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Is EEG-biofeedback an Effective Treatment in Autism Spectrum Disorders? A Randomized Controlled Trial

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Abstract EEG-biofeedback has been reported to reduce symptoms of autism spectrum disorders (ASD) in several studies. However, these studies did not control for nonspecific effects of EEG-biofeedback and did not distinguish between participants who succeeded in influencing their own EEG activity and participants who did not. To overcome these methodological shortcomings, this study evaluated the effects of EEG-biofeedback in ASD in a randomized pretest–posttest control group design with blinded active comparator and six months follow-up. Thirty-eight participants were randomly allocated to the EEG-biofeedback, skin conductance (SC)-biofeedback or waiting list group. EEG- and SC-biofeedback sessions were similar and participants were blinded to the type of feedback they received. Assessments pre-treatment, post-treatment, and after 6 months included parent ratings of symptoms of ASD, executive function tasks, and 19-channel EEG recordings. Fifty-four percent of the participants significantly reduced delta and/or theta power during EEG-biofeedback sessions and were identified as EEG-regulators. In these EEG-regulators, no statistically significant reductions of symptoms of ASD were observed, but they showed significant improvement in cognitive flexibility as compared to participants who managed to regulate SC. EEG-biofeedback seems to be an applicable tool to regulate EEG activity and has specific effects on cognitive flexibility, but it did not result in significant reductions in symptoms of ASD. An important finding was that no nonspecific effects of EEG-biofeedback were demonstrated.

Keywords EEG-biofeedback · Skin conductance · Autism spectrum disorders

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Introduction

Autism spectrum disorders (ASD) are characterized by qualitative abnormalities in social behavior and communication skills and restricted, repetitive, and stereotyped patterns of behavior, interests, and activities. Currently, many behavioral training programs exist to reduce symptoms of ASD. Behavioral training programs based on applied behavior analysis (ABA) are evidence based treatments that have been shown to be effective in the reduction of the core features of ASD (Peters-Scheffer et al. 2011). However, such behavioral training programs often do not entirely take away the symptoms of ASD, are expensive and demanding to administer, and take years to complete. Medication may also play a role in the management of associated symptoms of ASD, such as irritability, rigidity, hyperactivity, impulsivity, and inattention, but side-effects may compromise therapeutic benefits (King and Bostic 2006). In the search of alternative treatment options for children with ASD, electroencephalography (EEG)-biofeedback emerged as a promising option for reducing symptoms of ASD.

The rationale of applying EEG-biofeedback to children with ASD is based on findings from EEG studies. These studies have revealed abnormal patterns of EEG activity in individuals with ASD as compared to normal controls, such as increased delta and theta power (Chan et al. 2007; Murias et al. 2007; Pop-Jordanova et al. 2010), decreased alpha power (Chan et al. 2007; Murias et al. 2007), and increased beta (Murias et al. 2007) and gamma power (Orekhova et al. 2007). Another consistent finding concerns local over-connectivity and long-distance under-connectivity in individuals with ASD (Wass 2011). Finally, dysfunctions of the mirror neuron system were reflected in the EEGs of children with ASD (Oberman et al. 2005). Using EEG-biofeedback to influence the activation of neural mechanisms that underlie deviating EEG activity was proposed to result in reductions of symptoms of ASD (Demos 2005; Hammond 2006; Sterman and Egner 2006).

Previous studies that investigated the effects of EEG-biofeedback in children with ASD revealed improvement in social interactions and verbal and non-verbal communication skills after EEG-biofeedback (Coben and Padolsky 2007; Jarusiewicz 2002; Kouijzer et al. 2009b, 2010; Scolnick 2005; Sichel et al. 1995; Thompson et al. 2010). In addition, executive functions improved after EEG-biofeedback (Coben and Padolsky 2007; Kouijzer et al. 2009b, 2010). Finally, EEG-biofeedback was found to successfully influence several EEG frequency bands, such as theta power and low beta power (Scolnick 2005; Sichel et al. 1995; Kouijzer et al. 2009b).

However, many of the previous studies have methodological shortcomings which make it difficult to draw the conclusion that EEG-biofeedback for individuals with ASD is evidence based. That is, the apparent effects of EEG-biofeedback might have been produced by nonspecific (placebo) effects of EEG-biofeedback such as treatment expectancy, implicit training of attention, and intensive one-to-one contact with the therapist (Heinrich et al. 2007). Furthermore, previous studies did not differentiate between participants who succeed in regulating their own EEG activity, i.e. EEG-regulators, and participants who do not, i.e. EEG-non regulators. However, a difference in outcome can be expected between these two groups of participants. That is, a higher level of improvement should be expected in EEG-regulators as compared to EEG-non regulators (Lubar et al. 1995).

This study aimed to control for nonspecific effects of EEG-biofeedback and took into account differences between EEG-regulators and EEG-non regulators. In order to control for nonspecific effects of EEG-biofeedback, the effects of EEG-biofeedback were compared with the effects of skin conductance (SC)-biofeedback and a waiting list group. SC is the amount of electrical current the skin allows to pass after a very small electrical current is applied to the skin (Peek 2003). SC-biofeedback can be applied in the treatment of hypertensive patients in order to relax patients (McGrady and Linden 2003). In the present study, EEG- and SC-biofeedback sessions were similar and participants were not aware of the type of biofeedback they received. EEG-biofeedback aimed at reducing absolute power in the frequency band that showed maximal deviations from normality in the participant's pre-treatment EEG recording; SC-biofeedback aimed at reducing SC. Participants of the waiting list group received no treatment.

