 10.92 |
| Baseline all participants | BIS | 29 | 66.14 | 6.66 |
| | CFQ | 29 | 50.52 | 9.08 |
| | Stop task (SSRT) | 26 | 148.00 | 17.68 |
| | CPT (SDRT) | 26 | 88.42 | 35.11 |
| Baseline NFT group | Stop task (SSRT) | 14 | 146.25 | 22.81 |
| | CPT (SDRT) | 14 | 85.71 | 39.79 |
| Baseline sham group | Stop task (SSRT) | 12 | 149.6 | 19.31 |
| | CPT (SDRT) | 12 | 91.58 | 30.17 |

**Table 2**
*T-test scores of the variables handedness, age, gender, IQ, STIMP score, SSRT (Stop task), and SDRT (CPT task)*

| | t | df | p |
|---|---|---|---|
| handedness | .59 | 26 | .56 |
| age | .65 | 26 | .52 |
| gender | .59 | 26 | .56 |
| IQ | .33 | 26 | .74 |
| STIMP score | .09 | 26 | .93 |
| SSRT | .31 | 26 | .76 |
| SDRT | .32 | 26 | .75 |

## Apparatus

### *Questionnaires*

The Barratt Impulsivity Scale (Barratt, 1983) and the Broadbent Cognitive Failures Questionnaire (Broadbent, Cooper, Fitzgerald & Parkes, 1982) were used for selection and to assess problems with impulsivity and attention, respectively.

The STIMP, a questionnaire measuring state impulsivity, and the Adult Self-Report questionnaire (ASR) (Achenbach & Rescorla, 2003) were used as baseline and interim measures. The ASR has two relevant scales, one scale measuring ADHD problems based on factor analysis of the item pool, and a syndrome scale for ADHD based on DSM-IV criteria for ADHD. The remaining scales measure other forms of psychological problems. This scale has high test-retest reliability ($r$ = .91 and $r$ = .84) and high internal consistency ($\alpha$ = .87 and $\alpha$ = .84) for relevant scales. Content, criterion and construct validity for the ASR are good.

### *Computer Tasks*

In the screening process and as baseline and interim measures the reversed Connor's CPT task and Logan's Stop task were used. The CPT task is held to measure inattention and impulsivity and the Stop task is held to measure impulsivity as represented by response inhibition.

The CPT consisted of one uniquely randomized block of 608 trials comprised out of 16 letters (A,B,C,D,E,F,H,I,L,M,N,O,T,Y,Z, and X), each black letter was presented at the center of the screen on a gray background for 1000ms at a 1000ms interval, and each letter occurred in total 38 times. The goal was to react as soon and as accurate as possible by

pressing a button on a pad after presentation of each letter except the X. Dependent measures were reaction time variability (RTV) as reflected in the standard deviation of the reaction time and the proportion of false alarms (proportion of responding to the X).

Logan's Stop Task yields the Stop Signal Reaction Time (SSRT). Participants were presented with five blocks of unique randomized square-wave black-white grating sequences presented on a gray background. The gratings were 6 x 6 degrees in size and consisted of high or low fundamental spatial frequency, 3.62 cpd and 0.46 cpd respectively. After a fixation cross was presented for 500ms a grating was presented for 750ms. The duration from the end of presentation of a grating to the presentation of the fixation cross was variable between 1000ms – 1250ms.

The task consisted of two types of trials, Go trials and Stop trials. Stop trials were trials in which the grating was followed by a (1000 Hz, 80dB, and 400 ms in duration) tone and a response to the grating had to be withheld. A Go trial was a trial in which the grating was not followed by a tone and a response had to be made. In Go trials, participants had to respond by pressing the right button when presented with a high frequency grating and the left button when presented with a low frequency grating. Each block had the same amount of stop trials and high and low frequency gratings. No more than three stop trials were presented consecutively.

