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Neurofeedback improves executive functioning in children with autism spectrum disorders

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ABSTRACT

Seven autistic children diagnosed with autism spectrum disorders (ASD) received a neurofeedback treatment that aimed to improve their level of executive control. Neurofeedback successfully reduced children's heightened theta/beta ratio by inhibiting theta activation and enhancing beta activation over sessions. Following treatment, children's executive capacities were found to have improved greatly relative to pre-treatment assessment on a range of executive function tasks. Additional improvements were found in children's social, communicative and typical behavior, relative to a waiting list control group. These findings suggest a basic executive function impairment in ASD that can be alleviated through specific neurofeedback treatment. Possible neural mechanisms that may underlie neurofeedback mediated improvement in executive functioning in autistic children are discussed.

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Neurofeedback refers to a form of operant conditioning of electrical brain activity, in which desirable brain activity is rewarded and undesirable brain activity is inhibited. Neurofeedback is believed to elicit growth and changes at cellular levels of the brain, which in turn support brain functioning and behavioral cognitive performance (Demos, 2005). In the domain of intervention, neurofeedback training is useful in treatment of different disorders in adults and children. Positive effects of neurofeedback in adults have been found for Attention Deficit Hyperactivity Disorder

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(ADHD) (Kropotov et al., 2005), traumatic brain injury (Thornton, 2000), epilepsy (Sterman, 2000), depression (Hammond, 2003), migraine (Kropp, Siniatchkin, & Gerber, 2002), addiction (Trudeau, 2005), anxiety disorders (Moore, 2000), and general cognitive performance (Vernon et al., 2003).

Less is known about the effects of neurofeedback in children. In children, research on the effects of neurofeedback is mainly carried out in the area of ADHD (Fuchs et al., 2003; Monastra et al., 2005; Vernon, Frick, & Gruzelier, 2004), but positive effects of neurofeedback have also been found for children with migraine (Kropp et al., 2002) and learning disorders (Fernandez et al., 2003; Thornton & Carmody, 2005). ADHD is typically characterized by a heightened ratio between theta (4–8 Hz) and beta (12–21 Hz) activity in the ongoing EEG during rest. Neurofeedback protocols that have aimed at inhibiting theta activity while rewarding beta activity have led to successful alleviation of symptoms associated with ADHD such as deficits in sustained attention, impulsivity, and control over hyperactive behaviors (reviews in Butnik, 2005; Fox, Tharp, & Fox, 2005).

Several studies suggest that neurofeedback protocols that have been successful for treatment of ADHD may also be efficacious for treating children with autistic related deficits. Sichel, Fehmi, and Goldstein (1995) report on Frankie, a 8.5-year-old boy with a mild form of autism and attention impairments suggesting ADHD. Frankie's 19-channel QEEG demonstrated theta (4–8 Hz) to beta (13–21 Hz) ratios of 3.59 (Cz), 3.40 (C3), 3.03 (C4), 3.98 (Pz), 4.07 (P3), 3.63 (P4), and 3.02 (Fz). After 31 neurofeedback sessions aimed at inhibiting theta (4–8 Hz) and rewarding low beta (12–15 Hz), his mother reported positive changes in all the diagnostic criteria defining autism in DSM-III-R (e.g. attending and reacting to others, imaginative play, seeking comfort, more talking and eye contact). QEEG furthermore revealed that theta/beta power ratios had dropped below 3.0 at C3, C4, Fz, Pz, and P4.

Further support for a relation between theta/beta power and autism was provided by Jarusiewicz (2002) who conducted a group study investigating effects of neurofeedback in 12 autistic children, compared with matched controls. The main protocol aimed at inhibiting theta (2–7 Hz) and increasing sensory motor rhythm (SMR) activity (10–13 Hz) over the right motor area. Results indicated a substantial decline in autistic behavior (26% as compared to 3% for the controls) as reflected by the Autism Treatment Evaluation Checklist (ATEC). Parent reports furthermore indicated considerable improvements on socialization, vocalization, school work, anxiety, tantrums, and sleep, whereas no or minimal changes were found for the control group.

More recently Scolnick (2005) conducted a neurofeedback study with five children diagnosed with Asperger disorder, each with unique behavioral problems, i.e. poor social skills, lack of empathy, and inflexibility, coupled with abnormal high theta/beta ratios varying from 2.19 to 6.89. Each child's protocol was determined on the basis of their individual QEEG and consisted of variations on the theme of rewarding 12–15 Hz in the lower beta range while inhibiting slower 4–10 Hz activity in the theta band. After 24 sessions of neurofeedback, parents and teachers reported improvements in behavior, i.e. less anxiety, more flexibility, higher self-esteem, more empathy, improvement in frustration toleration, increased social interaction, and fewer severe mood changes. Furthermore, in two of the five children, theta/beta ratios changed into a positive direction.

The above studies suggest that neurofeedback protocols that inhibit theta and reward beta or SMR may hold particular value for the treatment of autistic children, similar to the treatment of ADHD. Surprisingly, however, no functional explanations currently exist for these improvements and little is known about the neural mechanisms involved. In light of the increasing popularity and clinical use of neurofeedback, however, fundamental explanations become increasingly more relevant. Vice versa, the efficacy of evolved protocols and practices may help to advance more fundamental insight into impairments underlying neuropsychological deficits such as autism and ADHD. The aim of the current study therefore is twofold. On the one hand we wished to contribute to clinical practice by evaluating the efficacy of neurofeedback for treatment of autism, whereas on the other hand we intended to further our understanding of the possible (neural) mechanisms supporting treatment effects.

In order to optimize the neurofeedback treatment protocol for children with ASD and its rationale, further methodological improvement is necessary in the form of controlled studies, larger sample sizes, a more accurate description of sample characteristics and collection of follow-up data. Another guiding principle should be the assessment of the clients' satisfaction with the treatment and procedure to enhance the social validity of the approach. Social validity refers to the use of evaluative

feedback from clients to guide program planning and evaluation (Schwartz & Baer, 1991). Social validity may be evaluated at three levels of treatment: goals, procedures, and outcomes (Wolf, 1978). In the current study we included the above guidelines and evaluative measures (cf. Heinrich, Gevensleben, & Strehl, 2007) to further validate the use of neurofeedback treatment of ASD.

In addition to the practical evaluation of neurofeedback treatment of ASD, the current study aimed to contribute to our understanding of the cognitive and neural mechanisms that underlie neurofeedback improvements in ASD. We hypothesize that the reason for the efficacy of neurofeedback protocols that reduce theta and reward beta lies primarily in the enhancement of activation in the anterior cingulate cortex (ACC). The ACC is one of the main generators of theta (Meltzer, Negishi, Mayes, & Constable, 2007; Onton, Delorme, & Makeig, 2005; Tsujimoto, Shimazu, & Isomura, 2006), and is well known for its role in regulating cognitive and emotional processes in the brain contributing to cognitive control and executive function (review in Bush, Luu, & Posner, 2000). Neuroimaging studies investigating the neural basis of ADHD and ASD have reported hypo-activation and functional under-connectivity of the ACC (Bush, Valera, & Seidman, 2005; Cherkassky, Kana, Keller, & Just, 2006) which could explain why cognitive deficits associated with ADHD and ASD often seem to fall within the domain of self-regulation and executive function (Barkley, 1997). Furthermore, combined EEG-fMRI studies have indicated a negative relationship between theta power and BOLD signal in the ACC (Meltzer et al., 2007), in line with the hypothesis that theta activation in autistic children is associated with under-activation of the ACC (Murias, Webb, Greenson, & Dawson, 2007).