In accordance with earlier research, we hypothesized that EEG-biofeedback has specific benefits for children and adolescents with ASD. That is, we expected EEG-biofeedback to change EEG activity in the direction of normality in a substantial subsample of the participants, i.e. the EEG-regulators (cf. Hanslmayr et al. 2005; Kouijzer et al. 2010; Kropotov et al. 2005; Lubar et al. 1995). We expected EEG-regulators to show reduced symptoms of ASD, improved executive functions, and altered 19-channel EEG recordings after EEG-biofeedback, as compared to participants who regulated the SC-signal. Based on earlier research, our second hypothesis is that EEG-biofeedback has no nonspecific benefits for children and adolescents with ASD. That is, EEG- and SC-non regulators were not expected to improve in symptoms of ASD, executive functions, and 19-channel EEG, as compared to participants of the waiting list group.

Methods

Participants

A total of 320 school files of a secondary school for special education were screened to select participants for the present study. Inclusion criteria were an age between 12 and 18 years, an IQ-score of 80 or above and the presence of autistic disorder, Asperger disorder or PDD-NOS as clinically diagnosed by a certified child psychiatrist or health care psychologist, according to the DSM-IV-TR criteria (American Psychiatric Association 2000). Excluded were students with a history of severe brain injury or comorbid diagnoses such as ADHD and epilepsy as diagnosed by a certified child psychiatrist or health care psychologist. Eighty-seven students were selected and invited to participate in the study. Thirty-eight students voluntarily signed in for participation in the study. These participants were randomly assigned to the EEG-biofeedback, SC-biofeedback or waiting list group. There were no pre-treatment statistical differences between these groups concerning demographic and clinical variables (see Table 1).

The diagnoses of 35 participants were confirmed by the results of the Autism Diagnostic Interview revised (ADI-R;

Table 1 Demographic and clinical characteristics (mean ± standard deviation) of the EEG-biofeedback, SC-biofeedback, and waiting list group and F and p values of statistical tests investigating differences between groups

	EEG-group $n = 13$	SC-group $n = 12$	Waiting list $n = 13$	F	p
Sex (*female/male*)	3/10	3/9	2/11		
Age (*years; months*)	15;3 ± 1;5	14;5 ± 1;5	15;9 ± 1;5	2.683	.082
Medication use (*yes/no*)	2/11	1/11	4/9		
IQ	104.1[1] ± 15.8	111.5[2] ± 14.5	109.8[3] ± 15.4	1.905	.178
Diagnosis (*autism/Asperger/PDD-NOS*)	3/4/6	1/6/5	4/2/7		
ADI reciprocal social interaction	15.8[4] ± 5.3	14.3[5] ± 7.4	14.6[6] ± 7.1	.221	.641
ADI communication	12.6[7] ± 5.5	13.5[8] ± 4.7	11.7[9] ± 5.2	.198	.659
ADI restricted, repetitive and stereotyped behavior	3.9[10] ± 3.1	3.5[11] ± 2.8	3.6[12] ± 3.1	.004	.949
SCQ total score	17.31[13] ± 6.23	15.25[14] ± 4.86	15.67[15] ± 5.37	1.686	.200
SCQ reciprocal social interactions	6.54[16] ± 3.31	4.75[17] ± 2.99	5.00[18] ± 2.76	1.286	.290
SCQ communication skills	7.38[19] ± 1.76	6.83[20] ± 2.04	7.25[21] ± 2.86	1.772	.185
SCQ restricted and stereotyped behavior	2.38[22] ± 1.85	1.75[23] ± 1.86	2.58[24] ± 1.68	.704	.502

IQ ranges are 80–119[1], 85–134[2], and 85–131[3]. Ranges of ADI scores are 6–25[4], 4–23[5], 3–26[6]; 2–22[7], 5–20[8], 3–20[9]; 0–11[10], 1–10[11], 3–12[12]. Ranges of SCQ scores are 9–29[13], 6–21[14], 2–21[15]; 2–12[16], 0–10[17], 3–9[18]; 4–10[19], 3–9[20], 1–9[21]; 0–6[22], 0–5[23], 3–;5[24]

Lord et al. 1994). All interviews were conducted by a certified clinician. On the ADI-R, autistic disorder and Asperger disorder are indicated if participants meet the criteria on all three subscales, i.e. reciprocal social interaction (cut-off $\geq$ 10), communication (cut-off $\geq$ 7), and restricted, repetitive, and stereotyped behavior (cut-off $\geq$ 3). Definite delays in language development are required for autistic disorder, but not for Asperger disorder. In the case of PDD-NOS, the criteria for only two of the three domains must be attained (cf. Verté et al. 2006). Diagnoses of three participants were not in accordance with the ADI-R criteria for ASD, but their scores on the Social Communication Questionnaire met the criteria for ASD (cut-off $\geq$ 15). Therefore, these participants were not removed from the sample. Eight participants used medication, i.e. Risperdal (anti-psychotic; $n = 4$), Risperdal and Fluoxetine (anti-depressant; $n = 1$), Fluvoxamine (anti-depressant; $n = 1$), Dipiperone (anti-psychotic; $n = 1$), and Enalapril (ACE inhibitor that lowers blood pressure; $n = 1$). The study was approved by the local medical-ethics committee. Written informed consent was obtained from the parents of all participants. Participants themselves provided written assent.