In total 630 trials were presented in five blocks of 126 trials. Each block was a unique randomized sequence of trials per participant. This was done in order to prevent issues with attrition and resulting methodological difficulties when using a rigid block design. Each block consisted of 76 go trials and 50 stop trials. For reliable Stop Signal Reaction times about 50% correct inhibitions need to be made, therefore Stimulus Onset Asynchrony (SOA) (go-stop intervals) were adjusted after each block using a tracking algorithm. Only in the first block the SOA was set at 250ms. The Go-stop interval was jittered around the SOA within a 250ms range to prevent strategies. Since omissions should not be seen as correct inhibitions a correction was performed.

***qEEG assessment***

The Deymed Truscan 32 acquisition apparatus was used for the qEEG electrophysiological assessment. This device uses 24 canals, the sampling rate is 128 per second with a high pass filter of 0,5 Hz and a low-pass filter of 70 Hz. Linked ears montage was used. The ground was placed 1cm in front of the Fz point. The reference was placed 1cm behind Fz.

Tin plated electrodes were used according to the 10/20 system.

The 5 ohm corrected impedances were kept constant between the electrodes.

## Procedure and Experimental Design

The procedure of the study can be seen in table 3.

**Table 3**
*Procedure of the study*

| Part of the study | Measurements |
|---|---|
| First selection | BIS, CFQ |
| Second selection | Stop task, CPT |
| Baseline | qEEG, Stop task, CPT, ASR, Stimp, IQ questionnaire |
| Interim | qEEG, Stop task, CPT, ASR, Stimp |

### *Baseline measurements*

After the selection of the participants, the baseline measurements took place. Baseline measurements consisted of the ASR questionnaires, the Stimp, both computer tasks and the qEEG measurements. At the time of the baseline measurement IQ was also measured for matching purposes.

### *Creating the sham and treatment condition*

The frequency bands to be reinforced (the treatment protocol for a participant) were based on the qEEG of the participants. The treatment protocols were created by the 'Neurofeedback Instituut Nederland' in Eindhoven. They also did the random group assignment, to make sure the experiment remained double blind.

### *Protocols*

In order to create an appropriate protocol, the participants' qEEGs were compared to qEEGs from a large database. Each participant was compared to a group norm with the same age and gender, on the basis of which a treatment protocol was created for each participant. Therefore multiple protocols, each tailored to the individual participant were used and some participants were reinforced for uptraining multiple frequency bands (see table 4). The frequency band inhibited the most was beta2 (22-30 Hz), in 12 participants. After that, the theta band (4-7 Hz) was inhibited the most (9 participants). The frequency band that was reinforced was mostly the SMR band (12-15 Hz). This was reinforced in 16 people. In three participants beta 1 activity was reinforced, and in seven participants, nothing was reinforced, only inhibited.

**Table 4**
*Frequency bands reinforced/inhibited per participant*

| Participant nr | Frequency bands reinforced | Frequency bands inhibited |
|---|---|---|
| 1 | SMR | Theta & delta |
| 2 | Beta1 | Theta |
| 3 | SMR | Beta2 |
| 4 | SMR | Theta |
| 5 | | Theta & beta2 |
| 6 | SMR | Beta2 |
| 7 | | Alfa |
| 8 | SMR | Beta1 |
| 9 | SMR | |
| 12 | Beta1 | Alfa & beta2 |
| 13 | | SMR & beta2 |
| 15 | SMR | Beta2 |
| 16 | SMR | Delta |
| 17 | SMR | Delta & beta1 |
| 18 | | Beta2 |
| 19 | | Theta & alfa |
| 20 | SMR | Theta |
| 21 | SMR | Beta2 |
| 22 | SMR | Delta |
| 23 | | Theta & beta2 |
| 24 | SMR | Theta |
| 25 | Beta1 | Beta2 |
| 26 | SMR | Theta |
| 27 | | Delta & beta2 |
| 28 | SMR | Delta |
| 29 | SMR | Beta2 |