Following the above reasoning we predict that down-regulation of theta activity should enhance activation of the ACC and executive control mechanisms of the brain, which should lead to more efficient behavior of ASD children on tasks requiring executive function. To investigate the hypothesized relationship between theta and executive function, a group of children from the autism spectrum were selected for neurofeedback training that reduced theta activity while rewarding low beta activity, in accordance with the standard ADHD treatment protocol. A waiting-list control group, also diagnosed with ASD, received neurofeedback training at a later time and served as a baseline to determine treatment effects of neurofeedback on children's executive, social and neurophysiological levels of functioning.

1. Method

1.1. Participants

Fourteen children with ASD (12 males; 2 females) with a mean age of 10.1 years (range 8–12 years) were recruited by an advertisement in a magazine for parents of ASD children. Inclusion criteria were an IQ-score of 70 and above and the presence of ASD as diagnosed by a child psychiatrist or health care psychologist. All participants had the diagnosis pervasive developmental disorder—not otherwise specified (PDD-NOS). Each diagnosis was confirmed by a clinical psychologist and by results on the CCC questionnaire. Excluded were children using medication, children with a history of severe brain injury, and children with co-morbidity such as ADHD and epilepsy. The seven children who applied first, were assigned to the intervention group. The control group included seven children who were recruited out of a larger group of children who applied later and were selected to match children of the intervention group on diagnosis, age, sex, and intelligence scores. Table 1 represents the demographic characteristics of the intervention and the control group. There were no significant differences between both groups with respect to the variables sex, mean age, total IQ, verbal IQ, and performal IQ. Children in the control group were invited for neurofeedback training after finishing the present study.

1.2. Procedure

A non-randomized pre-test–post-test control group design with individual matching was used with follow-up measurements after 3 months. During a baseline period, all participants were pre-tested on QEEG and a range of executive functions tasks, and parents completed a communication checklist. After 40 sessions of neurofeedback, or comparable time interval for the waiting-list control group, QEEG, executive functions skills, and communicative abilities were re-collected. During follow-

Table 1

Demographic characteristics of the intervention group (IG) and the control group (CG)

Variable	Intervention group (n = 7)	Control group (n = 7)	p
Gender (male/female)	6/1	6/1	–
Mean age (years) ^a	9.63 (1.53)	10.64 (1.41)	.220
Mean total IQ ^b	92.50 (16.05)	93.83 (13.67)	.891
Mean verbal IQ ^c	97.80 (18.38)	95.40 (18.15)	.841
Mean performal IQ ^d	99.60 (25.77)	93.40 (9.71)	.628

Note. Standard deviations are in parentheses.

^a Age range IG: 8–12, CG: 9–12.

^b Total IQ range IG: 73–111, CG: 82–199.

^c Verbal IQ range IG: 77–119, CG: 78–125.

^d Performal IQ range IG: 73–134, CG: 81–108.

up, 3 months after ending neurofeedback sessions, again, QEEG, executive functions skills and communicative abilities were measured together with a questionnaire to estimate behavioral improvements in children. For the intervention group, the follow-up measurement included a social validity questionnaire. The research design was authorized by an ethics committee for behavioral sciences.

An interview was conducted with the parents prior to the neurofeedback treatment to survey the anamneses of the child, family history, and current problems of the participant. Procedures and possible side effects were explained to all participants. All participants signed informed consent. Pre- and post-treatment measures took 2 h for each participant to complete. Tasks for executive functioning were given to all participants in a fixed order. Questionnaires were filled out by the parents at home.

1.3. QEEG measurement

Children's QEEG (quantitative electroencephalogram) was recorded and digitized with a TruScan 32 Acquisition EEG System (Deymed Diagnostic, USA). Data were acquired using a stretchable electrode cap embedded with 19 sensors at scalp locations Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2, according to the International 10/20 System (Jasper, 1958). A ground electrode was placed between Fp2 and F8 and two ear clips were used as reference electrodes (A1 and A2). Impedance was kept below 5 k Ω , with a maximum difference of 1 k Ω between electrodes. Data were collected for 3 min in an eyes open and an eyes closed condition.

1.4. Neurofeedback training

A portable NeXus-4 amplifier and recording system (Mindmedia, The Netherlands) was used for neurofeedback training and concurrent data collection. Ag/AgCl disposable snap-on sensors (MedCaT, The Netherlands) were applied to the patients' scalp at locations C3 and C4.

Each participant in the intervention group visited a private practice twice a week until 40 sessions were completed. Training was carried out by a state licensed psychotherapist with extensive training in neurofeedback. During each session a protocol was carried out, which consisted of a baseline of 3 min (i.e. no feedback), followed by seven 3-min intervals of neurofeedback. Neurofeedback intervals were separated by 1-min rest intervals, in which the participant was instructed to sit still and relax, without receiving feedback. Neurofeedback training followed a standard ADHD training protocol (Heinrich et al., 2007 for review) aimed at reducing theta activity (4–7 Hz) while increasing activity in the low beta band (12–15 Hz)¹ at C4 (reference at A1). The signal at location C4 was fed back to the patient in visual form. Theta and beta activity were visualized in separate bar graphs on the computer screen and participants were instructed to "try to move down the theta activity below the criterion line on the computer screen

¹ Sometimes low beta activity is also referred to as SMR (sensorimotor rhythm) to indicate its assumed rolandic origin.

and to move up the beta activity above the criterion line, using the feedback to guide you". During intervals when specified amplitude conditions were met, participants were rewarded by the continuation of a short movie that was selected to fit each child's individual interest and age. All movies were presented with audio. When participants failed to maintain power within the required range, the movie and music would stop playing. Individual criteria were set to allow each participant to reach the reward.

1.5. Executive function tasks

According to Smidts (2003), executive functions are typically divided into four separate subdomains, each including one or more executive function tests.

1.5.1. Attentional control

Attentional control encompasses selective attention, visual as well as auditory, and response inhibition. Visual selective attention was measured by the Continuous Performance Test (CPT), a subtest of the neurocognitive test battery CNS Vital Signs (CNSVS). In the CPT, the participant has to respond to one particular character on the computer screen while ignoring other characters during 5 min. The score for visual selective attention is based on the amount of errors of the CPT (range 0–200). Selective attention for auditory stimuli was measured by the Test of Sustained Selected Attention (TOSSA; Kovács, 2005b). In the TOSSA, participants have to respond to sets of 3 beeps while ignoring sets of 2 or 4 beeps. Beeps are presented during 8 min with variable speed. The test score reflects the percentage of good answers, calculated by dividing the number of hits by the total amount of items, times 100. Response inhibition is divided in a verbal and a motor variant. Verbal response inhibition was assessed by the Stroop test (Stroop, 1935). In this test, participants have to read aloud as soon as possible (A) 100 words (green, red, yellow, and blue), (B) the color of 100 colored rectangles, and (C) the color of the ink of 100 written incongruent color names. The goal in part C is to pronounce the name of the color of the ink, while ignoring reading the word. The score on this test is represented by the interferential time (time C minus time B). Motor response inhibition was assessed with the response inhibition score (RIS; range 0–100) of the TOSSA, based on the number of commission errors.