Measures

Symptoms of ASD

The version 'Current situation' of the Social Communication Questionnaire (SCQ; Rutter et al. 2003; translated into Dutch by Warreyn et al. 2003) is a 40-item questionnaire related to ADI-R criteria for ASD. Response categories are 'yes' and 'no'. The outcome measures are scores for the subscales 'reciprocal social interactions' (range 0–15), 'communication' (range 0–13), and 'restricted, repetitive, and stereotyped behavior' (range 0–8). A cut-off score of 15 or higher is indicative of a diagnosis of autistic disorder. Psychometric properties of the SCQ have been evaluated as good in several studies, i.e. sensitivity is .96 and specificity is .80 (Berument et al. 1999; Bolte et al. 2000). Parent ratings were collected at three time points: pre- and post-training and at follow-up after 6 months. The SCQ total score (range 0–36) constituted the primary outcome measure of this study.

Clinical Improvement

The 'improvement scale' of the Clinical Global Impression (CGI) requires a clinician to assess a client's overall symptomatic change as compared to baseline (Guy 1976). The CGI is a 7-point scale ranging from 'very much improved' to 'very much worse'. It has acceptable validity and was demonstrated to be sensitive to change (Berk et al. 2008). The CGI was filled out by the EEG- or SC-biofeedback therapist after the final session.

Cognitive Flexibility

The Trail Making Test (TMT; Reitan 1956) was used to measure cognitive flexibility. The TMT has a test–retest reliability of .54 (Echemendia et al.1999). Participants have to locate and connect 26 numbers (part A), 26 characters (part B), and 26 numbers and characters in the 1-A-2-B-3-C-order (part C), as soon as possible and in the right order. The score for cognitive flexibility is represented by the time in seconds that is needed to finish part C minus the time in seconds that is needed to finish part B.

Inhibition

The Stroop task (Stroop 1935) was used to measure inhibition. The Stroop task has a test–retest reliability of .67 (Franzen et al. 1987). Participants have to read aloud 100 words (part A), the color of 100 colored rectangles (part B), and the color of the ink of 100 written incongruent color names (part C) as soon as possible. The ability to inhibit reading aloud the written word in part C is represented by the interferential time, i.e., the time in seconds that is needed to finish part C minus the time in seconds that is needed to finish part B.

Planning

The Tower of London (TOL) was used to measure planning skills. The TOL has a test–retest reliability of .66 (Kovács 2005a). In this task, participants have to copy a construction of blocks and bars by moving three prearranged colored blocks along three bars of different lengths. The score for planning is calculated by dividing the number of correctly solved items by the maximum score of 12, times 100.

Attention

The Test of Sustained Selective Attention (TOSSA) was used to measure attention. The TOSSA has a test–retest reliability of .86 (Kovács 2005b). In this task, participants have to respond to sets of three beeps while ignoring sets of two or four beeps during 8 min. An attention score was calculated by dividing the number of hits by the total amount of items, times 100.

Working Memory

The subtest Digit Span was adopted from the Wechsler Intelligence Scale for Children, 3rd version, Dutch version (WISC-III-NL; Kort et al. 2002) to measure working memory. The test–retest reliability of this subtest is .83 (Rowe 2005). This task requires participants to repeat series of numbers of increasing length in forward and reverse order. The series are verbally presented. The score for working memory is the amount of correctly repeated series (maximum is 30).

19-Channel EEG

A Mitsar EEG 201 System was used for recording and digitizing EEG. Data were acquired using a stretchable electrode cap containing 19 sensors and ground (AFz), according to the International 10/20 System (Jasper 1958). Two ear clips were used as reference electrodes. Impedance was kept below 5 kΩ. Data were collected for 3 min in rest and task conditions. In rest conditions, participants were instructed to sit still and relax with their eyes open or closed. Task conditions included a 2-back task, self referential tasks, and movement task. In the 2-back task, participants pressed a button when they observed a number on a computer screen that was similar to the number that was shown two numbers before. This task was included to investigate EEG activity during cognitive demanding conditions (Gevins et al. 1997). In the self referential tasks, participants judged on a five point scale how applicable a series of personality traits were to themselves (part 1) or the Dutch queen (part 2) or they counted how often the letter 'e' appeared in each word (part 3; based on Rogers et al. 1977). This task was included to investigate EEG activity during tasks involving self- and other-referential processing (Gusnard et al. 2001). In the movement task, participants had to open and close their fist while watching their own movement (Oberman et al. 2005). This task was included to investigate EEG activity during movement. In contrast to our expectations, analyses of the EEG data of all conditions with MANOVA revealed no differences between conditions in any of the frequency bands delta (1–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), smr (12–15 Hz) low beta (15–18), and high beta (18–30) or 19 scalp sites (p's $> .05$). Consequently, for each frequency band and scalp site, EEG power values were averaged over conditions. These averages were used in the analyses to compare EEG- and SC-regulators and EEG- and SC-non regulators and waiting list controls.