***Neurofeedback training***

In total 30 sessions were planned in 15 weeks, but after the interim analysis halfway (after 16 sessions) the experiment was ceased. Neurofeedback was done two times a week, preferably keeping at least a day between the sessions. Each session lasted 30 minutes. Except the first and the last minute of the session, audio and visual feedback was given as a reward, in the form of brightening a screen (while watching movies) and hearing an audio click. The first minute served as baseline and during this period the threshold for the feedback was determined. Participants in the sham condition experienced the feedback as comparable to the real neurofeedback condition. The sham feedback was based on a simulated EEG signal and was comparable to real feedback. According to Fuchs et al. (2003) a sham neurofeedback is impossible because it is soon recognized by therapists patients. Hence, in order to make sure that the participants could not guess which condition they were in, a pilot study was conducted to test both conditions. Whereas during clinical neurofeedback training, the trainer is able to see what the client is doing on a separate screen, this study used only one screen which went on standby as soon as the participant's

number was entered. This way neither the participant nor the researcher was able to see which protocols were loaded. In the pilot study, the researchers themselves, already trained in neurofeedback, were not able to correctly guess in which condition –sham or real- the feedback was being given. Since the trained researchers themselves have not been able to identify the correct treatment condition, it has been assumed that the non-trained participants would not be able to identify the true condition they were in. This has also been proven in the follow-up questionnaire given to the participants after the cessation of all neurofeedback treatments since participants guessed their treatment condition at chance level.

***Neurofeedback apparatus***

Neurofeedback training was done using Brainmaster ATLANTIS II models (www.brainmaster.com).

***Interim (final) analysis***

Although 30 to 40 sessions of neurofeedback training are quite common before a permanent result is established, there are early signs of change showing themselves between 5 to 10 sessions (Gunkelman & Johnstone, 2005). Therefore, after 8 weeks of neurofeedback or sham sessions an interim analysis took place in order to identify changes in both the objective and the subjective measures. One student was excluded because she did not attend the last week before the interim analysis due to personal problems. At interim analysis, qEEG was measured, and the two computer tasks, as well as the two questionnaires were administered.

***Data analysis***

To assess differences between participants in the two treatment groups for matching effects a t-test was used.

The data were analyzed for outliers. A repeated measures general linear model was used to test the time (baseline vs interim) x group (neurofeedback vs sham) interaction. Within factors were SSRT (Stop task), SDRT and false alarms (CPT task), STIMP score and ADHD and Attention scores on the ASR.

For exploratory purposes correlations between dependent measures were calculated.

## Results

### *Outliers*

Box plots were made to test for outliers. No data were removed because all data fell within 3 SD of the mean scores.

**Table 5**
*Correlations qEEG patterns and dependent measures*

| | bSSRT | bSDRT | bFA | bATT | bADHD | bSTIMP |
|---|---|---|---|---|---|---|
| **bSMR** | .62* | .61* | -.25 | -.57* | .44 | -.27 |

**= significant at p < .05*

### *Correlation between qEEG patterns and attention and impulsivity measures*

For correlations see table 5.

There is a significant correlation between baseline SMR deficiency (bSMR) as measured by deviation of from population mean in z-scores (*r*=.62) measured by z-scores, and baseline SSRT (bSSRT, Stop task), but not in the expected direction. This is investigated only in the SMR uptraining protocol, because this was the largest group in this experiment. Participants who had more negative z-scores (associated with more attention problems) had faster reaction times. There is no significant correlation between baseline SMR deficiency and baseline false alarms (bFA, dependent measure CPT task). There is a significant correlation between baseline SMR deficiency, (*r*=.61) measured by z-scores, and baseline STDRT (bSDRT, dependent measure CPT task) but not in the expected direction. Participants who had more negative z-scores (associated with more attention problems) had faster reaction times. There is no significant correlation between baseline SMR deficiency, measured by z-scores, and baseline STIMP (bSTIMP). There is no significant correlation between baseline SMR deficiency, measured by z-scores, and baseline ADHD (bADHD, ADHD scale ASR). There is a significant correlation between baseline SMR deficiency, (*r*=-.57) measured by z-scores, and baseline ATT (bATT, Attention scale ASR).