1.5.2. Cognitive flexibility

Cognitive flexibility covers verbal memory and visual memory, set-shifting, concept generation, and feedback utilization. Verbal memory and visual memory were assessed by the Verbal Memory Test (VBM) and the Visual Memory Test (VIM) of the CNSVS, respectively. In the VBM and the VIM, participants have to memorize words ($n = 15$, VBM) and geometric figures ($n = 15$, VIM) and later recognize them in a series of distracters ($n = 15$ for both tests). The sum of correct responses was calculated to get a final score for verbal memory (maximum = 60) and a final score for visual memory (maximum = 60). Set-shifting was examined by the Trail Making Test (TMT; Reitan, 1956). In the TMT, participants have to switch between the numerical mode and the alphabetic mode by connecting 26 numbers and characters in the 1-A-2-B-3-C order. A score on the TMT is comprised of the total time needed to finish the test, translated into an age related t -score (range 20–75). Concept generation and feedback utilization were examined by the Milwaukee Card Sorting Test (MCST; Kovács, 2005a), a computerized version of the Wisconsin Card Sorting Test. The participant had to generate and apply a non-spoken rule for sorting cards ($n = 60$), based on feedback (e.g. 'good' or 'fault'). These card sorting principles can be either color, shape, or number and change after every 10 correct answers. An indicator for cognitive flexibility is the number of categories (range 0–6) a participant creates with 60 cards.

1.5.3. Goal setting

Goal setting was assessed by the Tower of London (TOL; Kovács, 2005c). Participants had to copy a construction of blocks and bars by moving three prearranged different colored blocks along three bars of different lengths. The score on the TOL, is a percentage calculated by dividing the participants' score by the maximum score, times 100.

1.5.4. Speed and efficiency

Speed and efficiency was measured by the Symbol Digit Coding (SDC) of the CNSVS. Participants had to code as many symbols as possible within 2 min, according to a set of eight symbol–digit pairings that are displayed continuously for reference on screen. A score for speed and efficiency is calculated by the number of correct responses minus the number of errors on the SDC.

1.6. Questionnaires

1.6.1. Children's Communication Checklist (CCC-2-NL)

The CCC-2-NL (Geurts, 2007) was used to assess improvement in participant's language structure, pragmatics, and social interaction. Language structure includes the subscales speech production, syntax, semantics, and coherence. The domain of pragmatics consists of the subscales inappropriate initiation, stereotyped conversation, use of context, and non-verbal communication. The domain of social interaction includes the subscales social relations and interests. An age-related standard score was calculated for each subscale and for the composed scales general communication (language structure and social interaction) and pragmatics.

1.6.2. AUTI-R

An adapted version of the AUTI-R (Berckelaer-Onnes & Hoekman, 1991) was used to study improvement of children in the intervention group on social interaction, communication, and restricted, repetitive, and stereotyped patterns of behavior, interests and activities. Eleven items of the AUTI-R that were considered not relevant for the present study and five items that did not fall into the categories social interaction, communication or restricted, repetitive, and stereotyped patterns of behavior, interests and activities, were excluded from the list. The adapted questionnaire contained 33 items, subdivided into the scales Social interaction ($n = 10$), Communication ($n = 8$), and Behavior ($n = 15$). Items on the questionnaire were rated on a 5-point scale, with 1 point indicating low progression and 5 points indicating strong progression. Mean scores were calculated for the subscales Social interaction, Communication, and Behavior, and for the complete questionnaire.

1.6.3. Social validity

Social validity was assessed by a self-constructed anonymous 5-point scale questionnaire with 15 items about the Goals of treatment ($n = 4$), Treatment procedures ($n = 8$), and Outcomes ($n = 3$) (Wolf, 1978). All items were scored, with 1 point indicating low satisfaction and 5 points indicating high satisfaction. Sum scores were calculated for each subscale to evaluate the acceptability of the neurofeedback treatment. Three open response questions were added to assess whether parents had any remarks, whether they had suggestions to improve neurofeedback treatment, and whether they would recommend neurofeedback treatment to others.

1.7. Data analysis

1.7.1. QEEG

Eye blinks and other artifacts were manually removed from the raw EEG data by an independent EEG specialist and statistician, who was blind to the subject's classification (i.e. intervention group vs. control group) and the type of EEG (i.e. pre- vs. post-training). The raw data were processed with fast Fourier transformation to determine the magnitude of each frequency band in microvolt. Separate power measures were calculated for delta (1–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), low beta (12–15 Hz), beta 2 (15–18 Hz), beta 3 (18–25 Hz), and high beta (25–30 Hz). EEG data of all individuals were compared with the Neuroguide (Thatcher et al., 2003) database, which provides reliable descriptors of normative brain electrical activity (John et al., 1988). Linked ears montages were used. Data from all 19 electrode sites were used for analysis. The split-half reliability and test–retest reliability of the artifact free data of all subjects were above .95 ($p < .05$). Absolute power (the amount of energy in μV^2), relative power (the percentage of power in a frequency band relative to the total power contained by all other frequency bands), and coherence were calculated for each participant, frequency band, and individual electrode lead. All power and coherence values were subsequently

transformed to Z-scores, reflecting deviancy from the normative database (Hughes & John, 1999). A 2 (Time: time1 vs. time2) \times 2 (Group: intervention vs. control) mixed MANOVA was performed to look for treatment effects in the intervention group relative to controls.

1.7.2. Session data

Eye blinks and other artifacts were manually removed from the raw EEG data of 40 sessions, collected at C3 and C4 during training intervals. The raw data were Fast Fourier Transformed (FFT) to determine the power of each frequency. Separate power measures were calculated for delta (1.5–3.5 Hz), theta (4–8 Hz), alpha (8–12 Hz), low beta (12–15 Hz), beta 2 (13–21 Hz), and high beta (22–30 Hz). Power values of each frequency band were log-transformed. A 2 (Time: first sessions vs. last sessions) \times 2 (Location: C3 vs. C4) mixed MANOVA was conducted to compare power during the first 20 sessions with the final 20 sessions. Furthermore, the efficacy of neurofeedback over sessions per frequency band was estimated for each individual subject by calculating a linear regression line and Spearman regression coefficient fitting the progression of power values over sessions.

1.7.3. Executive function tasks

Results of a one-sample Kolmogorov–Smirnov test showed that data on each variable did not deviate significantly from normality. A MANOVA was conducted to test differences in executive functions for the intervention group and the control group at time1. Neurofeedback related changes in executive functions were verified by performing a 2 (Time: time1 vs. time2) \times 2 (Group: intervention vs. control) mixed MANOVA.

1.7.4. Questionnaires

Results of a one-sample Kolmogorov–Smirnov test showed that data on each variable of the CCC-2 did not deviate significantly from normality. MANOVA was conducted to test for differences on the CCC-2 between the intervention group and controls at time1. Neurofeedback related changes on the CCC-2 were verified by performing a 2 (Time: time1 vs. time2) \times 2 (Group: intervention vs. control) mixed MANOVA.