Treatment Expectancy

A 5-item questionnaire on treatment expectancy was developed by the authors of this study and was based on Borkovec and Nau (1972). Response was given on a 9-point scale ranging from 'yes, completely' to 'no, completely not'. An example of an item is 'Do you think biofeedback will reduce your autistic symptoms?'. The outcome measure was the mean score on all items (range 1–9). This questionnaire was filled out by participants and parents before participants were randomly allocated to one of the research groups, but after they received oral and written information about biofeedback and the design of the study.

Procedure

A randomized pretest–posttest control group study design with blinded active comparator group and 6 months follow-up was used to investigate the effects of EEG-biofeedback. The set-up of EEG- and SC-biofeedback treatment was similar. The only difference was that in the EEG-biofeedback group, feedback was provided on EEG

and in the SC-biofeedback group, feedback was provided on SC. Both EEG- and SC-biofeedback treatment included 40 individual sessions that were provided twice a week and at the school of the participants. Pre-treatment assessment took place about 1 week before the first session. Post-treatment and follow-up assessments were done about 1 week after the last session and after 6 months respectively. Participants and parents were blinded for treatment allocation to EEG- or SC-biofeedback groups, but not for the waiting list group. Both treatments were introduced as experimental, but promising treatments for individuals with ASD. Participants of the waiting list group were given the opportunity to have biofeedback treatment after the 3 months follow-up was completed.

A Nexus-4 amplifier and recording system was used for EEG- and SC-biofeedback sessions. The scalp location, mastoid, and fingers where electrodes were applied were prepared with alcohol and an abrasive gel. Ag/AgCl disposable snap-on electrodes were used as skin conductance electrodes and EEG-reference and -ground electrodes applied at the mastoid; EEG disc electrodes were used as active electrodes and placed on the scalp. Impedance was checked before each session started and kept below 5 kΩ. During EEG- and SC-biofeedback sessions, participants sat in front of a computer screen while EEG and SC were measured concurrently. Participants were instructed to sit as still as possible in order to avoid movement artefacts and an EMG inhibit was set at 15 microvolt. Each session comprised seven 3-min intervals of EEG- or SC-biofeedback, separated by 1-min rest intervals. The task was to decrease the bar graph on the computer screen. This bar graph represented EEG activity in the EEG-biofeedback group and SC in the SC-biofeedback group. If the bar graph moved below the criterion line, participants were rewarded with a counter and a film clip with sound that was selected to be of interest to the participants. Different film clips and sounds were used in each biofeedback interval. The criterion line was set manually in such a way that rewards were provided in 50–80 % of the time. The exact reward rate was determined by the therapist and was adjusted to the participant's motivation. That is, participants who were not motivated to decrease the bar graph and to complete all neurofeedback sessions received more feedback in order to keep them in the study. The type and amount of feedback provided to participants in EEG- and SC-biofeedback sessions and to regulators and non regulators were similar.

SC feedback was derived from electrodes attached to the participants' index and ring fingers of their non-dominant hand. EEG feedback (reference: mastoids, bandwidth: 1–30 Hz, sampling rate: 250 Hz) was derived from an electrode attached to the scalp of the participant, i.e. Cz ($n = 8$) or FCz ($n = 5$). The electrode location and the EEG frequency band were individually defined by comparing each participant's pre-treatment 19-channel EEG recording to a normative database (i.e., Neuroguide). This database produces the deviation from normality for each single Hertz bin per electrode site. The electrode location and the EEG frequency band that showed the largest deviation from normality were selected for EEG-biofeedback. The Neuroguide database has a satisfactory level of reliability and construct and content validity (Thatcher et al. 2003).

Statistical Analyses

In order to analyze the data that were recorded during EEG- and SC-biofeedback sessions, eye blinks and other artifacts were manually removed from the EEG and SC signals. In line with our intention to distinguish between regulators and non regulators to EEG- and SC-biofeedback, we used Spearman correlations to calculate whether participants showed a significant negative correlation between the mean amplitude of the EEG (EEG-biofeedback group) or SC (SC-biofeedback group) signal that was used during the biofeedback sessions and the number of sessions. Participants who showed a negative correlation between the EEG or SC amplitude and the number of sessions were referred to as EEG- or SC-regulators; participants who did not show such a correlation were referred to as EEG- or SC-non regulators (cf. Hanslmayr et al. 2005; Kouijzer et al. 2010; Lubar et al. 1995).

Separate analyses were conducted to investigate specific and nonspecific effects of EEG-biofeedback on symptoms of ASD and executive functions. In order to investigate specific effects of EEG-biofeedback, the data of EEG- and SC-regulators were compared with a repeated measures MANOVA with within-subjects factor Time (3 levels: pre-, post-, and follow-up-treatment) and between-subjects factor Group (2 levels: EEG- and SC-regulators). In order to investigate nonspecific effects of EEG-biofeedback, the data of EEG- and SC-non regulators and waiting list controls were compared with a repeated measures MANOVA with within-subjects factor Time (3 levels: pre-, post-, and follow-up treatment) and between-subjects factor Group (3 levels: EEG- and SC-non regulators and waiting list controls). Specific effects of EEG-biofeedback on clinical improvement were examined with ANOVA with between-subjects factor Group (2 levels: EEG- and SC-regulators). Nonspecific effects of EEG-biofeedback on clinical improvement were examined with ANOVA with between-subjects factor Group (3 levels: EEG- and SC-non regulators and waiting list controls).