The only correlation found in the expected direction is between SMR deviation and ASR attention problems, with subjects with more SMR deviation having more attention problems. Other correlations not in the expected directions are between SMR deviation and SDRT and between SMR deviation and SSRT. This indicates that subjects with more SMR deviation have less variable reaction times and better motor inhibition.

***The effect of neurofeedback training on objective measures (qEEG)***

SMR activity at baseline was compared with SMR activity at interim. It was investigated whether the treatment group improved more than the sham group (see figure 1).

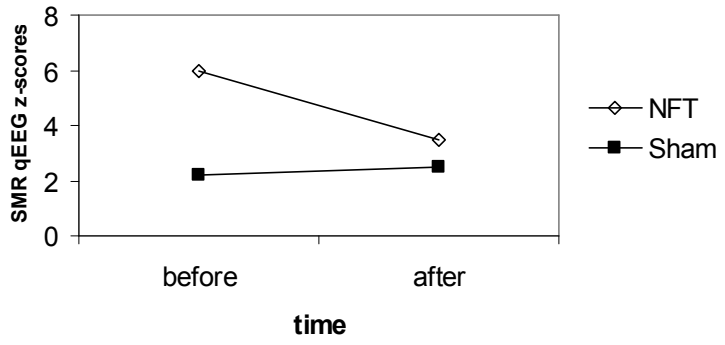


*Figure 1. The effect of neurofeedback on qEEG z-scores.*
The differences in z-scores indicating SMR activity at baseline level (before) and at interim level (after). NFT represents the neurofeedback condition, sham represents the sham condition.

No significant main effect was found for both conditions combined ($F(1,24)=3.841$, $p=.086$). But a trend towards a significant time x group interaction ($F(1,24)=5.086$, $p=.054$) can be seen. However, baseline group differences were significant ($t=2.802$, $p<.05$), so this results should be interpreted with caution.

***The effect of neurofeedback training on objective measures (computer tasks)***

The scores of the average SSRT (Stop task) for both groups combined, increased from the baseline scores (148 ms, SD 17.68) to the interim scores (154.6 ms, SD 23.17) (see figure 2). This main effect of time was not significant ($F(1,24)=1.821$, $p=.202$). The time (baseline vs interim) x group (neurofeedback vs. sham) interaction was not significant either ($F(1,24)=0.43$, $p=.838$).

From baseline to interim the main effect for time on the SDRT scores (reaction time variability, CPT) was not significant ($F(1,24)=3.354$, $p=.079$), although a trend towards significance can be seen (figure 3a). The time (baseline vs. interim) x group (neurofeedback vs sham) interaction between the two groups was also nonsignificant ($F(1,24)=.211$, $p=.650$). The main effect of time on the percentage of false alarms (CPT) was not significant ($F(1,24)=3.295$, $p=.082$), although it did approach significance (figure 3b). The time (baseline vs interim) x group (neurofeedback vs sham) interaction for false alarms was not significant either ($F(1,24)=.295$, $p=.592$).

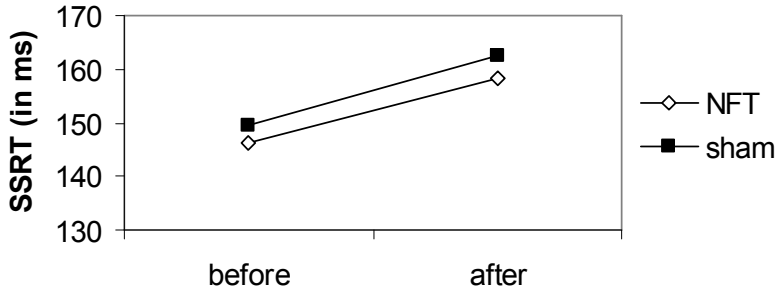


*Figure 2.* The effect of neurofeedback on SSRT (Stop task). Average SSRT of the Stop task at baseline (before) and at interim (after) in milliseconds. NFT represents the neurofeedback condition, sham represents the sham condition.