In order to assess whether the intervention group decreased in ASD-symptoms more than the control group, a comparison between scores on the adapted AUTI-R of the intervention group and controls was made using a MANOVA with between-subjects factor Group.

The social validity of the neurofeedback treatment was evaluated via the sum scores of the subscales Goals, Procedures, Outcomes, and via open response questions.

2. Results

2.1. Session data

At the individual level, Spearman's correlation coefficients showed a significant reduction of theta power (4–7 Hz) over 40 sessions of neurofeedback in five participants at C4 ($ps < .05$, $r = -.596$ to $-.718$) and in the same five participants at C3 ($ps < .05$, $r = -.496$ to $-.771$). Two participants did not show significant reduction of theta power at C4 ($p = .411$, $r = .035$; $p = .359$, $r = .056$) and C3 ($p = .018$, $r = .453$; $p = .170$, $r = .135$). Results of theta reduction at C3 and C4 for all participants can be found in Fig. 1.

Low beta power (12–15 Hz) increased significantly over time for five participants at C4 ($ps < .05$, $r = .218$ – $.410$) and for six participants at C3 ($ps < .05$, $r = .253$ – $.529$). Two participants did not show significant increase of low beta power at C4 ($p = .311$, $r = .079$; $p = .173$, $r = -.145$) and one participant did not show significant increase at C3 ($p = .372$, $r = .051$) (see Fig. 2).

Besides changes in theta and low beta power, changes in delta power (1.5–3.5 Hz) were found as well. Delta power decreased significantly in five participants at C4 ($ps < .05$, $r = -.449$ to $-.555$) and in five participants at C3 ($p < .05$, $r = -.291$ to $-.562$). No increase in delta power was found in two participants at C4 ($p = .125$, $r = .177$; $p = .356$, $r = .125$) and at C3 ($p = .263$, $r = .098$; $p = .054$, $r = -.243$). Results can be found in Fig. 3. In alpha power (8–12 Hz), beta 2 power (13–21 Hz), and high beta power (22–30 Hz), no unanimous patterns of change were found.

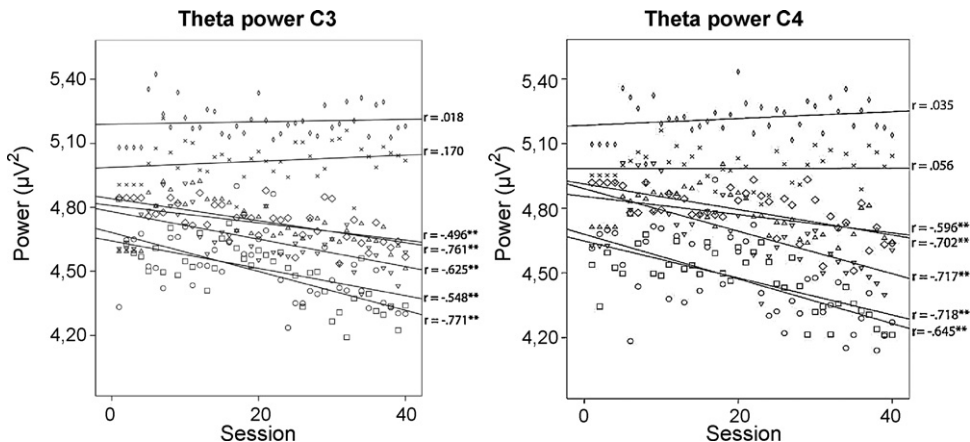


Fig. 1. Average theta (4–7 Hz) power during neurofeedback sessions recorded over C3 (left graph) and C4 (right graph) indicating the reduction in theta power over consecutive sessions. Regression lines reflect the slope of theta reduction over time for each individual patient, with * $p < .05$ and ** $p < .01$.

Analysis at group level further supported our results. A 2 (Time: first sessions vs. last sessions) \times 2 (Location: C3 vs. C4) mixed MANOVA showed significant reduction of theta power (4–7 Hz) ($F(1,6) = 11.419, p < .05, \eta = .656$) and significant increase of low beta (12–15 Hz) ($F(1,6) = 21.922, p < .01, \eta = .785$) at C3 and C4 over 40 sessions of neurofeedback. Besides power changes in theta and low beta, a significant decrease of delta power (1.5–3.5 Hz) over time was found as well ($F(1,6) = 6.982, p < .05, \eta = .538$). For alpha power (8–12 Hz), beta2 power (13–21 Hz), and high beta power (22–30 Hz), no significant effects of time were found.

Decrease of delta power was significantly correlated with decrease in theta power ($r = .667, p < .01$) and with increase in low beta power ($r = -.695, p < .01$). The correlation between decrease in theta and increase in low beta power was highly significant ($r = -.811, p < .001$).

2.2. QEEG

The absolute and relative power of each frequency band for all 19 channels for the intervention group and the controls were compared using MANOVA. In order to claim a treatment effect, we need

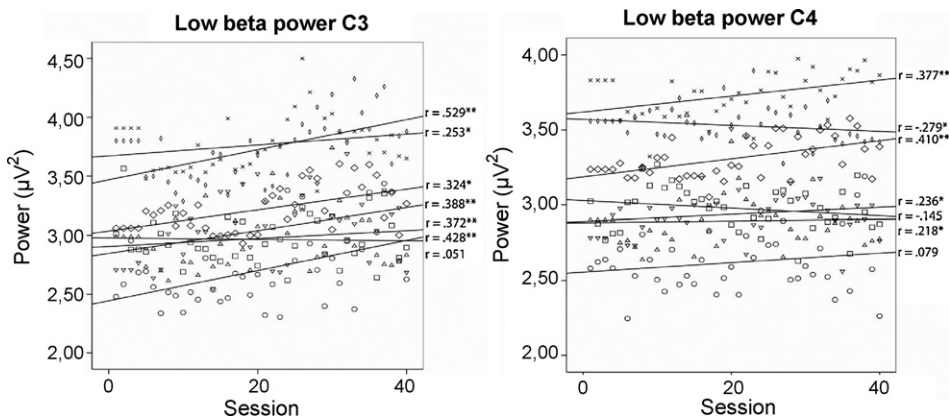


Fig. 2. Average low beta (12–15 Hz) power during neurofeedback sessions recorded over C3 (left graph) and C4 (right graph) indicating the increase in low beta power over consecutive sessions. Regression lines reflect the slope of beta enhancement over time for each individual patient, with * $p < .05$ and ** $p < .01$.