In order to analyze 19-channel EEG data that were collected pre- and post-treatment and at follow-up after

6 months, eye blinks and other artifacts were manually removed from the raw data. The data were explored by comparing the data to the Neuroguide database. Subsequently, the data were processed with fast Fourier transformations and averages were calculated per electrode and frequency band (delta: 1–4 Hz, theta: 4–8 Hz). In order to identify specific effects of EEG-biofeedback, the data of EEG- and SC-regulators were compared with a repeated measures MANOVA with within-subjects factors Time (3 levels: pre-, post-, and follow-up-treatment), Electrode (3 levels: Fz, Cz, and Pz), and Frequency band (2 levels: delta and theta) and between-subjects factor Group (2 levels: EEG- and SC-regulators). In order to identify nonspecific effects of EEG-biofeedback, the data of EEG- and SC-non regulators and waiting list controls were compared with a repeated measures MANOVA with within-subjects factors Time (3 levels: pre-, post-, and follow-up-treatment), Electrode (3 levels: Fz, Cz, and Pz), and Frequency band (2 levels: delta and theta) and between-subjects factor Group (3 levels: EEG- and SC-non regulators).

Effects of treatment expectancy were investigated with ANOVA with between-subjects factor Group (3 levels: EEG- and SC-biofeedback and waiting list controls). In order to exclude effects of expectancy on regulating EEG- and SC-biofeedback, ANOVA with between-subjects factors Group (2 levels: EEG- and SC-biofeedback) and Response (2 levels: regulators and non regulators) was performed. Separate analyses were applied for participant and parent ratings of treatment expectancy.

All analyses were carried out at the pre-specified two-sided alpha level of .05.

Results

EEG Database Comparisons

The 19-channel EEG recordings of all participants were compared to the Neuroguide database. This comparison revealed excessive delta (1–4 Hz) and theta (4–8 Hz) activity in frontal and central areas of the brain in all participants. The outcomes of the database comparisons were used to define the scalp site and frequency band that were used during EEG-biofeedback sessions. More specifically, the pre-treatment EEG recording of each participant in the EEG-biofeedback group showed maximal deviations from normality at Cz ($n = 8$) or FCz ($n = 5$). These deviations were found in delta and/or theta power (2–9 Hz), resulting in the following frequency bands to be inhibited during EEG-biofeedback treatment: 2–7 Hz ($n = 4$), 3–7 Hz ($n = 2$), 3–9 Hz ($n = 1$), 4–7 Hz ($n = 4$), 5–7 Hz ($n = 1$), and 5–9 Hz ($n = 1$).

Evaluation of the EEG- and SC-Biofeedback Treatment

Number of Sessions

Because of frequent school absence due to illness, tiredness or overstimulation during school days, four participants of the EEG-biofeedback group and five participants of the SC-biofeedback group did not complete the 40 EEG- or SC-biofeedback sessions. In stead, these participants completed 34, 33, 29, and 23 EEG-biofeedback sessions and 38, 35, 32, 31, and 29 SC-biofeedback sessions, respectively. Data of both participants who completed 40 sessions and participants who did not were included in the analyses.

Blinding

There were no statistical differences between the EEG- and SC-biofeedback groups regarding the success in blinding. Fifty-eight percent of the participants of both groups thought they had received a combination of EEG- and SC-biofeedback; 33 % of the EEG-biofeedback group and 42 % of the SC-biofeedback group thought they had received EEG-biofeedback.

Identification of Regulators and Non Regulators

Analyzing mean EEG amplitudes during EEG- and SC-biofeedback sessions indicated that seven participants of the EEG-biofeedback group showed a negative correlation of delta and/or theta power across sessions, r's $-.356$ to $-.803$, p's $< .05$. In accordance with Lubar and colleagues (2005) and Kouijzer et al. (2010), these participants were identified as being able to regulate delta and/or theta power during the EEG-biofeedback sessions and referred to as EEG-regulators. For none of the participants in the SC-biofeedback group a correlation between delta and/or theta power across sessions was found.

Analyzing mean SC amplitudes during EEG- and SC-biofeedback sessions indicated that eight participants of the SC-biofeedback group showed a negative correlation of SC across sessions, r's $-.365$ to $-.840$, p's $< .05$. These participants were identified as SC-regulators. Five participants of the EEG-biofeedback group (one EEG-regulator, four EEG-non regulators) showed such a correlation, r's $-.472$ to $-.741$, p's $< .05$. This was not unexpected, since familiarity with EEG-biofeedback might enhance relaxation and thus decrease SC.