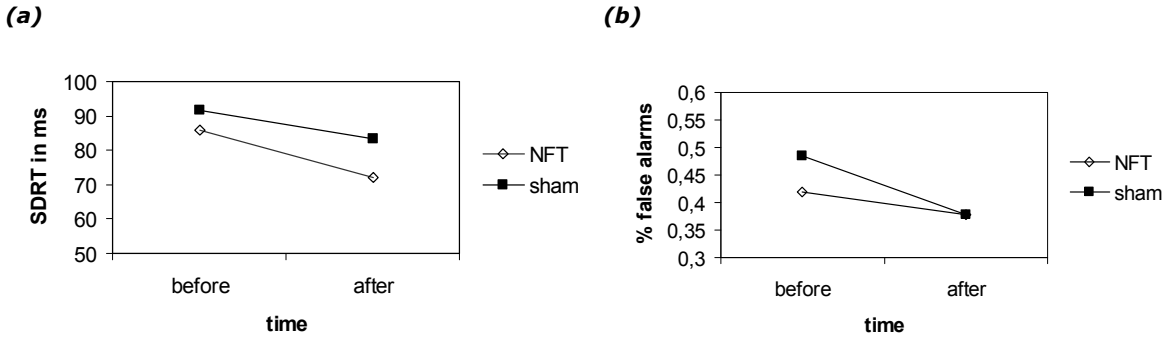


*Figure 3.*The effect of neurofeedback on both CPT measures. *(a)* Average SDRT of the CPT computer task at baseline (before) and at interim (after) in milliseconds. *(b)* Average proportion of false alarms of the CPT computer task at baseline (1) and at interim (2). NFT represents the neurofeedback condition, sham represents the sham condition.

***The effect of neurofeedback training on subjective measures (questionnaires)***

No significant main effect or time x group interaction was found (F(1,24)=0.18, p=.894) and (F(1,24)=.000, p=.998) between the scores of the treatment and the sham condition on STIMP scores (see figure 4).

The ASR attention score decreased significantly from baseline (12.73, SD 5.40) to interim (11.19, SD 5.93) for both groups combined (F(1,24)=4.949, p<.05) (see figure 5a). The group x time interaction was not significant (F(1,24)=1.790, p=.193). The ASR ADHD score at interim (8.58, SD 5.63) was lower compared than at baseline (10.5, SD 4.89) for both groups combined (Figure 5b). This main effect proved to be significant (F(1,24)=5.199, p<.05). The group x time interaction was not significant (F(1,24)=.911, p=.349).

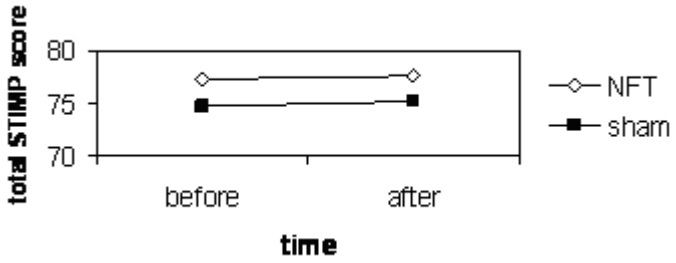


*Figure 4. The* effect of neurofeedback on STIMP score.
Average STIMP score at baseline (before) and at interim (after).
NFT represents the neurofeedback condition, sham represents the sham condition.