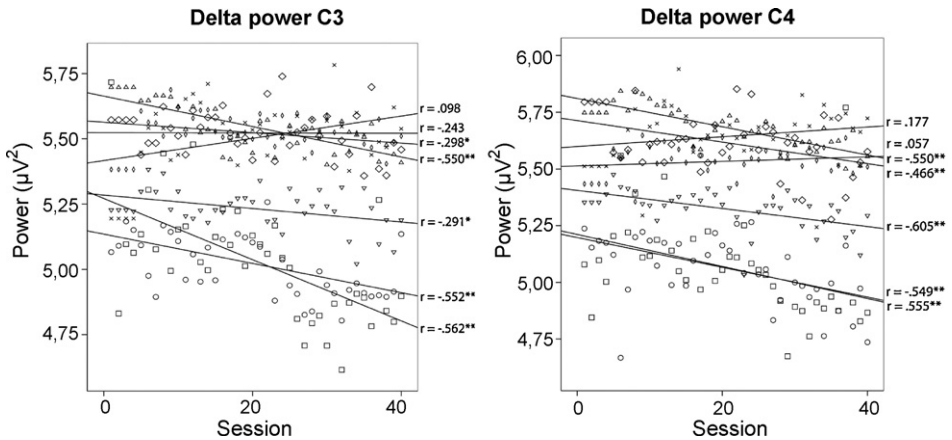


Fig. 3. Average delta (1.3–3.5 Hz) power during neurofeedback sessions recorded over C3 (left graph) and C4 (right graph) indicating the reduction in delta power over consecutive sessions. Regression lines reflect the slope of delta reduction over time for each individual patient, with * $p < .05$ and ** $p < .01$.

the interaction between Time (time1 vs. time2) and Group (intervention vs. control) to be significant. The mixed MANOVA suggested no significant multivariate interaction between Time and Group in the target frequency bands, i.e. absolute ($F(1,12) = 2.382, p = .149, \eta = .166$) or relative theta power ($F(1,12) = .986, p = .340, \eta = .076$) and absolute ($F(1,12) = .018, p = .897, \eta = .001$) or relative low beta power ($F(1,12) = .614, p = .449, \eta = .049$). Univariate results of absolute and relative theta and low beta power in 19 separate electrodes revealed no significant interaction effects either (range of F -values = .000–3.977, $ps > .05$). A similar 2 (Time: time1 vs. time2) \times 2 (Group: intervention vs. control) MANOVA for the other frequency bands, i.e. delta, alpha, beta2, beta3 and high beta revealed no significant multivariate effects, neither for absolute nor relative power (range of F -values = .000–1.820, $ps > .05$).

For the analysis of coherence, a 2 (Time: time1 vs. time2) \times 2 (Group: intervention vs. control) mixed MANOVA was performed. Univariate results revealed a significant reduction of hypo connectivity in theta power at time2 (F -values up to 17.572, $ps < .05$), especially between frontal and central/temporal electrodes. However, since this reduction was found in both the intervention and the control group, no significant interaction effects were found (range of F -values = .000–2.914, $ps > .05$).

2.3. Executive function tasks

A MANOVA was conducted to test the hypothesis that participants in the intervention group would display the same scores as participants in the control group at time1. No statistical significant differences between intervention and control group were found on tests for executive functioning at time1 ($F(1,12) = 1.066, p = .577, \eta = .842$).

To analyze whether children in the intervention group scored significantly higher on tests for executive functioning at time2 compared to the matched control group, a 2 (Time: time1 vs. time2) \times 2 (Group: intervention vs. control) mixed MANOVA was performed. In order to claim a treatment effect and to control for practice effects, we need the interaction to be significant.

2.3.1. Attentional control

Participant's capacity for attentional control was tested using separate measures targeting children's attentional capacity in the visual and auditory domains and their ability to inhibit verbal and manual response tendencies. Table 2 reports the behavioral results of all executive function tests gathered for both groups at time1 and time2. No significant interaction between Time and Group was found for measures of visual selective attention ($F(1,11) = .047, p = .832, \eta = .004$). Both groups made very little errors in detecting a target letter in a continuous stream of distractors, leaving little or no

Table 2

Means and standard deviations of the intervention group (IG) and the control group (CG) at time 1 and time 2 on tests for executive functions

	Time1		Time2	
	IG, M (S.D.)	CG, M (S.D.)	IG, M (S.D.)	CG, M (S.D.)
Attentional control				
- Visual selective attention	4.33 (2.81)	9.14 (14.44)	4.17 (4.26)	7.29 (8.90)
- Auditory selective attention	47.87 (14.21)	67.79 (25.61)	62.40 (14.18)	68.90 (27.30)
- Inhibition of verbal responses	68.17 (18.87)	65.71 (31.53)	30.00 (12.12)	50.14 (26.59)
- Inhibition of motor responses	78.50 (13.16)	89.84 (11.02)	89.93 (9.20)	91.47 (9.66)
Cognitive flexibility				
- Verbal memory	53.33 (3.62)	51.29 (2.63)	52.17 (4.07)	50.57 (6.604)
- Visual memory	46.00 (3.74)	41.00 (5.57)	45.00 (4.34)	40.29 (8.321)
- Shifting	30.00 (15.68)	29.71 (10.50)	47.00 (13.27)	34.00 (13.29)
- Concept generation	2.55 (1.48)	3.50 (1.70)	4.96 (.45)	3.83 (1.42)
Goal setting	55.45 (9.07)	55.84 (18.17)	75.85 (9.17)	57.03 (11.89)
Speed and efficiency	34.33 (7.06)	41.00 (15.52)	41.33 (5.13)	43.86 (10.96)

Note. M: mean, S.D.: standard deviation.

room for improvement at time2 (values for visual selective attention in Table 2 represent the amount of errors found in 200 items). However, a significant Time \times Group interaction effect was found for measures of auditory selective attention ($F(1,11) = 8.437, p = .014, \eta = .434$). Children in the intervention group showed a considerable improvement in their ability to correctly detect auditory targets in the TOSSA, from 48% to 62% correct responses after neurofeedback training, as compared to the control group who showed minimal improvement from 68% to 69% correctly detected targets. In addition, a significant interaction between Time and Group was found for children's capacity to inhibit verbal responses ($F(1,11) = 4.890, p = .049, \eta = .308$). Interference effects of written names were strongly reduced from 68 s before to 30 s after neurofeedback training for the intervention group. The control group also showed a difference between interference effects at time1 and time2 (66 and 50 s respectively) but this reduction was about half the size of the effect found for the intervention group. Consistent with the increased ability to inhibit verbal responses, children of the intervention group were also better able to inhibit impulsive tendencies in responding on the TOSSA, suggesting improved inhibition capacity after neurofeedback training (78% correctly inhibited before training vs. 90% after neurofeedback training). Only minimal improvements in impulse control were found for the controls (89% correct inhibitions at time1 followed by 91% correct inhibitions at time2), resulting in a significant Time \times Group interaction, $F(1,11) = 5.064, p = .046, \eta = .315$.