Symptoms of ASD

Table 2 presents SCQ total scores of EEG- and SC-regulators and -non regulators and waiting list controls. In order

Table 2 Scores of the Social Communication Questionnaire (SCQ) and executive function tasks (mean scores ± standard deviations) from EEG- and SC-regulators and -non regulators and waiting list controls

	EEG-biofeedback group						SC-biofeedback group		
	Regulators			Non regulators			Regulators		
	Pre	Post	Follow-up	Pre	Post	Follow-up	Pre	Post	Follow-up
SCQ total score	18.14 ± 6.04	17.14 ± 6.41	13.60 ± 3.58	16.33 ± 6.89	15.33 ± 7.60	15.33 ± 6.01	14.71 ± 5.20	14.38 ± 4.57	11.50 ± 4.64
Cognitive flexibility[1]	23.57 ± 17.83	11.57 ± 13.58	2.85 ± 2.91	14.50 ± 14.84	8.75 ± 10.56	11.25 ± 11.59	12.50 ± 5.86	18.38 ± 12.31	11.12 ± 7.84
Inhibition[1]	40.14 ± 15.51	29.86 ± 10.95	32.14 ± 21.42	32.67 ± 8.94	43.00 ± 20.12	42.75 ± 10.15	22.00 ± 9.85	30.13 ± 12.07	27.87 ± 9.64
Planning[2]	66.04 ± 15.24	69.86 ± 12.87	79.60 ± 4.81	68.98 ± 10.84	54.35 ± 22.59	59.05 ± 19.32	59.67 ± 27.20	84.96 ± 15.19	85.88 ± 13.68
Attention[2]	73.66 ± 23.35	83.66 ± 14.01	82.77 ± 18.44	80.90 ± 23.44	81.20 ± 13.84	88.33 ± 7.01	93.97 ± 5.23	90.83 ± 6.23	88.52 ± 8.47
Working memory[2]	13.57 ± 1.90	14.00 ± 3.42	14.57 ± 3.40	14.83 ± 2.40	15.75 ± 2.06	16.75 ± 2.87	18.00 ± 2.00	17.88 ± 4.70	19.00 ± 5.15

	SC-biofeedback group			Waiting list controls		
	Non regulators					
	Pre	Post	Follow-up	Pre	Post	Follow-up
SCQ total score	16.34 ± 4.86	17.67 ± 5.69	11.00 ± 5.07	15.67 ± 5.37	15.09 ± 6.22	13.11 ± 6.75
Cognitive flexibility[1]	8.75 ± 10.56	11.25 ± 11.59	4.33 ± 5.86	12.46 ± 11.37	15.69 ± 25.64	15.43 ± 13.65
Inhibition[1]	43.00 ± 20.12	42.75 ± 10.15	22.00 ± 9.85	39.85 ± 21.16	36.46 ± 9.36	31.57 ± 15.88
Planning[2]	54.35 ± 22.59	59.05 ± 19.32	59.67 ± 27.20	70.79 ± 3.59	81.04 ± 1.74	81.57 ± 16.72
Attention[2]	81.20 ± 13.84	88.33 ± 7.01	93.97 ± 5.23	78.47 ± 3.98	82.72 ± 4.36	75.54 ± 32.52
Working memory[2]	15.75 ± 2.06	16.75 ± 2.87	18.00 ± 2.00	12.54 ± 3.33	13.23 ± 4.21	13.57 ± 4.65

[1] Lower scores indicate improvement; [2] Higher scores indicate improvement

to identify specific effects of EEG-biofeedback, the SCQ total scores (primary outcome measure) of the EEG- and SC-regulators were compared, but no significant effects were found. Analysis of the SCQ subscales also revealed no significant effects.

In order to identify nonspecific effects of EEG-biofeedback, the SCQ scores as rated by the parents (primary outcome measure) of the EEG- and SC-non regulators and the waiting list controls were compared, but no significant effects were found.

Clinical Improvement

Analyses comparing clinical improvement ratings of EEG- and SC-regulators showed no specific clinical improvement after EEG-biofeedback. Comparing clinical improvement ratings of EEG- and SC-non regulators and waiting list controls did not demonstrate nonspecific differences between the global improvement ratings of these participants.

Executive Functions

An overview of the scores of executive function tasks from EEG- and SC-regulators and -non regulators and waiting list controls is presented in Table 2. In order to identify specific effects of EEG-biofeedback on executive functions, the scores on executive function tasks of the EEG- and SC-regulators were compared and revealed a significant interaction effect, $F(10,20) = 6.320, p = .002, \eta\rho^2 = .927$. Univariate analyses revealed a significant interaction effect for cognitive flexibility, $F(2,15) = 5.455$, $p = .017$, $\eta\rho^2 = .421$, but not for inhibition, planning, attention, and working memory. Subsequent analyses comparing pre- and post-treatment measures revealed that EEG-regulators improved in cognitive flexibility, $F(1,6) = 16.346$, $p = .007$, $\eta\rho^2 = .731$, but SC-regulators did not. Analyses comparing post- and follow-up-treatment measures revealed no significant interaction, suggesting maintenance of the effect in cognitive flexibility 6 months later.

In order to identify nonspecific effects of EEG-biofeedback on executive functions, the scores on executive function tasks of the EEG- and SC-non regulators and the waiting list controls were compared, but revealed no significant effects.

19-Channel EEG

19-channel EEG data from EEG- and SC-regulators were compared in order to identify specific effects of EEG-biofeedback, but revealed no significant effects. 19-channel EEG data from EEG- and SC-non regulators and waiting list controls were analyzed in order to identify nonspecific effects of EEG-biofeedback, but revealed no significant effects.