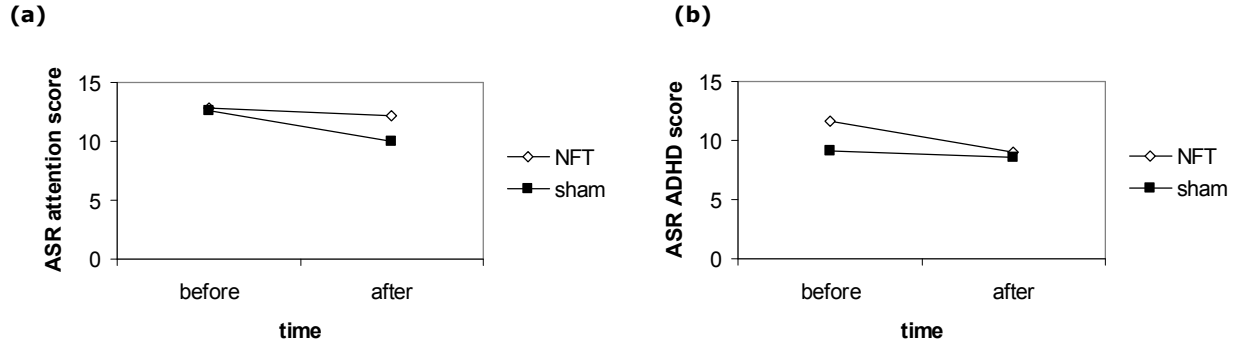


*Figure 5.* The effect of neurofeedback on ASR scores. *(a)* Average ASR attention score at baseline (before) and at interim (after). *(b)* Average ASR ADHD score at baseline (before) and at interim (after). NFT represents the neurofeedback condition, sham represents the sham condition.

## Discussion

This master research was conducted to investigate the effect of neurofeedback training on attention and impulsivity in healthy subjects. To assess changes on subjective and objective level, different questionnaires and computer tasks were used, besides a measurement of participants' qEEG.

A significant correlation between qEEG abnormality and different measures of inattention and high impulsivity was expected (specifically an association between low SMR and problems with attention and impulsivity). A significant correlation in the expected direction was found only between attention problems on the ASR and deviation from the mean in qEEG, suggesting that problems with attention as measured by the ASR are accompanied by a deviation in SMR activity. The correlations between qEEG deviation and CPT and Stop task measures were significant, but not in the expected direction. Contrary to earlier studies (Beauregard & Lévesque, 2006; Monastra et al., 2002), no overall relation between qEEG deviation and measures of attention and impulsivity in the healthy subjects (without ADHD

diagnosis) with problems of attention and impulsivity was observed. This may be attributed to the fact that our participants were healthy students, their problems with attention and impulsivity did not cause clinical impairment.

The treatment group was not able to successfully learn to regulate brain wave activity associated with attention and impulsivity (SMR activity). This is also contradictory to results found in earlier studies (Beauregard & Lévesque, 2006; Lubar et al., 1995; Monastra et al., 2002; Thompson & Thompson, 1998; Vernon et al., 2003). The results did indicate a trend towards the possibility of self-regulation of SMR activity, but this may partly be attributed to fact that the deviation in SMR activity at baseline was greater in the treatment group. This trend towards SMR regulation has no visible effect on objective and subjective measures of attention and impulsivity. Other feedback protocols in this study were given to a too small group of participants to draw conclusions on learning effects. This can possibly be ascribed to the small number of neurofeedback sessions given up to interim measurement.

No interaction effects for neurofeedback were found. This indicates that the treatment group did not make more progress than the sham control group. On objective measures (stop and CPT task) no main effects of training were found. There was a trend towards better CPT performance at interim measurement. This is not due to test-retest practice effects, since on the whole subjects tend to perform more poorly when retested on the CPT due to boredom (Lubar et al., 1995). Boredom however, might have influenced performance on the STOP task, which, in this study, inclines to get worse at post-treatment. This could indicate that participants were not motivated and bored by the task, which was always conducted directly after the CPT task.

As far as subjective measures are concerned, significant improvements in attention and ADHD problems as measured by the ASR were found, the scores decreased significantly in both the treatment and sham group. The STIMP score did not change during the course of this experiment, possibly because state impulsivity is not solely related to problems with impulsivity, but also dependent on other factors.