2.3.2. Cognitive flexibility

Children's cognitive flexibility was investigated using measures of visual and verbal memory, set-shifting and concept generation. Neurofeedback training did not influence children's capacity to memorize and recognize words ($F(1,11) = .021, p = .889, \eta = .002$) and geometric shapes ($F(1,11) = .004, p = .952, \eta = .000$). Both groups showed a minimal non-significant reduction of performance from time1 to time2 (see Table 2), on verbal memory, $F(1,11) = .355, p = .563, \eta = .031$, and visual memory, $F(1,11) = .138, p = .717, \eta = .012$. However, children's set-shifting ability as indexed by the TMT did show a significant Time \times Group interaction ($F(1,11) = 5.602, p = .037, \eta = .337$), reflecting improved cognitive flexibility and sequencing after neurofeedback treatment. For the intervention group t -scores improved from 30 (time1) to 47 (time2), whereas only a small improvement was found for the control group with t -scores improving from 30 (time1) to 34 (time2). Also concept generation and use of feedback, as measured by the MCST, were found to improve significantly for the intervention group as compared to the control group, $F(1,11) = 5.081, p = .046, \eta = .316$. After neurofeedback, ASD children discovered an average of 5 (out of 6) card sorting rules, whereas before training they only reached an average of 2.5. In contrast, the performance of the control group was comparable at time1 (3.5 rules) and time2 (3.8 rules).

2.3.3. Goal setting

Analysis of children's goal setting capacity as assessed by the TOL showed a significant interaction between Time and Group, $F(1,11) = 7.198$, $p = .021$, $\eta = .396$, reflecting a clear improvement in complex sequential problems after neurofeedback training, as compared to the control children. At time1 children from both groups reached an average performance of 55 (range 0–138). However, whereas children of the control group showed little improvement (57 at time2), children of the intervention group drastically improved their capacity score to 76 at time2.

2.3.4. Speed and efficiency

Children's combined score for speed and efficiency on the SDC indicated a stronger improvement for the intervention group than for the control group (see Table 2), but the required interaction between Group and Time was not found significant ($F(1,11) = .397$, $p = .542$, $\eta = .035$).

A 2 (Time: time2 vs. follow-up) \times 2 (Group: intervention vs. control) mixed MANOVA indicated no significant differences between post-treatment and 3-month follow-up measurements of children's executive functioning at follow-up, $F(1,11) = .987$, $p = .602$, $\eta = .832$.

2.4. Questionnaires

2.4.1. CCC-2

The CCC-2 measured parents' appreciation of their children's communication skills for different aspects (subscales) of communication. A MANOVA was conducted in order to test the hypothesis that participants in the intervention group would display the same scores on the CCC-2 questionnaire as participants in the control group at time1. No statistically significant differences between intervention and control group were found on the CCC-2 questionnaire collected at time1, $F(1,12) = 54.149$, $p = .106$, $\eta = .998$.

To analyze whether children in the intervention group scored significantly higher on the CCC-2 at time2 compared to the matched control group, a 2 (Time: time1 vs. time2) \times 2 (Group: intervention vs. control) mixed MANOVA was performed. Separate analysis of the communication subscales of the CCC-2 showed a significant Time \times Group interaction effect for non-verbal communication, $F(1,12) = 5.505$, $p = .037$, $\eta = .314$, reflecting an improvement in non-verbal communication for the intervention group, relative to the control group. For none of the other subscales the interaction between Time and Group was found significant, all $ps > .05$. In Table 3 the average ratings of children's communication skills are reported for sub- and compound-scales of the CCC-2 for the control group and the intervention group at time1 and time2. Lower values in Table 3 reflect better communication skills. Analysis of the two compound scales, general communication and pragmatics, revealed a significant interaction effect between Time and Group for general communication, $F(1,12) = 5.379$,

Table 3

Test results of the CCC-2 for the intervention group (IG) and the control group (CG) at time1 and time2

	Time1		Time2	
	IG, M (S.D.)	CG, M (S.D.)	IG, M (S.D.)	CG, M (S.D.)
General communication	115.14 (10.45)	115.86 (9.42)	101.29 (12.09)	114.29 (16.45)
Pragmatics	60.57 (7.00)	60.71 (7.25)	54.14 (5.579)	65.86 (20.84)
- Speech production	12.86 (2.54)	12.14 (3.63)	10.86 (2.96)	11.43 (4.08)
- Syntax	12.71 (1.89)	14.43 (1.40)	11.29 (2.69)	14.71 (1.89)
- Semantics	12.29 (2.29)	13.14 (1.57)	12.00 (2.08)	13.43 (1.40)
- Coherence	15.43 (1.81)	15.43 (1.51)	14.28 (1.50)	14.14 (3.58)
- Inappropriate initialization	14.29 (1.89)	13.57 (2.76)	13.86 (1.57)	14.57 (2.57)
- Stereotyped conversation	15.14 (2.27)	15.57 (1.40)	13.57 (1.81)	14.43 (3.64)
- Context use	15.14 (1.77)	16.71 (1.89)	13.71 (1.80)	16.14 (2.54)
- Non-verbal communication	15.86 (2.34)	14.86 (2.85)	13.71 (2.50)	15.57 (2.76)
- Social relations	15.57 (1.90)	14.42 (2.63)	14.57 (2.07)	14.57 (2.44)
- Interests	13.57 (1.90)	14.00 (2.16)	12.14 (3.67)	14.14 (2.04)

Note. M: mean, S.D.: standard deviation.

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Table 4

Means and standard deviations of the subscales of the adapted AUTI-R for the intervention group (IG) and the control group (CG)

	IG, M (S.D.)	CG, M (S.D.)
Social interaction	36.50 (3.51)	30.71 (.92)
Communication	29.00 (1.79)	24.14 (.64)
Typical behavior	48.33 (3.44)	44.14 (1.06)
Total	113.83 (7.17)	99.00 (1.95)

Note. M: mean, S.D.: standard deviation.

$p = .039$, $\eta = .310$, but not for pragmatics, $F(1,12) = .036$, $p = .852$, $\eta = .003$. Parents of children in the intervention regarded their children's communication skills as more advanced after neurofeedback training than before, whereas no such difference was found for the control group.

A 2 (Time: time2 vs. follow-up) \times 2 (Group: intervention vs. control) mixed MANOVA indicated no significant changes in scores on the CCC-2 3 months after neurofeedback training was ended ($F(1,12) = .253$, $p = .930$, $\eta = .752$).

2.4.2. AUTI-R

The AUTI-R measured parents' evaluation of children's improvements on social interaction, communication, and typical behavior. Table 4 shows the average improvement for the intervention and control group for each subscale of the AUTI-R. Following treatment, parents' ratings suggested improvements for children in the intervention group on social interaction, communication, and typical behavior as compared to children in the control group. A MANOVA with between subjects factor Group was used to analyze the results of the three subscales of the adapted AUTI-R. A significant increase in desired behavior after neurofeedback training was found for the intervention group in comparison with the control group. Children's social interaction ability was valued to be improved following treatment, as compared to the control group, $F(1,12) = 17.775$, $p = .001$, $\eta = .618$. Children's communication ability was assessed to be enhanced in comparison to the assessment of children in the control group, $F(1,12) = 29.054$, $p = .000$, $\eta = .725$. Furthermore, typical autistic behavior was found to be attenuated as compared to the assessment of children in the control group, $F(1,12) = 7.782$, $p = .018$, $\eta = .414$.