Treatment Expectancy

Analyses of data on treatment expectancy revealed no statistical differences between the EEG- and SC-biofeedback and waiting list group. Comparing treatment expectancy of EEG- and SC-regulators and -non regulators revealed no differences in treatment expectancy. Using treatment expectancy as a covariate in any analysis did not reveal significant effects.

Treatment of the Control Groups

Participants of the waiting list group were promised to receive EEG- or SC-biofeedback after the study was completed. However, due to the disappointing effects on the symptoms of ASD, the board of the participating school decided not to continue with EEG- or SC-biofeedback. None of the participants of the waiting list groups asked for additional treatment with biofeedback.

Discussion

The present study showed that EEG-biofeedback resulted in a negative correlation of delta and/or theta power across sessions in 54 % of the participants, i.e. the EEG-regulators. In these EEG-regulators, EEG-biofeedback resulted in a specific effect in cognitive flexibility. Importantly, the improvement in cognitive flexibility was found to be sustained after 6 months, implicating a long lasting effect of the treatment. There were no specific effects of EEG-biofeedback on symptoms of ASD and 19-channel EEG recordings. No nonspecific effects of EEG-biofeedback were identified on symptoms of ASD, executive functions or 19-channel EEG recordings. The absence of nonspecific effects of EEG-biofeedback implies that treatment expectancy, implicit training of attention, and intensive one-to-one contact with the therapist did not result in improvement in symptoms of ASD, executive functions or 19-channel EEG.

The improvement in cognitive flexibility that was found after EEG-biofeedback is consistent with the results of previous studies that also found improved cognitive flexibility after EEG-biofeedback (Coben and Padolsky 2007; Kouijzer et al. 2009b; Kouijzer et al. 2010). Improvement in cognitive flexibility seems to be a specific consequence of the successful application of EEG-biofeedback and can not be explained by implicit attention training, intensive one-to-one contact with the therapist during EEG-biofeedback or treatment expectancy. That is, no improvement

in cognitive flexibility was found in EEG-non regulators and in participants of the SC-biofeedback group, who had similar quantities of attention training, one-to-one contact with the therapist, and expectancies of the treatment.

Cognitive flexibility in the present study was measured with the TMT, in which participants had to locate and connect as quickly as possible and in the right order 26 numbers (part A), 26 characters (part B), and 26 numbers and characters in the 1-A-2-B-3-C-order (part C). In a study investigating the construct validity of the TMT (Sánchez-Cubillo et al. 2009), it was concluded that this task primarily reflects task switching abilities. Task switching, or cognitive flexibility, is defined as the ability to shift to a different thought or action according to situational changes (Hill 2004). In the TMT, cognitive flexibility is defined as the additional time that is needed to shift between numbers and characters (part C) compared to the time that is needed to connect characters only (part B). EEG-regulators showed a 49 % reduction in time that was required to complete part C, as compared to the time acquired for part B. Six months after EEG-biofeedback had ended the improvement in TMT performance was found to have increased even further to an 88 % reduction of the original processing time of part C. These percentages are in accordance with previous studies that showed a similar improvement in TMT performance following EEG-biofeedback, i.e. 57 % (Kouijzer et al. 2009b) and 43 % (Kouijzer et al. 2010), relative to the TMT performance of a control group. Follow-up studies after 6 and 12 months showed further reductions of TMT performance time to 79 % (Kouijzer et al. 2010) and 60 % of the original completion time of part C (Kouijzer et al. 2009a). Multiple studies thus suggest substantial improvement in cognitive flexibility as measured with the TMT following EEG-biofeedback.

The pre-treatment cognitive flexibility skills of the participants of this study were different between groups. Although not significant, the pre-treatment level of cognitive flexibility of EEG-regulators (23.57) was lower (i.e., higher scores reflect lower capacity) than the pre-treatment levels of EEG-non regulators (14.50), SC-regulators (12.50), SC-non regulators (8.75) and waiting list controls (12.46). The reason for this initial difference is unclear. Varying averages between groups might reflect large individual differences in cognitive flexibility that are expressed because of the relatively small group sizes. One other interesting alternative speculation is that individuals with low cognitive flexibility may actually be more responsive to EEG-biofeedback treatment, and thus turned out to become EEG regulators. In line with this suggestion, studies have found a clear relation between theta power and cognitive flexibility in visual attention (Kostandov 2010), a main component of the TMT. In future studies with larger sample sizes, it would be interesting to further investigate the relation between cognitive flexibility skills and EEG regulation abilities.

Deficient cognitive flexibility is found as one of the characteristic deficits in ASD (e.g. Hughes et al. 1994). Children and adolescents with ASD often lack the ability to adapt to continuously changing social situations, which makes it hard to adjust their behavior to varying circumstances. As a result, children and adolescents with ASD often become distressed in changing situations and insist on sameness. Deficient cognitive flexibility is furthermore associated with a lack of improvement in social adaptive functioning following treatment (Berger et al. 2003). Hence, the positive effect of EEG-biofeedback on cognitive flexibility could be of importance to the lives of children and adolescents with ASD. For instance, they might be better able to deal with changing situations at home and in school and their abilities to improve in social adaptive functioning might improve after EEG-biofeedback. The hypothesis that EEG-biofeedback results in better cognitive flexibility in daily life situations, however, could not be confirmed by the results of the present study and requires further investigation in future studies.