The main effects found for ASR score and the trend towards CPT improvement in both the treatment and the sham group suggest the improvement may be attributed to the placebo effect. The lack of interaction effects stresses the importance of including control groups while studying the effect of neurofeedback treatment. The improvement found, might be due to trainings effects (concentrating to the brightening of the screen), placebo effects and to the phenomenon of regression to the mean.

From these results it can be concluded that 16 sessions of neurofeedback training have no effect above placebo on measures of attention and impulsivity.

***Limitations and implications of this study***

The selected subclinical population was very small and consisted of healthy subjects, ceiling or floor effects could be the consequence of this, there simply might not have been enough room for improvement. Future research should use participants diagnosed with ADHD to study the benefit of neurofeedback training for this group.

Several different protocols were used in this study which makes it very difficult to disentangle the effects of different treatment protocols. Some feedback protocols might have more effect on attention and impulsivity than others. qEEG selection of participants could address this limitation. By selecting participants diagnosed with ADHD and by matching feedback protocols to qEEG profiles this matter could be addressed.

The type feedback used itself might also have been a problem. While watching a movie other cognitive processes other than attention play an important role. Emotions and story lines might distract the participant from benefiting from the feedback. However, this type of feedback is used in most neurofeedback treatment centers. Other, simpler types of feedback could be more effective in achieving improvement.

Because of ethical reasons, 16 sessions of 20-minute feedback were given during this study at interim measurement. In neurofeedback literature participants are usually given 30 to 45 sessions of neurofeedback, or a smaller number of sessions lasting about 45 minutes (Monastra et al., 2005). In the study of Monastra and colleagues (2002) an average number of 43, 40-minute sessions was needed before participants were able to demonstrate a level of brain activity within 1 standard deviation of their peers. Based on the study mentioned above, the number of sessions in this study might not have been enough to show significant effects on qEEG measures and subjective and objective measures of attention and impulsivity. Through clinical experience therapists at the Neurofeedback Instituut Nederland (Neurofeedback therapists) argue for at least 40 sessions, two times a week, to achieve the significant changes in EEG and behavior. This change should be observable after 30-35 sessions. On the other hand, Gunkelman & Johnstone (2005) have argued that early signs of change are observable somewhere between 5 to 10 sessions. These changes are often not subjectively reported by the patients, but can be seen on objective measures. This study can endorse the first statement by looking at the STIMP scores, but not by looking at the ASR scores. The latter statement has not been confirmed in this study. Nor subjective, nor objective treatment effects have been found after 16 sessions, let alone 5 to 10.

Without appropriate sham controlled studies the effect of neurofeedback treatment above placebo and trainings effects can not be assessed. It could therefore be argued that some of

the current positive findings on the effect on neurofeedback training could be ascribed to a placebo effect. Furthermore, it can be doubted whether other studies that have not been able to find any positive significant effects of neurofeedback training have been published, or that we are dealing with a file-drawer problem here. This would explain the few studies reporting nonsignificant effects of neurofeedback. Hopefully, this study will inspire other researchers to study the effects of neurofeedback training and to investigate whether neurofeedback training truly shows its effect on people with attentional and impulsivity problems above the placebo effect. Currently, passive and active control groups have been used in neurofeedback studies. However, in order to really investigate the effects of neurofeedback training, double blind and sham controlled studies are needed. This study shows that creating a neurofeedback sham condition is possible, based on the fact that neither the participants nor the researchers themselves could guess the condition they were in. We hereby invite fellow researchers to refute our on this study based skepticism about neurofeedback by using a double blind, sham controlled study.

***Conclusion***

Neurofeedback treatment was not found to be effective in enhancing attention and reducing impulsivity above the placebo effect in a healthy population. But these results have to be interpreted with caution because of the limitations of this study, and should not be generalized to the ADHD population. Different patterns of pathology and different processes could play a role in the ADHD population. Therefore more studies using double blind, sham controlled should investigate this matter.

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