2.4.3. Social validity

Social validity of the intervention was assessed using 5-point rating scales (5 = high satisfaction, 1 = low satisfaction). Neurofeedback treatment was a socially acceptable treatment method with respect to its goals, procedures, and outcomes. Parents of children in the intervention group indicated that they were well informed about the goals of treatment before intervention started ($M = 4.67$). Parents rated the treatment as being neither aggravating for their child ($M = 4.47$), nor for themselves ($M = 3.34$). Viewing video's during training was rated not to be aggravating at all for the children ($M = 5$), as was placement of electrodes on the scalp ($M = 4.83$). The requirement of visiting the private practice twice a week for training ($M = 3.17$) and for pre- and post-assessment ($M = 3.17$) was considered the most aggravating part of the procedure for the parents, although the mean scores on these items are still relatively positive, i.e. in the direction of 'not aggravating'. Parents indicated to be satisfied with the outcomes of the treatment with respect to children's social behavior ($M = 3.83$), communication skills ($M = 3.83$), and typical behavior ($M = 3.83$). All parents would recommend neurofeedback treatment to other parents of children with ASD. Only two parents had suggestions for improvement of neurofeedback treatment, which were a real life experience of neurofeedback treatment for the parents themselves and more time for evaluation during treatment. No parent had any other further remarks in addition to the personal explanation of their answers on the 5-point scales.

3. Discussion

The present study evaluated the effects of a specific ADHD neurofeedback training protocol for treatment of autistic children. Reduction of theta power was hypothesized to improve children's

executive capacities by enhancing activation of the ACC, which is one of the main generators of theta activation over central areas. Consistent with our prediction, children of the intervention group made large improvements in performance on a range of executive function tasks after neurofeedback training, whereas no such effects were found for a matched control group. These findings provide further support for the impairment of executive functions in autism, and reinforce existing neurobiological views on autism that suggested abnormal functioning of the ACC. Furthermore, our findings provide further evidence in support of the view that neurofeedback may hold particular value for treatment of children with ASD which might be comparable with the effects found with ADHD.

At a neurophysiological level, neurofeedback training successfully reduced theta power (4–7 Hz) and significantly increased low beta power (12–15 Hz) in all but two of seven participants in the intervention group. Interestingly, and consistent with our hypothesis that neurofeedback protocols that target children's theta/beta ratio mainly work because they reduce theta power, attenuation of theta power was found more reliable than enhancement of beta power over sessions. Children's individual Spearman correlation coefficients reflected significant reductions of theta in five participants showing consistent effects over both hemispheres at C4 (average $r = .68$) and C3 (average $r = .64$), and enhancement of beta in five participants at C4 (average $r = .30$) and C3 (average $r = .38$). Furthermore, consistent decreases in delta power (1.5–3.5 Hz) were found for five participants at C4 (average $r = .55$) and at C3 (average $r = .45$). The gradual reduction in delta power probably co-occurred in conjunction with the reduction in theta power, which is further supported by the strong correlation between power reductions of both frequencies over time ($r = .67$).

Considering the consistent suppression of theta and delta frequencies and enhancement of low beta activation over time across sessions, one could imagine structural changes in QEEG to develop between pre- and post-test recordings. However, no significant changes were found in the QEEG of the intervention group as compared with QEEG data of the control group. Our findings are in line with results of Kropotov et al. (2007) who found no notable changes in QEEG power spectra of children with ADHD after neurofeedback training, although neurofeedback was found to affect the amplitude of event-related potential (ERP) components.

Coben and Padolsky (2007) found changes in children's QEEG coherence after neurofeedback training reflecting a decrease in cerebral hyper-connectivity in 76% of all children of the intervention group. QEEG coherence values were only available for the intervention group, not for the control group. In the present study, changes in connectivity were found for both the intervention and the control group. These findings suggest a test–retest effect between pre- and post-test EEG assessment which could, e.g. reflect differences in vigilance or arousal between the two assessments. That is, young children may be more alert and attentive during their first EEG assessment as compared to the second time. This different mental state may be responsible for the observed differences in QEEG coherence between the pre- and post-test in both groups. Another explanation for the absence of differences in QEEG coherence is the small sample size that was used.

At a cognitive level, neurofeedback training was hypothesized to improve the executive functions of children with ASD, comparable with the success of the protocol in the treatment of ADHD (Butnik, 2005). Significant improvement in attentional control, cognitive flexibility and goal setting were noted for children in the intervention group when compared to children in the control group. These results are important because they reflect a serious cognitive improvement in the intervention group that cannot be reduced to differences in perceived well-being, e.g. by parents. Instead, these findings indicate that neurofeedback training was associated with a clear improvement in cognitive functioning on tasks requiring executive control. Improvements were found for the majority of tasks taxing executive control, with strong improvements on sustained auditory selective attention (30% more correct responses), inhibition of verbal responses (55% reduction in response interference time), inhibition of motor responses (15% reduction of commission errors), set shifting (57% reduction of time needed to switch between the numerical and alphabetical mode), concept generation (50% increase in the number of card sorting categories created), and planning ability (37% increase in performance on the Tower of London task). Symbol digit coding was found improved (20% more accurate) for the treatment group, but the difference with the improvement of the control group (7%) was not significant. No noteworthy improvements were found on tasks taxing verbal and visual memory, and sustained visual attention. Most children showed to be already highly efficient on these

tasks before the start of the neurofeedback treatment at time1, leaving little room for further improvement. Coben and Padolsky (2007) evaluated executive functioning of children with ASD after neurofeedback training using a questionnaire completed by parents and teachers. In agreement with the present results a significant improvement on measures of executive functions was reported. The present experimental findings further extend these previous results by showing enhanced performance on a range of cognitive tasks requiring executive control. Whereas the appraisal of a child's level of executive functioning might be influenced by wishful thinking or social expectation, such factors cannot explain a 40% average increase in cognitive performance. The fact that similar improvements were found over a range of different executive tasks further strengthens the conclusion that neurofeedback substantially enhanced the executive capacity of children with ASD. These results are furthermore in line with recent models that suggest a single genetic factor to underlie most executive functions (Friedman et al., 2008).

We hypothesized that the elevated theta power that characterizes autistic children is functionally related to their executive impairment. Electroencephalographic and magnetoencephalographic studies have localized frontal theta activation to the rostral ACC (Gevins, Smith, & McEvoy, 1997; Ishii et al., 1999) and studies combining EEG and fMRI have consistently found correlations between theta power and BOLD signal in rostral ACC (Meltzer et al., 2007; Pizzagalli, Oakes, & Davidson, 2003; Sammer et al., 2007). Interestingly, ACC activation and theta power appear to be inversely related. High-functioning autistic individuals show hypoactivation and reduced connectivity of the ACC (Cherkassky et al., 2006; Kana, Keller, Minshew, & Just, 2007) whereas EEG measures consistently indicate elevated levels of theta power over medial frontal areas in ASD (e.g. Murias et al., 2007). Meltzer et al. (2007) found increasing working memory load to be associated with enhancements of EEG theta power which correlated negatively with BOLD signal in a network of areas including the rostral ACC (Meltzer et al., 2007). Similar findings were reported by Sammer et al. (2007) using mental arithmetic-induced workload and Kana et al. (2007) using a response inhibition paradigm. Interestingly, deactivation of the ACC during cognitive demanding tasks is often found in association with deactivations of other (medial) areas, such as the precuneus, which together have been labeled the default mode network (DMN) reflecting its high default metabolism during rest (Gusnard, Raichle, & Raichle, 2001). Much interest has developed in understanding the function of the DMN and several interesting views have been formulated which appear to converge on the idea that the DMN is involved in self-referential processing (Northoff et al., 2006) and understanding others' intentions through mental simulation (Uddin et al., 2007). These findings may have implications for understanding social impairments in ASD. However, for the present discussion it is first important to note that the rostral ACC is not directly involved in executing cognitive control (Rushworth et al., 2004), but that its activation is inversely related to other areas that are activated during cognitive tasks, such as the lateral prefrontal cortex (Greicius, Krasnow, Reiss, & Menon, 2003). Following this suggestion, Fox, Snyder, et al. (2005) discovered strong spontaneous anticorrelations between a "task-negative" DMN and an opposing "task-positive" attentional network, in a resting state. Kelly et al. (2008) furthermore found differences in individual attentional capacity to depend on the strength of the negative correlation between the two opposing networks, with a reduced antiphase relation resulting in more variable behavioral performance. In addition, a recent fMRI study by Weissman et al. (2006) indicated that a failure to suppress the DMN may result in lapses of attention. Uddin et al. (in press) yield further support for this view by indicating that the balance between the two networks is primarily controlled by the DMN.