In contrast to the large effect of EEG-biofeedback on cognitive flexibility, the present study revealed no specific effects on the clinical symptoms of ASD. This finding contradicts previous studies that did find reductions in sociability problems and communication deficits after EEG-biofeedback (Coben and Padolsky 2007; Jarusiewicz 2002; Kouijzer et al. 2010). The absence of reductions in ASD symptoms in the present study provides some support for the suggestion that previous findings of parental report may have been confounded by nonspecific effects of EEG-biofeedback. At the same time, however, we need to be careful in drawing firm conclusions considering that the EEG- and SC-biofeedback groups showed no improvement over the waiting list group. That is, if it would really be the case that nonspecific effects of EEG-biofeedback would be responsible for reductions in ASD symptoms, one would have expected a significant difference between non regulators of the treatment groups and the waiting list group.

There may be alternative reasons for the difference in outcomes in symptoms of ASD between the present and previous studies, such as differences in sample characteristics. That is, the samples of participants with ASD of present and previous studies were different with respect to age and co morbidity. Participants of the present study were 12–18 years old, whereas most other studies included younger participants. Older children and adolescents may be less sensitive to changes in behavior, or alternatively, parents of older children and adolescents may have less notion of their children's behavior throughout the day. Furthermore, the participants in the present study had no co

morbid ADHD diagnoses, whereas previous studies might have included participants with ASD and ADHD. Perhaps neurofeedback acts more on the ADHD characteristics of participants than on their ASD characteristics and thus the effects are more obvious in participants with both ASD and ADHD. This idea is supported by recent studies indicating substantial improvements in impulsive and hyperactive behavior in ADHD following EEG-biofeedback (Arns et al. 2009). Future studies investigating the behavioral effects of EEG-biofeedback in individuals with ASD with or without ADHD may further explicate this possibility.

In the 19-channel EEG data, excessive slow wave activity over frontal and central sites was found in participants with ASD. This finding is in accordance with previous research on EEG and ASD, where excessive delta and theta power were observed as well (Chan et al. 2007; Kouijzer et al. 2010; Murias et al. 2007; Pop-Jordanova et al. 2010). Fifty-four percent of the participants in this study successfully reduced delta and/or theta power during EEG-biofeedback sessions. Unexpectedly, this reduction was not seen in the post-treatment 19-channel EEGs of these participants. Perhaps participants in this study learned to regulate their EEG in the context of EEG-biofeedback sessions, but did not master the task of changing their brainwave patterns to the degree that it generalized beyond the training sessions. This lack of generalization might explain why the decreases in ASD symptoms after EEG-biofeedback were not significantly greater than those seen after SC-biofeedback. That is, previous EEG-biofeedback studies that did reported changes in 19-channel EEG, did also report reductions in symptoms of ASD (Coben and Padolsky 2007; Kouijzer et al. 2010; Sichel et al. 1995). Surprisingly, there were no power differences in EEG resting and task conditions between any of the frequency bands or scalp locations. At least a difference in alpha activity between the conditions eyes open and eyes closed was expected (Barry et al. 2007). The small sample size of the present study relative to the large number of variables in the analyses of the EEG data is likely to be responsible. Even an additional analysis in which the data of all seven tasks was pooled and limited to two frequencies (delta and theta) and three scalp sites (Fz, Cz, and Pz) yielded no significant effects.

Some suggestions for future research can be drawn from the present study. It was demonstrated that EEG-regulators showed long-term improvement in cognitive flexibility after EEG-biofeedback. Future research should investigate the effects of EEG-biofeedback on cognitive flexibility in real life situations. If it could be demonstrated that cognitive flexibility in daily life improves after EEG-biofeedback training, the treatment would be far more interesting for children and adolescents with ASD. It would be interesting to include measures of cognitive flexibility in real life situations in future studies to determine whether EEG-biofeedback results in better coping with situational changes and may improve social adaptive functioning of children and adolescents with ASD. Another issue is that no significant reductions in symptoms of ASD were observed by parents. This result contrasts with findings of previous studies that did report reductions in symptoms of ASD after EEG-biofeedback. Future research should aim for more clearness considering the effects of EEG-biofeedback on clinical symptoms of ASD, for example by using observations at home and in class situations. Such observable reductions in symptoms of ASD might be important criteria for children and adolescents with ASD and their parents in opting for EEG-biofeedback. In addition, future research should disentangle the effects of specific elements of treatment plans that have been used in EEG-biofeedback studies, such as the inhibition of delta and/or theta power, the enhancement of low beta power, and the inhibition of high beta power. In our research line, we decided to focus on the inhibition of delta and theta power, but other treatment plans might result in different clinical and non clinical outcomes. Finally, the sample size in the present study likely was not large enough to differentiate between the effects of EEG-biofeedback in children and adolescents with different diagnostic sub-types within the autistic spectrum. Future studies should include larger sample sizes in order to create more opportunities to investigate the benefits of EEG-biofeedback for children and adolescents with different diagnostic sub-types.

The results of the present study suggest that EEG-biofeedback can be used to regulate EEG activity during EEG-biofeedback sessions in a subsample of children and adolescents with ASD and that these participants improve in cognitive flexibility. However, the results are inconclusive with respect to the clinical application of EEG-biofeedback in children and adolescents with ASD.

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