Importantly, these findings provide a possible mechanism through which we can understand the relation between theta power, ACC activation, and executive function. The enhancement of theta that is consistently found during cognitive effortful tasks, such as use of working memory (Jensen & Tesche, 2002), mental arithmetic (Mizuhara, Wang, Kobayashi, & Yamaguchi, 2004), error monitoring (Luu, Tucker, & Makeig, 2004), and sentence comprehension (Bastiaansen, van Berkum, & Hagoort, 2002), probably reflects deactivation of the rostral ACC/DMN, to allow activation in (task-positive) areas supporting the processing of external goals (cf. Fransson, 2005). Consistent with the hypothesis that the executive problems of autistic individuals may originate from a defective DMN, Kennedy et al. (2006) recently found that autistic subjects, as compared with controls, did not deactivate their DMN during a range of cognitive and emotional Stroop tasks. Inability to deactivate or modulate activation of the DMN might thus impair the engagement of task-positive areas exerting cognitive control.

So far we have mainly focused on theta and its possible contribution to improvements in executive control. However, in addition to theta reduction the neurofeedback protocol also operated to enhance beta activation, which might also have contributed to the success of the treatment. Interestingly, whereas theta activation is negatively related to activation in medial frontal areas, beta power appears to be positively related to activation in those same areas, as is indicated by recent EEG-fMRI studies (Laufs et al., 2003; Mantini, Perrucci, Del Gratta, Romani, & Corbetta, 2007) and intracerebral recordings studying the neural origins of the beta rhythm (Bočková, Chládek, Jurák, Haláček, & Rektor, 2007). That is, comparable with the effect of theta, enhancing beta should also increase activation in the DMN. In other words, the effects of reducing theta and at the same time enhancing beta power may actually work together in parallel to increase activation of hypoactive areas of the DMN in ASD patients.²

Interestingly, the hypothesis that ASD is primarily characterized by underactivation of the DMN may explain both executive dysfunctioning and social deficits that are typical of ASD. As was indicated earlier, parts of the DMN are known to be involved in self-referential processes and internal models of the self (reviews in Northoff & Bermpohl, 2004; Northoff et al., 2006). Importantly, the capacity to mentalize about others' intentions and their internal states is thought to rely for a large part on our ability to simulate others' thoughts and feelings via the self. That is, we can understand what others might be feeling, thinking, or aiming for, by putting ourselves into their shoes, i.e. by imagining what we would feel, think or do in their situation (Keysers & Gazzola, 2007). In other words, impairments of the DMN supporting self-referential thought could well be held responsible for a reduced ability to represent intentions and mental states of others, which in turn would result in various social impairments. Consistent with this perspective, several studies have indicated similar activations of DMN areas in conditions that required subjects to either think about themselves or think about close others (see review in Mitchell, Macrae, & Banaji, 2006; Moriguchi et al., 2006; Ochsner et al., 2005; Seger, Stone, & Keenan, 2004; Uddin et al., 2007). Furthermore, studies investigating structural abnormalities in autistic brains have been identified to overlap areas that are known to support theory of mind tasks and social cognition (Abell et al., 1999; Barnea-Goraly, Kwon, & Menon, 2004; Haznedar et al., 2000).

In line with the above suggestion that neurofeedback enhancement of DMN activation may both reduce ASD executive dysfunctions and at the same time improve children's social and communicative abilities, a significant improvement in general communication was found for children in the treatment group (14%), but not for children in the control group (−7%) on the CCC-2. This result was further supported by the estimated improvement of children in the treatment group on levels of social interaction (16%), communication (17%), and typical behavior (9%) as measured by the AUTI-R. These findings are in line with previous studies that reported significant reductions in ASD symptoms (Coben & Padolsky, 2007; Jarusiewicz, 2002) and improvements in behavior on several social and cognitive factors (Scolnick, 2005; Sichel et al., 1995) following neurofeedback training inhibiting theta activation.

Although the present findings are encouraging, studies with improved methodology regarding the effectiveness of neurofeedback training for children with ASD and other types of ASD are needed. This study used the same training protocol for each participant, but evidence is now growing for the use of an individualized protocol based on the individual EEG. We intend to incorporate protocols based on individualized EEGs in future research. The most important methodological improvement would be to control for direct, unintentional effects of neurofeedback training, such as providing extra time and attention to participants in the intervention group twice a week and learning them to handle an attention-demanding task like neurofeedback (Heinrich et al., 2007). We also expect indirect influence of neurofeedback training on children in the intervention group via their parents. Parents have brief talks or conversations with the neurofeedback trainer the minutes before and after neurofeedback sessions and during evaluations, and they get advice, encouragement, support, and compliments. These occasions raise expectations of improvement in parents, act upon parents' answers on behavior

² As a side-note, however, bear in mind that both theta and beta rhythms are not the sole property of the DMN areas or its associated function. Both theta and beta rhythms have been found in association with other areas and in support of different cognitive functions (e.g. Mantini et al., 2007).

questionnaires, and change the parents' approach to their children. A solution for this problem would be randomized double blind studies with random feedback for controls. However, the use of such a placebo condition raises ethical questions and therefore does not seem feasible. Instead of placebo feedback, neurofeedback training could be compared with established interventions like medication and behavior therapy (Heinrich et al., 2007), like Fuchs et al. (2003) did in ADHD. However, in the case of ASD it does not seem easy to create such a design. Comparison with medication is not attainable, since no appropriate medication is available for ASD (Buitelaar & Willemsen-Swinkels, 2000). Comparison with an intervention like behavior therapy seems almost impossible, since time and intensity of both the neurofeedback training and the time-consuming and more intensive behavior therapy should be kept constant (Matson and Smith, 2008).

In conclusion, application of a typical ADHD neurofeedback protocol to a group of ASD children was found to be highly affective. Neurofeedback treatment resulted in clear improvements in children's executive functioning as reflected in a wide range of tasks. These findings provide further evidence for a basic executive function impairment in ASD and suggest a relationship between enhanced theta/beta ratio's and hypoactivation of the ACC as a possible neural origin of this impairment.

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